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13th INTERNATIONAL CRITICAL CARE AND EMERGENCY MEDICINE CONGRESS

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A Deceptive Nail Gun Injury: Deep Patellar Penetration with Normal Physical Findings

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INTRODUCTION

Nail guns are tools that can cause serious penetrating injuries due to their high-pressure mechanisms. These injuries most commonly affect the extremities and are frequently encountered as occupational accidents (1).

In penetrating trauma, the external appearance may be misleading, and significant underlying tissue damage can be overlooked. Early diagnosis and treatment are particularly important in cases involving joint and bone involvement (2).

CASE REPORT

A 26-year-old male patient was brought to the emergency department after a nail became embedded in the anterior aspect of his right knee while using a nail gun at work. The patient was in good general condition, his vital signs were stable, and physical examination findings were unremarkable. Plain radiography of the right knee revealed a metallic foreign body at the level of the patella. An orthopedic and traumatology consultation was obtained. Due to the deep location of the foreign body, the patient was admitted and taken to the operating room, where the foreign body was surgically removed. No postoperative complications occurred, and the patient was discharged with recommendations.

Figure 1. External examination findings and radiographic imaging of the patient.

DISCUSSION

Although nail gun injuries are generally considered low-velocity penetrating traumas, they may lead to significant complications (1). In particular, bone and joint involvement increases the risk of infection, osteomyelitis, and functional impairment (2). Plain radiography is usually sufficient for diagnosis; however, advanced imaging modalities may be required in complex cases (3). Surgical removal is recommended for deeply embedded foreign bodies. Early intervention reduces the risk of complications (2,3). In the present case, despite normal physicalexamination findings, imaging revealed a significant penetrating injury, highlighting the importance of careful evaluation.

CONCLUSION

Although nail gun injuries may appear superficial, they can involve deep tissues. Clinical assessment should always be supported by imaging studies, and surgical intervention should not be delayed when indicated.

Keywords: Nail gun, penetrating trauma, patella, foreign body, orthopedics

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1026 **Syncope due to lithium intoxication: A case report**

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Abstract

In this report, we emphasise the importance of haemodialysis and a multidisciplinary approach in the management of lithium intoxication with cardiac involvement. A 67-year-old woman with a history of bipolar disorder and epilepsy was referred to our emergency department in an intubated and unconscious state. During the initial assessment, her Glasgow Coma Scale score was 3, and her pupils were mydriatic. Her medical history revealed that she had experienced chills, tremor, nausea, and fatigue for the preceding two to three days. During this period, she had received antibiotics and non-steroidal anti-inflammatory drugs

prescribed at another medical centre. Syncope developed following severe nausea and vomiting, which led to endotracheal intubation. On physical examination, bilateral rales were detected on auscultation, and chest radiography demonstrated a consolidation in the apex of the right lung. Thoracic computed tomography revealed an area of infiltration in the right upper lobe consistent with pneumonia. Given her history of lithium use, serum lithium levels were measured. A nephrology consultation was requested, and the patient was admitted to the intensive care unit for emergency haemodialysis. Emergency haemodialysis was performed for three hours within the first twenty-four hours. Following treatment, the serum lithium level decreased to 1.75 mmol/L. Renal function normalised during follow-up, and the initial consolidation observed on chest radiography was found to have completely regressed. The patient was discharged without sequelae on the twenty-sixth day of hospitalisation. Lithium intoxication should be considered in the differential diagnosis of patients presenting with syncope and unexplained altered consciousness, particularly in those with a history of bipolar disorder.

Keywords: Haemodialysis, Lithium toxicity, Syncope.

Introduction

Lithium is considered the gold standard in the treatment of bipolar disorder; however, due to its pharmacokinetic properties, it requires careful and regular monitoring (1). As lithium is almost entirely eliminated via the kidneys, any condition that impairs renal perfusion may result in dangerous elevations in serum lithium levels (2). Patients with lithium toxicity may present not only with neurological manifestations such as tremor, ataxia, and confusion, but also with cardiac complications including sinus node dysfunction and ventricular arrhythmias (4). In patients receiving chronic lithium therapy who present with syncope, lithium toxicity should therefore be considered a priority in the differential diagnosis (3,5). In this report, we emphasise the importance of haemodialysis and a multidisciplinary approach in the management of lithium intoxication with cardiac involvement.

Case Presentation

A 67-year-old woman with a history of bipolar disorder and epilepsy was referred to our emergency department in an intubated and unconscious state. During the initial assessment, her Glasgow Coma Scale (GCS) score was 3, and her pupils were mydriatic. Her medical history revealed that she had experienced chills, tremor, nausea, and fatigue for the preceding two to three days. During this period, she had received antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs) prescribed at another medical centre. Syncope developed following severe nausea and vomiting, which led to endotracheal intubation. On physical examination, bilateral rales were detected on auscultation, and chest radiography demonstrated a consolidation in the apex of the right lung (Figure 1). Thoracic computed tomography revealed an area of infiltration in the right upper lobe consistent with pneumonia (Figure 2). Given her history of lithium use, serum lithium levels were measured (Table 1). A nephrology consultation was requested, and the patient was admitted to the intensive care unit for emergency haemodialysis. Emergency haemodialysis was performed for three hours

within the first twenty-four hours. Following treatment, the serum lithium level decreased to 1.75 mmol/L. Renal function normalised during follow-up, and the initial consolidation observed on chest radiography was found to have completely regressed (Figure 3). The patient was discharged without sequelae on the twenty-sixth day of hospitalisation.

Discussion

Lithium toxicity is a critical condition because its elimination depends almost entirely on renal function (2). Elevated lithium levels are frequently precipitated by dehydration or by the use of non-steroidal anti-inflammatory drugs (NSAIDs), which impair renal perfusion (3). In the present case, it is presumed that the medications used to treat the infection reduced renal clearance, resulting in lithium accumulation and subsequent syncope. Although the cardiac effects of lithium are most commonly reported in the literature as electrocardiographic changes, profound altered consciousness and syncope were the predominant clinical manifestations in our patient (4). It is well recognised that neurological injury may become permanent when lithium levels exceed 2 mmol/L; however, owing to early haemodialysis, our patient recovered without any sequelae (1,5).

Conclusion

Lithium intoxication should be considered in the differential diagnosis of patients presenting with syncope and unexplained altered consciousness, particularly in those with a history of bipolar disorder.

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Table 1. Course of the patient's laboratory values

Parameter	Admission	Post-Dialysis	Day 4	Day 12	Discharge
Lithium (mmol/L)	2.46	1.75	0.94	-	~0.6
Urea (mg/dl)	62.8	46	82	-	30
Creatinine (mg/dl)	2.0	1.3	0.8	-	0.7

Parameter	Admission	Post-Dialysis	Day 4	Day 12	Discharge
AST (U/L)	994	341	24	-	20
ALT (U/L)	911	707	196	-	30
Troponin (ng/L)	335	304	52	-	Normal
pH	7.29	7.39	-	-	7.40
WBC (10^9/L)	17.6	-	-	-	7.0

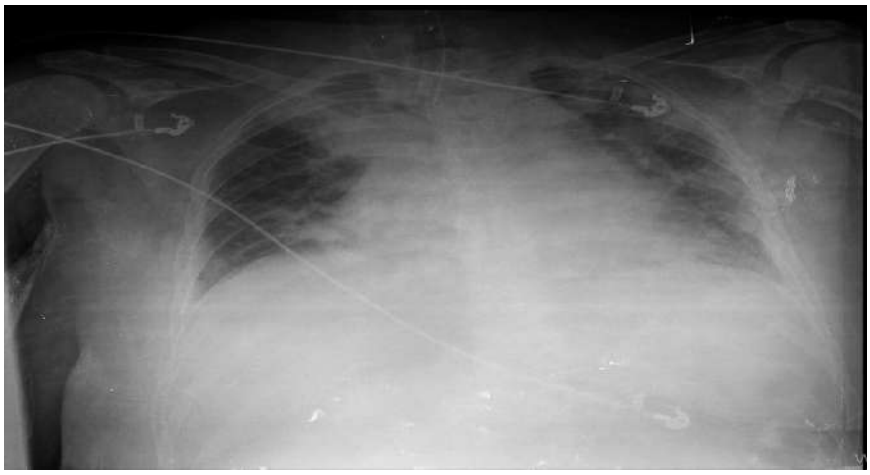

Figure 1: Consolidation area in the apex of the right lung.

Figure 2: Infiltration area in the right upper lobe.

Figure 3: Posterior-anterior chest X-ray at discharge.

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Antimicrobial Dynamics in Emergency Medicine: A Review of the Literature

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ABSTRACT

Introduction and Aim: Currently, challenges persist in translating the rational use of antibiotics (ABs) into clinical practice, particularly in emergency medicine. Emergency departments (EDs) are interfaces in the relationship between individuals and hospital services. In particular, EDs are an important environment for addressing the problem of inappropriate AB prescribing and the accompanying antimicrobial resistance. The aim of this study is to provide practical recommendations for important strategies and methods that can be applied in EDs, based on existing evidence.

Materials and Methods: A retrospective worldwide literature review revealed statistically significant data and developed AB management systems in this area.

Result and Conclusion: Across the literature, appropriate antibiotic use and the standards for treating AS should be adapted to each AS condition. The most suitable approach should be developed considering local sensitivities. According to the American Infectious Diseases Society (IDSA) guidelines, there are important recommendations regarding antibiotic treatment and duration for simple infections in AS patients. The IDSA primarily recommends macrolides for community-acquired pneumonia, nitrofurantoin for uncomplicated cystitis, ciprofloxacin for pyelolephritis, clindamycin for skin abscesses, and cephalexin for cellulitis.

The recommended duration of antibiotic use ranges from 3 to 7 days on average. According to the literature, various management systems exist regarding the use of antimicrobial drugs in AS. These include patient and physician education, the development of clinical treatment algorithms, the presence of an emergency department pharmacist in the emergency department, the use of antibiotic prescribing forms completed by the emergency physician and consulted by a pharmacist/infectious disease specialist, evaluation of drug use based on patient clinical and microbiology data 48-72 hours after antibiotic treatment, increased use of molecular and rapid diagnostic tests in the decision-making phase of antibiotic treatment in the emergency department, antibiotic dose optimization, antibiogram applications, and the presence of computer-aided clinical decision support systems that consider patient history and local sensitivities related to the emergency department during the prescription phase, can recommend drugs, and provide feedback on treatment success.

In conclusion, even though various antimicrobial management strategies specific to the emergency department have been developed in the global world, more literature information is needed to define these strategies due to observed shortcomings in their implementation. Since not all antimicrobial strategies are suitable for all emergency departments, these strategy programs should be developed among stakeholders such as hospital administrators, pharmacists, local communities, and infectious disease specialists. However, the emergency department physician is the most critical expert in deciding on the correct antimicrobial treatment, possessing the ability to determine the most effective strategies taking into account all local sensitivities.

Keywords: Antibiotic, emergency department, antimicrobial treatment

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Recurrent Emergency Department Visits Within 72 Hours: Clinical Characteristics and Sequential Analysis

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Abstract

Objective:

Repeated emergency department visits within 72 hours are an important indicator in the evaluation of healthcare utilization and quality of care. However, in the literature, these visits are mostly evaluated according to the total number of visits, and data regarding the separate assessment of consecutive visits are limited. In this study, repeated visits were aimed to be analyzed separately as second, third, and fourth visits.

Materials and Methods:

In this retrospective observational study, a total of 5,280 emergency department visits within 72 hours were evaluated. A total of 235 patients with repeated visits were included in the study. Visits were classified as second, third, and fourth visits and analyzed separately as distinct clinical episodes. Demographic data, triage categories, diagnoses, administered treatments, requirements for additional diagnostic tests and consultations, and hospitalization status were recorded. Statistical analyses were performed using SPSS version 27.

Results:

The median age of patients with repeated visits was 42 years (IQR: 28–57). Of the patients, 91.5% presented twice, 7.7% presented three times, and 0.9% presented four times. The majority of patients were evaluated in the green triage area, and the hospitalization rate was low. The most common diagnoses were upper respiratory tract infection (36.6%) and myalgia (23.4%). Additional diagnostic tests were performed in 32.3% of second visits. As age increased, the rates of diagnostic testing, consultation, and higher triage acuity also increased; however, no significant association was found between age and hospitalization. Most third and fourth visits were associated with low-acuity conditions, while a history of psychiatric illness was notable among patients with four visits.

Conclusion:
The majority of repeated emergency department visits within 72 hours are associated with low-acuity and self-limiting conditions. These visits may be related more to symptom perception, patient expectations, and psychosocial factors rather than clinical severity. Patient education and appropriate referral strategies may play an important role in reducing unnecessary emergency department visits.

Keywords: Emergency department, repeated visits, 72 hours, triage, myalgia

Intorduction

Emergency departments are frequently utilized healthcare units for the rapid evaluation and management of acute health problems. However, repeated emergency department visits, particularly those occurring within 72 hours, represent an important issue in terms of effective healthcare utilization, patient safety, and quality of care. Although these visits are often considered an indicator of quality of care, the underlying causes are influenced by multiple factors related to the natural course of the disease, patient characteristics, and the healthcare system. Triage systems used in emergency departments to prioritize patients enable classification according to clinical severity and play an important role in the efficient use of healthcare resources.

Previous studies have shown that a substantial proportion of repeated emergency department visits are associated with low-acuity and self-limiting conditions such as upper respiratory tract infections and non-specific pain syndromes (1). These visits are generally related to persistent symptoms, patient anxiety, or unmet expectations rather than progression of the disease itself. In addition, inadequate evaluation during the initial visit, defensive medical practices, and psychosocial factors are among the important determinants of repeated visits (2). Furthermore, the easy accessibility of emergency departments and patients' expectations for rapid results may also contribute to the increase in these visits.

The majority of studies in the literature evaluate repeated visits according to the total number of presentations and classify patients as "frequent visitors." However, studies evaluating second, third, and fourth visits separately as distinct clinical episodes are quite limited. Nevertheless, sequential evaluation of repeated visits may contribute to a better understanding of patient behavior, clinical course, and healthcare utilization patterns.

The aim of this study was to evaluate patients with repeated emergency department visits within 72 hours separately as second, third, and fourth visits, and to investigate the clinical characteristics, diagnostic distributions, and management approaches associated with these visits.

Materials and Methods

This retrospective observational study was conducted in the emergency department of a tertiary care hospital. Within the scope of the study, a total of 5,280 emergency department

visits occurring within a 72-hour period were evaluated. Among these visits, 235 patients with repeated presentations were included in the study. Repeated visits were classified as second, third, and fourth visits, and each visit was analyzed as a separate clinical episode.

Patients' demographic data (age and sex), clinical characteristics (triage category and time of presentation), diagnoses, treatment modalities, requirements for additional diagnostic tests, consultation status, and hospitalization data were recorded. Triage categories were classified as green, yellow, and red zones in accordance with standard emergency department triage protocols.

Statistical analyses were performed using IBM SPSS Statistics version 27 software. The conformity of continuous variables to normal distribution was assessed using the Kolmogorov–Smirnov test. Parametric data were expressed as mean ± standard deviation, whereas non-parametric data were presented as median and interquartile range (IQR). For intergroup comparisons, the independent samples t-test was used for normally distributed variables, while the Mann–Whitney U test was used for non-normally distributed variables. The chi-square test was applied for the comparison of categorical variables. A p-value of <0.05 was considered statistically significant.

Results

Patient Demographics and General Visit Characteristics

A total of 5,280 emergency department visits within 72 hours were identified. Repeated emergency department visits were detected in 235 patients. The median age of all patients with repeated visits was 42 years (IQR 25–75: 28–57). When the distribution of patients according to the number of visits was evaluated, 91.5% (n=215) presented to the emergency department twice, 7.7% (n=18) three times, and 0.9% (n=2) four times. None of the patients with repeated visits died.

Initial Visit Data

When the treatments administered to patients with repeated visits were evaluated, 49.8% (n=117) received intravenous (IV) treatment, 32.3% (n=76) received intramuscular (IM) treatment, and 17.9% (n=42) did not receive any treatment.

Analysis of Second Visits

The median age of patients presenting for a second visit was 42 years (IQR 25–75: 28–57). Of these visits, 59.6% (n=140) occurred during daytime hours. A total of 56.2% (n=132) of patients were evaluated in the green triage area, while 1.7% (n=4) were examined in the red triage area. During the second visit, 32.3% of patients (n=76) underwent additional diagnostic testing that had not been performed during the previous visit. The hospitalization rate was found to be 4.7% (n=11), whereas the consultation rate was recorded as 12.8% (n=30). Regarding diagnostic distribution, upper respiratory tract infection (URTI) accounted for

36.6% of cases and myalgia for 23.4%, while the remaining 40% consisted of other diagnoses.

The statistically significant findings identified during second visits were as follows:

- The mean ages of patients who underwent additional diagnostic testing and those presenting during daytime hours were significantly higher (p=0.001 and p=0.013, respectively).
- The mean age was higher among patients requiring consultation and among those triaged in the yellow/red categories (p=0.027 and p=0.001, respectively).
- No statistically significant association was found between age and hospitalization (p=0.079).
- IM treatment was preferred in younger patients, whereas the mean age was higher in patients who received IV treatment or no treatment (p=0.02).

Table 1. Distribution of Diagnosis Categories Among Patients (n = 235)

Diagnosis Category	Number of Patients (n)	Percentage (%)
Upper Respiratory Tract Infection (URTI)	86	36,6
Myalgia	55	23,4
General Medical Examination	21	8,9
Headache	10	4,3
Urinary Tract Infection (UTI)	9	3,8
Acute Gastroenteritis (AGE)	9	3,8
Abdominal Pain	7	3
Urticaria	6	2,6
Eye Diseases	3	1,3
Dizziness (Vertigo)	2	0,9
Nausea	2	0,9
Other	25	1,6
Total	**235**	**100**

Third Visit Data

The mean age of patients presenting three times was 37.44 ± 6.44 years (Mean ± SD). Of this group, 61.1% (n=11) were male, and 61.1% (n=11) of the visits occurred during daytime hours. A total of 66% of patients (n=12) were evaluated in the green triage area, and no patients were admitted to the red triage area. Additional diagnostic tests that had not been performed during previous visits were ordered in 27.8% of patients (n=5). Regarding treatment modalities, 38.9% (n=7) received IV treatment, while 38.9% (n=7) received no treatment. Only one patient (5.6%) was hospitalized. The most common diagnoses were myalgia (38.9%) and upper respiratory tract infection (27.8%). Analyses comparing age between groups revealed no statistically significant differences ($p > 0.05$ for all variables).

Table 2. Distribution of Diagnosis Categories Among Patients (n = 18)

Diagnosis Category	Number of Patients (n)	Percentage (%)
Myalgia	7	38.9
Upper Respiratory Tract Infection (URTI)	5	27.8
General Medical Examination	1	5.6
Nausea	1	5.6
Other	4	22.2
Total	**18**	**100.0**

Characteristics of Fourth Visits

A total of two patients were identified as having presented to the emergency department four times. Both patients (one female and one male, aged 56 and 61 years, respectively) presented during daytime hours and were triaged in the green area. Both patients had a history of psychiatric illness in their medical records. During these visits, no additional diagnostic tests were requested, no consultations were performed, no treatment was administered, and neither hospitalization nor exitus (death) occurred.

Discussion

In our study, the median age of patients with repeated emergency department visits was found to be 42 years (IQR: 28–57). Similarly, previous studies investigating repeated emergency department visits have reported that these visits are frequently concentrated in the 40–45-year age group (3). We believe that repeated presentations are more common in this age group because the population defined as “frequent users” of emergency departments predominantly consists of young and middle-aged adults of working age.

In this study, the vast majority of patients who re-presented within 72 hours visited the emergency department twice (91.5%), whereas three or more visits were relatively rare (8.6%). Similarly, previous studies have reported that early repeated emergency department visits are generally limited to a single additional presentation, while higher numbers of repeated visits are uncommon (4). We believe that this finding may be related to the administration of adequate treatment during the first two visits, resulting in a limited number of subsequent presentations.

In the present study, no mortality was observed among patients with repeated visits. Likewise, the literature suggests that early repeated emergency department visits are generally associated with low mortality risk and are more commonly related to the clinical course of the disease or persistence of symptoms (4). We consider that the short-term prognosis of this patient group is generally favorable.

In our study, evaluation of the treatment patterns administered during the initial presentation of patients with repeated visits revealed that 49.8% received intravenous (IV) treatment, while 32.3% underwent intramuscular (IM) intervention. Similar studies in the literature have reported that a substantial proportion of patients with repeated emergency department visits present with pain-related complaints such as renal colic, myalgia, and abdominal pain, resulting in high rates of analgesic use (1). In our dataset, the predominance of upper respiratory tract infection (36.6%) and myalgia (23.4%) as the most common diagnoses supports these high rates of parenteral treatment.

However, an important point emphasized in the literature is that parenteral treatment modalities, particularly IV hydration and analgesia, may paradoxically increase repeated emergency department visits by creating a sense of “emergency department dependence” or an expectation of rapid symptom relief among patients. The overall high rate of parenteral treatment in our study (82.1%) may therefore be associated with the increased perception of symptom severity among patients returning to the emergency department and the pressure felt by clinicians to provide rapid symptomatic relief, as described in previous studies.

In our study, the majority of patients (56.2%, n=132) were evaluated in the green triage area, whereas only a very small proportion (1.7%, n=4) were examined in the red triage area. Previous studies conducted in Türkiye have demonstrated that approximately 50–70% of emergency department visits consist of non-urgent green triage patients, while red triage

patients representing true emergency conditions account for only 1–3% of presentations (5). This indicates that our findings are fully consistent with national data. We believe that this pattern reflects the extensive use of emergency departments by low-priority cases that could otherwise be managed within primary healthcare services, representing a consequence of inappropriate emergency department utilization.

During the second visit, 32.3% of patients (n=76) underwent additional diagnostic tests that had not been performed during the initial presentation. The consultation rate in these patients was 12.8% (n=30), while the hospitalization rate was found to be 4.7% (n=11). Previous studies have reported that completion of previously omitted diagnostic evaluations and increased consultation requests are common during repeated visits (6). We believe that this may be associated both with the dynamic clinical course of diseases and with defensive medical practices.

Regarding diagnostic distribution, upper respiratory tract infection (URTI) was observed in 36.6% of patients and myalgia in 23.4%, while the remaining 40% presented with other diagnoses. Similarly, a previous study reported that patients diagnosed with upper respiratory tract infections constituted the most common group among repeated emergency department visits (7). This finding is consistent with the literature, and we believe that a considerable proportion of repeated emergency department visits may represent unnecessary utilization.

The mean age was found to be significantly higher among patients who underwent additional diagnostic testing and those presenting during daytime hours ($p=0.001$ and $p=0.013$, respectively). Similarly, patients requiring consultation and those triaged in the yellow/red categories had significantly higher mean ages ($p=0.027$ and $p=0.001$, respectively). In contrast, no statistically significant association was identified between age and hospitalization ($p=0.079$). Furthermore, intramuscular treatment was more frequently preferred in younger patients, whereas the mean age was higher among patients receiving intravenous treatment or no treatment ($p=0.02$).

Although the need for consultation and additional diagnostic testing increased with age, no significant difference was observed in hospitalization rates among older patients. These findings, which are also consistent with the literature, suggest that advanced age alone is not a sufficient criterion for hospitalization and that admission decisions should primarily be based on clinical findings and disease severity (8–10).

The mean age of patients presenting three times was 37.44 ± 6.44 years (Mean ± SD). Of these patients, 61.1% (n=11) were male, and 61.1% (n=11) of the visits occurred during daytime hours. A total of 66% of patients (n=12) were evaluated in the green triage area, and no patients were triaged in the red area. Additional diagnostic tests that had not been performed during previous visits were ordered in 27.8% of patients (n=5). Regarding treatment modalities, 38.9% (n=7) received IV treatment, while 38.9% (n=7) received no treatment. Only one patient (5.6%) required hospitalization. The most common diagnoses

were myalgia (38.9%) and upper respiratory tract infection (27.8%). No statistically significant differences were found between groups in terms of age ($p > 0.05$ for all variables).

In the literature, repeated emergency department visits are generally evaluated based on the total number of visits, and studies separately analyzing second, third, and fourth visits are quite limited. In this respect, our study provides a more dynamic and original perspective to the literature by evaluating repeated visits in sequential phases. When the second, third, and fourth visits were assessed together in our study, it was notable that the patient profile progressively demonstrated lower-acuity characteristics.

The mean age of patients presenting for a third visit was 37.44 ± 6.44 years, and 61.1% of these patients were male, indicating an increase in the proportion of male patients compared to the second-visit group. In this group, 66% of patients were evaluated in the green triage area, and no patients were triaged in the red area, suggesting that repeated visits were largely associated with benign and low-risk clinical conditions. Regarding diagnostic distribution, similar to the second-visit group, the most common diagnoses were myalgia (38.9%) and upper respiratory tract infection (27.8%). However, compared to second visits, the proportion of myalgia increased while the proportion of upper respiratory tract infections relatively decreased during third visits. This finding suggests that subjective symptoms, particularly pain-related complaints, may lead to a higher likelihood of repeated emergency department presentations.

Evaluation of treatment approaches demonstrated that 38.9% of patients received intravenous treatment during the third visit, whereas 38.9% received no treatment at all. This finding may indicate that physicians tend to avoid unnecessary interventions during repeated visits and adopt a more conservative approach in clinically stable patients. Consistent with this observation, the hospitalization rate was very low (5.6%), supporting that most patients in this group could be managed on an outpatient basis.

Furthermore, no statistically significant difference was found between second, third, and fourth visits in terms of age, suggesting that repeated presentations may be associated more with persistence of symptoms and patients' perceived need for further treatment rather than an age-dependent condition. Taken together, these findings indicate that a substantial proportion of repeated emergency department visits are related to low-acuity, self-limiting, and symptomatic conditions. Therefore, emergency department utilization appears to be influenced not only by clinical necessity but also by patient perception and expectations. In this context, strengthening patient education, symptom management strategies, and alternative healthcare access pathways may contribute significantly to reducing repeated emergency department visits.

Only two patients were identified as presenting to the emergency department four times, and notably, both patients (aged 56 and 61 years; one female and one male) presented during daytime hours and were triaged in the green area. The presence of a psychiatric disease history in both patients suggests that repeated emergency department visits may be associated not only with organic pathologies but also with psychosocial factors. Similarly, a previous

study emphasized that individuals with psychiatric disorders are at high risk for repeated emergency department visits and that these presentations may often be related more to underlying psychosocial needs than to true clinical necessity (11).

In our study, the absence of additional diagnostic testing, consultation requirements, treatment administration, or hospitalization among patients presenting four times further supports that these visits were associated with low-acuity and clinically stable conditions. Nevertheless, we believe that four emergency department presentations within a short period, particularly within 72 hours, do not represent a healthy pattern of healthcare utilization. This situation may be related to factors such as inadequate patient referral, insufficient symptom control, or unmet psychiatric support needs. Therefore, in this patient group, not only acute symptom management but also psychiatric evaluation, patient education, and appropriate healthcare guidance may play an important role in reducing unnecessary emergency department visits.

Conclusion

This study demonstrated that repeated emergency department visits within 72 hours are largely associated with low-acuity, self-limiting, and symptomatic conditions. Most presentations were related to benign diagnoses such as upper respiratory tract infection and myalgia, while hospitalization rates were low. Although the rates of diagnostic testing and consultation increased with age, no significant association was found between age and hospitalization, indicating that age alone is not a sufficient criterion for hospital admission. Furthermore, repeated presentations within a short period may be associated more with symptom perception, patient expectations, and psychosocial factors rather than true clinical necessity. Therefore, patient education, appropriate referral strategies, and effective symptom management should be prioritized in order to reduce repeated emergency department visits.

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1151

Dissection or Occlusion?

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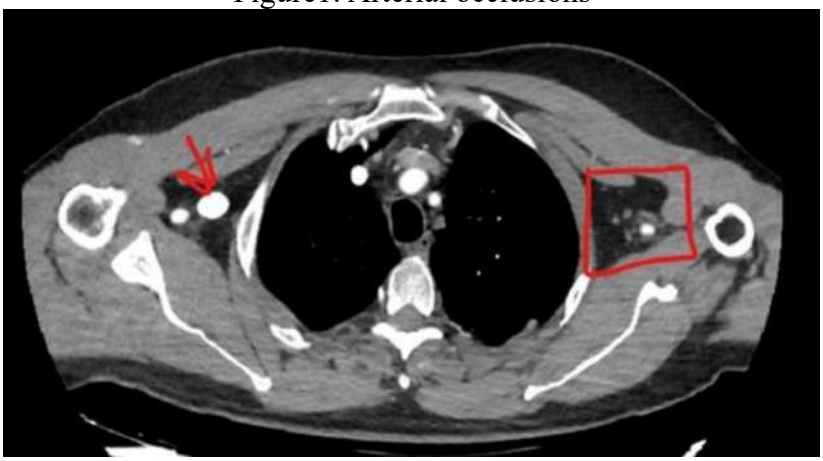
Introduction and Purpose: Arterial occlusions can present to the emergency department not only with impaired tissue perfusion but also with symptoms mimicking myalgia. Following physical examination, Doppler ultrasonography is the first choice; CT angiography is the gold standard for definitive diagnosis. In this case, we address the clinical management of a single-kidney patient who presented to the emergency department with sudden onset back pain radiating to the left arm. The diagnostic approach, imaging strategy, and treatment decision-making process are reviewed in the emergency care.

Materials and Methods: A 65-year-old male patient presented to the emergency department with sudden onset, tearing back pain radiating to the left arm, which had started three hours prior. His medical history included congenital renal agenesis. On physical examination, extremity muscle strength was 5/5 on both sides. Extremity temperature, color, and capillary refill were equal and normal. However, the left radial and ulnar pulses were not palpable. Blood pressure: 80/40 mmHg in the left arm and 130/80 mmHg in the right arm. Given the patient's known history of congenital renal agenesis, initial imaging with Doppler ultrasonography was planned. Doppler US showed detectable arterial flow. However, on

repeated examinations, despite detectable flow with Doppler, the left radial and ulnar pulses were not palpable. Therefore, the patient was assessed as clinically unstable, and emergency CT angiography was performed without waiting for biochemical results. CT angiography revealed complete occlusion of the left subclavian artery. Cardiovascular surgery consultation was performed, and endovascular intervention was planned for the patient.

Figure1: Arterial occlusions



Results and Conclusion: In patients presenting to the emergency department with non-traumatic extremity pain, myalgia is frequently among the preliminary diagnoses. However, life-threatening vascular pathologies with high mortality rates, such as aortic dissection and acute arterial occlusion, should also be considered in the differential diagnosis. Even if skin findings appear normal during extremity examination, pulse assessment is essential. If clinical suspicion persists, bilateral blood pressure measurement and further diagnostic evaluation should be performed. In emergency situations, especially in patients describing sudden onset tearing pain, it is vital to consider not only dissection but also vascular conditions with high mortality rates, such as arterial occlusion.

Keywords: blood pressure, CT angiography, pulse

1182 **Relationship Between Lactate and Length of Hospital Stay in Isolated Extremity Gunshot Injuries in the Emergency Department**

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Abstract

Introduction:
Firearm injuries are a significant cause of morbidity and mortality worldwide. In recent years, an increase has been reported, particularly in non-fatal cases. A large proportion involves

extremity injuries, and most patients present to the emergency department in a hemodynamically stable condition. Although isolated extremity firearm injuries are generally not life-threatening, they may cause serious morbidity due to vascular, nerve, and soft tissue damage. Additionally, even in low-energy civilian injuries, unpredictable tissue damage related to bullet ballistics can complicate diagnosis and management.

Objective:
Although considered relatively low risk, predicting the clinical course of isolated extremity gunshot wounds remains challenging. This study aimed to evaluate the relationship between routinely measured laboratory parameters and the length of hospital stay.

Materials and Methods:
This retrospective study included patients presenting with isolated extremity gunshot wounds between September 2025 and February 2026. Patients with abdominal, thoracic, or cranial injuries were excluded. Demographic data and laboratory parameters, including albumin, C-reactive protein (CRP), platelet count, hemoglobin, neutrophils, monocytes, lymphocytes, pH, and lactate levels, were analyzed. Statistical analyses were performed using SPSS version 27, and relationships were evaluated using Spearman correlation analysis.

Findings:
A total of 37 patients were included, 94.6% of whom were male. The mean age was 30.84 ± 10.05 years, and the mortality rate was 2.7%. Most patients had normal laboratory values and a stable course. Median lactate level was 2.3 mmol/L, and median hospital stay was 1 day. Lactate showed a moderate positive correlation with hospital stay ($r=0.517$, $p=0.003$). Weak positive correlations were found for CRP ($r=0.355$, $p=0.036$) and age ($r=0.337$, $p=0.042$), while other parameters were not significant.

Conclusion:
Lactate was the most prominent parameter associated with hospital stay and may serve as a simple biomarker for early risk stratification.

Keywords:
Gunshot wound, extremity injury, lactate, emergency department, length of hospital stay

Introduction

Gunshot injuries are currently a major cause of morbidity and mortality in both developed and developing countries (1,2). In recent years, a marked increase has been reported particularly in non-fatal firearm injuries. A significant proportion of these injuries involve the extremities, and most patients present to the emergency department in a hemodynamically stable condition (3,4).

Although isolated extremity gunshot injuries are generally not considered life-threatening, they may lead to substantial morbidity due to accompanying vascular, neural, and soft tissue injuries (5,6). Furthermore, even in low-energy civilian injuries, unpredictable tissue damage associated with bullet ballistics may occur, complicating the diagnostic and therapeutic process.

The management of this patient group in the emergency department remains controversial regarding imaging, evaluation of vascular injury, and the necessity for surgical consultation. In particular, the absence of clear management standards in patients without associated systemic injuries leads to heterogeneity in clinical practice (6,7).

Therefore, evaluating the clinical characteristics and emergency department management processes of isolated extremity gunshot injuries is of considerable importance.

In our study, we investigated the relationship between blood parameters assessed in the emergency department and the length of hospital stay in patients with isolated extremity gunshot injuries.

Materials and Methods

Study Design and Data Source

This study was conducted retrospectively by reviewing patients presenting to our emergency department with isolated extremity gunshot injuries between September 2025 and February 2026. Data were obtained from the hospital information management system.

Inclusion and Exclusion Criteria

Only patients with isolated extremity gunshot injuries were included in the study. Patients with abdominal, thoracic, or cranial injuries were excluded.

A total of 45 patients were evaluated. Thirty-seven patients who met the inclusion and exclusion criteria were included in the study.

Evaluated Parameters

Patient age (years), albumin (g/L), C-reactive protein (CRP, mg/L), platelet count (PLT, $\times 10^9$/L), hemoglobin (HGB, g/dL), neutrophil count ($\times 10^9$/L), monocyte count ($\times 10^9$/L), lymphocyte count ($\times 10^9$/L), pH, and lactate (mmol/L) values, as well as hospitalization status, were evaluated.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics 27.0 (IBM Corp., Armonk, NY, USA). The normality of variable distributions was assessed. Parametric data were expressed as mean ± standard deviation, whereas non-parametric data were expressed as median (minimum–maximum). Categorical variables were presented as frequency and percentage (%). Relationships between length of hospital stay and other parameters were evaluated using Spearman correlation analysis. Correlation coefficients were interpreted according to the classification proposed by Mukaka: <0.30 negligible, 0.30–0.49 weak, 0.50–0.69 moderate, 0.70–0.89 strong, and ≥0.90 very strong correlation (8). A p-value <0.05 was considered statistically significant.

Results

A total of 37 patients were included in the study. Of these, 35 (94.6%) were male and 2 (5.4%) were female. The mortality rate was determined to be 2.7%, with one patient reported as deceased.

Normality analysis demonstrated that age, platelet count (PLT), hemoglobin (HGB), and monocyte values showed parametric distribution, whereas the remaining variables showed non-parametric distribution.

The mean age of the patients was calculated as 30.84 ± 10.05 years. Median albumin level was 4.5 g/dL (IQR: 4.2–4.7), and median CRP level was 0.2 mg/L (IQR: 0.1–0.6).

Mean platelet count was 274.14 ± 28.20 ×10⁹/L, and mean hemoglobin level was 15.16 ± 1.60 g/dL. Median neutrophil count was 6.5 ×10⁹/L (IQR: 5.05–10.73), mean monocyte count was 0.81 ± 0.24 ×10⁹/L, and median lymphocyte count was 3.22 ×10⁹/L (IQR: 2.08–4.18).

In blood gas analysis, the median pH value was 7.38 (IQR: 7.31–7.40). Median lactate level was 2.3 mmol/L (IQR: 0.8–6.85). Median length of hospital stay was calculated as 1 day (IQR: 0–6.5 days). Detailed findings are presented in Table 1.

Table 1. Demographic, Clinical, and Laboratory Characteristics of the Study Population

					IQR	
Variables	n	Mean	SD	Median	25%	75%
Age (year)		30.84	10.05			
Plt (10^3/uL)		274.14	28.20			
Hgb (mmol/L)		15.16	1.60			
Mono (10^3/uL)		0.81	0.24			
Albumin (g/L)				4.5	4.2	4.7
Crp (mg/L)				0.2	0.1	0.6
Neu (10^3/uL)				6.5	5	10.7
Lym (10^3/uL)	37			3.2	2	4.1
PH				7.38	7.31	7.40
Lactat				2.3	0.8	6.8
HALP				0.7	0.4	1.3
SIRI				1.5	1	4.2
SII				619.7	369.6	1128.74
Length of hospital stay (day)				1	0	6.5

The relationships between length of hospital stay and other parameters were evaluated using Spearman correlation analysis. A moderate positive correlation was identified between length of hospital stay and lactate level (r=0.517, p=0.003).

Additionally, a weak positive correlation was found between CRP level and length of hospital stay (r=0.355, p=0.036), and between age and length of hospital stay (r=0.337, p=0.042).

No statistically significant relationship was observed between length of hospital stay and the other parameters, including albumin, platelet count, hemoglobin, neutrophil count, monocyte count, lymphocyte count, and pH (p>0.05).

Discussion

Gunshot injuries are currently a major cause of morbidity and mortality in both developed and developing countries (1,2). In recent years, a marked increase has been reported particularly in non-fatal firearm injuries. A substantial proportion of these injuries involve the extremities, and most patients present to the emergency department in a hemodynamically stable condition (3,4).

In our study, the majority of patients were young males, and mortality was low. This finding is consistent with recent studies reporting that firearm injuries are more common in the young male population and that mortality is generally low in isolated extremity injuries (3,9).

We did not detect a marked systemic inflammatory response in laboratory findings. The fact that CRP and albumin levels were mostly within normal ranges is consistent with studies demonstrating limited systemic involvement in isolated and low-energy gunshot injuries (5). This finding is also compatible with the clinically stable course observed in most patients.

The most remarkable finding of our study was the moderate positive correlation between lactate level and length of hospital stay. Lactate is known to be an early indicator of tissue hypoperfusion in trauma patients and has been associated with clinical outcomes (5,10). Our findings are in agreement with the literature and suggest that lactate may be valuable not only in major trauma but also in predicting the clinical course of isolated extremity injuries.

In addition, we identified weak correlations between CRP and length of hospital stay, and between age and length of hospital stay. Although these findings suggest that increased inflammatory response and advanced age may prolong the clinical course, these associations appeared to be more limited compared with lactate.

In conclusion, our study demonstrates that lactate level is the most prominent parameter associated with the length of hospital stay in isolated extremity gunshot injuries. This finding suggests that lactate may be used as a simple and accessible biomarker for early risk stratification in emergency department practice.

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1184

ACUTE PULMONARY EDEMA DEVELOPING AFTER HYPERTONIC SALINE ADMINISTRATION FOR THE TREATMENT OF SYMPTOMATIC HYPONATREMIA

(Case Report)

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ABSTRACT

Although 3% hypertonic saline is an effective agent in the treatment of symptomatic hyponatremia, it carries a risk of iatrogenic volume overload and acute pulmonary edema, particularly in elderly patients and those with underlying chronic kidney disease. In this report, we present a case of acute pulmonary edema that developed approximately 3 hours after initiation of a 3% hypertonic saline infusion in a 72-year-old female patient with a history of hypertension and chronic kidney disease. Following rapid stabilization with non-

invasive mechanical ventilation, intravenous furosemide, and perlinganit, complete clinical recovery was achieved. The clinical course is discussed in light of the current literature.

Keywords: hyponatremia, hypertonic saline, acute pulmonary edema, iatrogenic complication, non-invasive ventilation, emergency departmen

1. INTRODUCTION

Hyponatremia is the most common electrolyte disorder encountered in hospitalized patients and is closely associated with increased morbidity and mortality (1). It is generally defined as a serum sodium concentration below 135 mEq/L; however, clinical severity depends not only on the sodium level but also on the rate of development and accompanying symptoms. In cases of severe hyponatremia (Na <125 mEq/L) accompanied by neurological manifestations such as confusion, lethargy, seizures, or coma, 3% hypertonic saline (NaCl) is considered a life-saving treatment option (2). Nevertheless, the therapeutic window of 3% hypertonic saline is narrow. Excessively rapid correction of sodium may lead to osmotic demyelination syndrome, whereas uncontrolled volume expansion may precipitate acute pulmonary edema and hypertensive crisis, particularly in patients with limited cardiovascular and renal reserve (2,3). Current literature suggests that administration as a 100–150 mL bolus is safer than continuous infusion; however, continuous infusion remains a common practice in many centers in Türkiye (2). In this case report, we present a patient who developed acute pulmonary edema approximately 3 hours after initiation of 3% hypertonic saline therapy in the setting of advanced age and chronic kidney disease (CKD). The mechanisms of this iatrogenic complication, early diagnostic clues, and emergency department management are discussed in light of the literature.

2. CASE

Presentation

A 72-year-old woman presented to the emergency department with complaints of nausea and vomiting that had persisted for approximately one day. Her medical history was significant for hypertension and stage 3 chronic kidney disease (CKD). She was receiving regular antihypertensive therapy and had no history of dialysis

Physical Examination

The patient appeared moderately ill, fatigued, and lethargic. She was conscious but partially disoriented. Initial vital signs indicated hemodynamic stability: arterial blood pressure 110/70 mmHg, heart rate 92 beats/min, oxygen saturation (SpO_2) 94% on room air, and body temperature 36.7°C. Pulmonary auscultation was unremarkable, heart sounds were rhythmic, and there was no peripheral edema or jugular venous distention. Abdominal examination was normal.

Laboratory Findings

Initial laboratory evaluation revealed a serum sodium level of 125 mEq/L (reference: 135–145), creatinine 1.35 mg/dL (reference: 0.6–1.1), blood urea nitrogen (BUN) 55 mg/dL (reference: 15–45), normal potassium levels, and low serum osmolality consistent with

hypotonic hyponatremia. The clinical presentation (lethargy, nausea, and vomiting) together with laboratory findings was considered compatible with symptomatic hyponatremia. While further investigations into the underlying etiology were ongoing, controlled intravenous infusion of 3% hypertonic saline was initiated, and close monitoring of serum sodium levels and clinical status was planned.

Clinical Course

Approximately 3 hours after initiation of hypertonic saline infusion, the patient developed sudden-onset dyspnea, tachypnea, abdominal breathing, and profuse sweating. Repeat vital sign measurements demonstrated a rise in arterial blood pressure to 200/110 mmHg and heart rate to 110 beats/min, while oxygen saturation on room air decreased to 68%. Bilateral basal crackles were detected on pulmonary auscultation. Posteroanterior (PA) chest radiography demonstrated bilateral diffuse infiltrates, perihilar congestion, mild cardiothoracic enlargement, and blunting of the costophrenic angles (Figure 1). Arterial blood gas analysis revealed hypoxemia. The clinical and radiological findings were consistent with acute pulmonary edema.

Figure 1. PA chest radiograph obtained at the time of complication, demonstrating bilateral diffuse infiltrates, perihilar congestion, mild cardiothoracic enlargement, and blunting of the costophrenic angles.

Treatment

The 3% hypertonic saline infusion, considered the causative agent, was immediately discontinued. High-flow oxygen therapy was initiated, followed by non-invasive mechanical ventilation (BiPAP). Intravenous furosemide was administered to reduce volume overload and decrease preload. Due to the hypertensive state and elevated afterload, intravenous perlinganit (nitroglycerin) infusion was added at a rate of 20 mL/hour. With this combined therapeutic approach, the patient's respiratory distress gradually improved, oxygen requirements decreased, and hemodynamic stability was restored.

Follow-Up and Outcome

Follow-up PA chest radiography after treatment demonstrated marked clearing of the lung fields, substantial resolution of bilateral infiltrates, reduction of perihilar congestion, reopening of the costophrenic angles, and normalization of the cardiothoracic ratio (Figure 2).

Clinically, dyspnea and tachypnea resolved, bilateral crackles disappeared, oxygen saturation on room air increased to >95%, and blood pressure returned to normotensive levels. The patient achieved complete clinical recovery and was admitted for further monitoring and management.

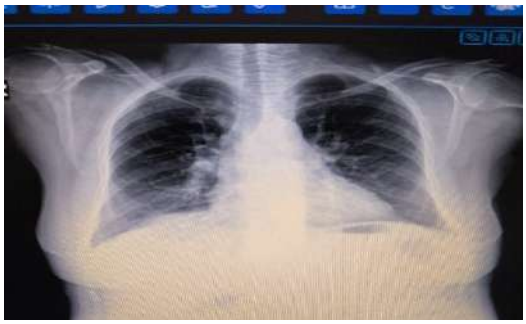

Figure 2. Follow-up PA chest radiograph after treatment showing marked clearing of the lung fields, substantial resolution of bilateral infiltrates, open costophrenic angles, and normalization of the cardiothoracic ratio.

3. DISCUSSION

3.1. Pathophysiological Framework

The development of acute pulmonary edema following hypertonic saline administration can be explained by a four-step mechanism: (i) administration of 3% NaCl creates an osmotic load within the intravascular compartment; (ii) water shifts from intracellular and extracellular spaces into the intravascular space along the osmotic gradient; (iii) rapid intravascular volume expansion increases left ventricular filling pressure and pulmonary capillary pressure, resulting in significant increases in preload and afterload; and (iv) in patients with limited cardiac and renal reserve, this process leads to alveolar fluid accumulation, hypoxemia, and dyspnea characteristic of acute pulmonary edema (4,5).

3.2. Risk Factors

In our patient, the coexistence of four important risk factors largely explains the development of this complication. First, advanced age (72 years) was associated with reduced cardiovascular reserve and decreased venous compliance. Second, chronic kidney disease limited the capacity for sodium and water excretion, thereby increasing susceptibility to volume overload. Third, a long-standing history of hypertension likely contributed to diastolic dysfunction and a lower threshold for pulmonary congestion. Fourth, the infusion rate and cumulative osmotic load may have exceeded the patient's physiological tolerance.

3.3. Comparison with the Literature

The findings of our case are consistent with several key studies addressing serious complications associated with hypertonic saline therapy. The classical formula proposed by Adrogué and Madias (2000) is widely used to estimate the expected effect of administered fluids on serum sodium levels; however, Mohmand et al. (2007), in a retrospective cohort study of 62 patients, demonstrated significant discrepancies between predicted and actual

clinical responses, particularly in hyponatremic patients with accompanying aquaresis, where risks of rapid correction and volume overload were increased (1,3). Maeda et al. (2007) reported marked systemic edema and pleural effusion in a patient with SIADH receiving prolonged hypertonic saline therapy; these findings regressed following discontinuation of therapy and fluid restriction (4). Thompson's classic report published in 1971 described acute pulmonary edema after intrathecal hypertonic saline administration, emphasizing that this complication may occur through different routes of administration (5). In a recent review, Rondon-Berrios and Sterns (2022) noted that administration of 100–150 mL boluses over 10–20 minutes significantly reduces the risks of overcorrection and volume overload compared with continuous infusion (2).

3.4. Unique Aspects of Our Case

Our case differs from previously reported cases in two important respects. First, unlike the case reported by Maeda et al., in which complications developed after 45 days of treatment, acute pulmonary edema occurred within only 3 hours of infusion in our patient. This observation demonstrates that serious complications may arise during the early stages of therapy, particularly in susceptible individuals. Second, the complication developed directly in the emergency department observation unit rather than during inpatient or intensive care follow-up. Consequently, early recognition and rapid intervention were critical determinants of the favorable outcome. Aggressive yet controlled treatment with NIV, intravenous furosemide, and perlinganit resulted in complete clinical recovery.

3.5. Clinical Implications

Four key clinical lessons can be drawn from this case. (a) When traditional continuous infusion is used instead of the bolus approach recommended by current guidelines, patient characteristics and infusion rate may predispose to this complication (2). (b) In elderly patients and those with CKD, monitoring of hemodynamic and pulmonary parameters should be considered equally important as monitoring serum sodium levels (3). (c) Acute dyspnea and hypertensive crisis occurring even during the first hours of hypertonic saline infusion should raise suspicion for pulmonary edema. (d) Once the complication develops, prompt recognition and controlled intervention using NIV, diuretics, and vasodilators are crucial determinants of outcome.

4. CONCLUSION

Although 3% hypertonic saline is a life-saving treatment option for symptomatic hyponatremia, it also carries substantial potential for adverse effects. This case once again demonstrates that treatment administered in elderly patients with chronic kidney disease may precipitate acute pulmonary edema and hypertensive crisis even within the first few hours of infusion. In emergency department practice, monitoring should extend beyond serum sodium levels to include blood pressure, oxygen saturation, respiratory rate, and overall clinical status. When complications occur, rapid stabilization can be achieved through immediate discontinuation of the causative agent, oxygen supplementation, non-invasive ventilation, intravenous diuretics, and vasodilator therapy when indicated. Our case further emphasizes the importance of wider adoption of the bolus approach recommended by current guidelines

and implementation of standardized monitoring protocols during hypertonic saline administration.

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The Relationship Between the C-Reactive Protein/Albumin Ratio and Length of Hospital Stay in Patients with Acute Cholecystitis

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Abstract

Objective:

Acute cholecystitis is one of the most commonly encountered inflammatory hepatobiliary diseases in the emergency department. In recent years, biomarkers reflecting both inflammation and physiological reserve have attracted attention in predicting clinical outcomes. This study aimed to evaluate the relationship between the C-reactive protein/albumin ratio (CAR) and the length of hospital stay in patients with acute cholecystitis

Materials and Methods:

A total of 27 patients who presented to the emergency department between February 15, 2026, and March 15, 2026, and were diagnosed with acute cholecystitis were retrospectively analyzed. Demographic data and laboratory parameters were recorded. Variables with normal distribution were expressed as mean ± standard deviation, while non-normally distributed variables were presented as median and interquartile range (IQR). The relationships between

length of hospital stay and laboratory parameters were evaluated using Spearman correlation analysis.

Study Design and Patient Selection
This study was designed as a retrospective observational study. Data were obtained by reviewing the medical records of patients who were hospitalized and followed with a diagnosis of cholecystitis within the last month at the Malatya Training and Research Hospital. Patients for whom hospitalization was indicated were included in the study, whereas those managed on an outpatient basis and not hospitalized were excluded.

This study was conducted to evaluate the relationship between clinical and laboratory parameters, as well as inflammatory indices, and length of hospital stay and mortality in patients hospitalized due to cholecystitis.

Demographic characteristics, length of hospital stay, surgical status, and laboratory results were obtained from patient files and the electronic hospital information system. Mortality status was assessed during the hospitalization period. Post-discharge data were not included in the analysis.

Results:
Of the 27 patients included in the study, 15 (55.6%) were male and 12 (44.4%) were female. The mean age of the patients was 52.9 ± 14.2 years. The median CRP level was 9.6 mg/L (IQR: 3.4–157), platelet count was 253 $\times 10^9$/L (IQR: 223–303), and CAR was 0.29 (IQR: 0.11–4.22).
Correlation analysis revealed a statistically significant positive relationship between CAR and length of hospital stay ($r = 0.455$, $p = 0.017$). Additionally, a positive correlation was found between CRP and length of hospital stay ($r = 0.465$, $p = 0.014$), while a negative correlation was observed between amylase levels and length of hospital stay ($r = -0.480$, $p = 0.011$).

Conclusion:
A significant relationship was found between the C-reactive protein/albumin ratio (CAR) and length of hospital stay in patients with acute cholecystitis. CAR may serve as a practical and easily calculable biomarker for assessing the clinical course in patients with acute cholecystitis.

Keywords:
Acute cholecystitis, C-reactive protein/albumin ratio, CAR, length of hospital stay, inflammation, prognosis

Introduction

Acute cholecystitis is a common hepatobiliary disease, most often associated with gallstones, and represents a significant portion of emergency department visits. The clinical spectrum can range from mild inflammation to severe infection and complications, with some patients requiring hospitalization [1,2]. Therefore, early determination of disease severity and

prediction of clinical outcomes, such as length of hospital stay, is crucial for effective patient management.

In the diagnosis and severity assessment of acute cholecystitis, various laboratory parameters are used alongside clinical findings. In particular, C-reactive protein (CRP) is considered an important marker of the inflammatory response and has been associated with disease severity [3]. However, in recent years, it has been suggested that combined indices reflecting both inflammation and physiological reserve may be more valuable for prognostic evaluation than a single biochemical parameter [4].

In this context, the C-reactive protein/albumin ratio (CAR) has emerged as an easily calculable biomarker that reflects both systemic inflammation and nutritional/physiological reserve. CAR has been reported to be associated with clinical outcomes and prognosis in various inflammatory and infectious diseases [5,6]. However, studies evaluating the relationship between CAR and clinical course in patients with acute cholecystitis are limited.

Length of hospital stay is considered an important indicator of disease severity and healthcare utilization. Therefore, assessing the relationship between inflammatory markers and length of hospital stay may provide valuable insight for clinical prediction. This study aimed to evaluate the relationship between CAR and length of hospital stay in patients presenting to the emergency department with a diagnosis of acute cholecystitis.

Materials and Methods

Between February 15, 2026, and March 15, 2026, a total of 27 patients who presented to the emergency department and were diagnosed with acute cholecystitis were retrospectively analyzed. Demographic data and laboratory parameters were recorded. Variables with a normal distribution were expressed as mean ± standard deviation, while non-normally distributed variables were presented as median and interquartile range (IQR). The relationships between length of hospital stay and laboratory parameters were evaluated using Spearman correlation analysis.

Study Design and Patient Selection

Our study was designed as a retrospective observational model. Data were obtained by reviewing the records of patients who were hospitalized and followed with a diagnosis of cholecystitis within the last month at the Malatya Training and Research Hospital. Patients for whom hospitalization was indicated were included in the study, while those managed on an outpatient basis and not hospitalized were excluded.

This study was conducted to evaluate the relationship between clinical and laboratory parameters, as well as inflammatory indices, and length of hospital stay and mortality in patients hospitalized due to cholecystitis.

Demographic data, length of hospital stay, surgical status, and laboratory results of the patients were obtained from patient files and the electronic hospital information system. Mortality was assessed during the hospitalization period, and post-discharge data were excluded from the analysis.

Statistical Analysis

The normality of continuous variables was assessed using the Shapiro–Wilk test. Descriptive statistics were presented as mean ± standard deviation for normally distributed variables and as median with interquartile range (IQR) for non-normally distributed variables. Categorical variables were expressed as counts (n) and percentages (%).

To evaluate the relationships between length of hospital stay and laboratory parameters, Spearman correlation analysis was performed, taking into account the distribution characteristics of the variables. Correlation coefficients (r) and significance levels were reported. A p-value of < 0.05 was considered statistically significant.

Results

Of the 27 patients included in the study, 15 (55.6%) were male and 12 (44.4%) were female.

Variables with a normal distribution were presented as mean ± standard deviation. The mean age of the patients was 52.9 ± 14.2 years, albumin level was 37.8 ± 8.1 g/L, neutrophil count was 6.84 ± 2.84 $\times 10^9$/L, lymphocyte count was 2.61 ± 1.24 $\times 10^9$/L, hemoglobin was 13.6 ± 1.82 g/dL, and monocyte count was 0.77 ± 0.26 $\times 10^9$/L (p-values > 0.05).

Non-normally distributed variables were presented as median with interquartile range (IQR). The median CRP level was 9.6 mg/L (IQR: 3.4–157), amylase was 45 U/L (IQR: 31–63), platelet count was 253 $\times 10^9$/L (IQR: 223–303), length of hospital stay was 0 days (IQR: 0–5 days), and CAR was 0.29 (IQR: 0.11–4.22) (p-values < 0.05). Details are provided in Table 1.

Table 1. Demographic and Laboratory Characteristics of the Study Group

Variables	n	p value	Mean	SD	Median	IQR	
						25%	75%
age(years)	27	0.935	52,8	14,2			
crp (mg/L)	27	<0.001			9,6	3,4	157
albumin (g/L)	27	0.302	37,8	8,09			
neutrophile ($\times 10^9$/L)	27	0.262	6,84	2,84			
amylase(U/L)	27	0.009			45	31	63
lymphocites($\times 10^9$/L)	27	0.057	2,61	1,24			

plt (×10⁹/L)	27	0.01			253	223	303
hgb (g/dL)	27	0.9	13,6	1,82			
monocytes(×10⁹/L)	27	0.417	0,77	0,26	0,76	0,58	0,92

Correlation Analysis

Correlation analysis was performed to evaluate the relationships between length of hospital stay and laboratory parameters. A moderate and statistically significant positive correlation was found between length of hospital stay and CAR ($r = 0.455$, $p = 0.017$).

Similarly, length of hospital stay showed a significant positive correlation with CRP ($r = 0.465$, $p = 0.014$). In contrast, a significant negative correlation was observed between amylase levels and length of hospital stay ($r = -0.480$, $p = 0.011$).

For other variables, no statistically significant correlations were found with length of hospital stay: age ($r = 0.088$, $p = 0.661$), albumin ($r = -0.304$, $p = 0.123$), neutrophils ($r = 0.097$, $p = 0.630$), lymphocytes ($r = -0.329$, $p = 0.093$), platelets ($r = -0.277$, $p = 0.162$), hemoglobin ($r = -0.001$, $p = 0.998$), and monocytes ($r = 0.072$, $p = 0.721$).

Discussion

In this study, the relationship between the C-reactive protein/albumin ratio (CAR) and length of hospital stay was evaluated in patients hospitalized with a diagnosis of acute cholecystitis. A statistically significant moderate positive correlation was found between CAR and length of hospital stay. This finding supports the notion that combined biomarkers reflecting both inflammation and physiological reserve may be valuable in predicting the clinical course.

Acute cholecystitis is a clinical condition characterized by a pronounced inflammatory response, with systemic inflammatory markers increasing as disease severity rises [1]. In this context, CRP is widely used as an acute-phase reactant and has been shown in numerous studies to correlate with disease severity. In our study, a positive correlation was also observed between CRP and length of hospital stay, consistent with the existing literature. However, a key limitation of CRP is that, while it reflects inflammation, it does not provide information about the patient's nutritional status or physiological reserve.

n recent years, combined indices that assess both the inflammatory process and the patient's nutritional and physiological status have gained prominence. CAR, calculated as the ratio of CRP to albumin, is a practical parameter reflecting both inflammation and protein reserve [10]. Albumin levels serve not only as an indicator of nutritional status but also as an indirect marker of systemic inflammation, tending to decrease during inflammatory processes. Therefore, CAR is thought to provide a more comprehensive reflection of disease severity compared to CRP alone [11].

The literature reports that CAR is associated with mortality and prognosis in sepsis, malignancies, and various inflammatory diseases [12]. However, studies specifically focusing

on acute cholecystitis are limited. In our study, the positive correlation observed between CAR and length of hospital stay suggests that this biomarker may also be useful in predicting the clinical course in patients with acute cholecystitis. Additionally, the correlation coefficient being similar to that of CRP raises the possibility that, due to the small sample size, the potential superiority of CAR could not be clearly demonstrated.

Another notable finding in our study was the negative correlation between amylase levels and length of hospital stay. This may be explained by the fact that patients with mild biliary pancreatitis tend to present earlier and are monitored more closely, resulting in shorter hospital stays. However, larger series are needed to clarify the clinical significance of this observation.

No significant relationship was observed between other laboratory parameters and length of hospital stay. This finding supports the notion that, in diseases with a heterogeneous clinical course such as acute cholecystitis, a single parameter may be insufficient for prognostic assessment, and combined indices may provide greater value.

In conclusion, this study demonstrates a significant relationship between CAR and length of hospital stay in patients with acute cholecystitis. As an easily accessible and calculable biomarker, CAR may assist in predicting disease severity and hospital stay in clinical practice. However, confirmation of these findings requires larger, prospective, and multicenter studies.

Limitations

The main limitations of this study are its single-center design, small sample size, and retrospective nature. Therefore, the findings should be validated in larger patient populations through prospective studies

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A RARE CAUSE OF SCIWORA: ISCHEMIC STROKE

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Abstract

Introduction and Purpose: Spinal Cord Injury Without Radiographic Abnormality (SCIWORA) is a term used for cases showing clinical signs of spinal cord injury but without radiographic evidence after trauma. It is commonly seen after flexion-extension injuries. The diagnosis of SCIWORA is based on physical examination and MRI findings. This condition, which is quite rare in adults, should be considered in all patients presenting with post-traumatic neurological deficits but without any pathology seen on conventional radiography or computed tomography (CT).

Material and Methods: A 59-year-old female patient fell to the ground following vision loss in her left eye and presented to the emergency department with numbness and weakness in all four limbs, more pronounced in the lower limbs, following the fall. Radiological imaging was

ordered for the patient. The brain CT was normal, and there was no fracture or subluxation on cercival CT. Diffusion-weighted magnetic resonance imaging (MRI) was planned for the patient, who was suspected of having an ischemic stroke, and diffusion restriction was observed in the right occipital region. Neurology and neurosurgery consulted were placed for the patient. Since the patient's clinical condition could not be explained by the existing radiological images, cervical and thoracic MRI scans were performed on the patient. The patient's cervical MRI showed significant anterior compression of the spinal cord at the C4-C5 levels. The patient was hospitalized by the neurosurgery clinic.

Results and Conclusion: The diagnosis of SCIWORA is made based on physical examination and MRI. A careful neurological examination is crucial for assessing neurological deficits that may appear even several days after the injury. MRI can identify pathologies of nerve tissues, including intervertebral discs, the spinal cord, and nerve roots; therefore, it has become the gold standard imaging modality in patients with suspected spinal cord injury. Considering the neurological deficits that occur in these patients, SCIWORA should be included among the differential diagnoses, and spinal MRI should be considered.

Keywords: Ischemic stroke, SCIWORA, spinal MRI

Introduction

SCIWORA (Spinal Cord Injury Without Radiographic Abnormality) is a term that describes the clinical symptoms of post-traumatic spinal cord injury without radiographic abnormalities in spinal imaging, typically seen in childhood. The acronym SCIWORA was first used by Pang and Wilberger Jr. in a series of 24 children who had traumatic myelopathy but showed no signs of fracture, dislocation, or misalignment of the spine. When defined by Pang and Wilberger Jr. conventional radiographs and computed tomography (CT) scans showed no pathology despite the presence of post-traumatic neurological symptoms. When the term SCIWORA was first defined, magnetic resonance imaging (MRI), which is the imaging modality that distinguishes spinal cord injury, was not sufficiently developed and was therefore not included in the definition (1). Twenty years after his initial definition, Pang acknowledged the diagnostic potential of MRI (2). SCIWORA is most commonly seen in childhood and more frequently in the cervical spine. The causes of this condition include a large head-to-body ratio, greater mobility of the cervical spine, congenital ligamentous laxity, weak neck muscles, incomplete ossification of the vertebrae, and shallower opening of the facet joints. The most common injury mechanisms in the pediatric age group are motor vehicle accidents and sports injuries, while in the adult age group, the most common injury mechanism is falls. In the majority of patients, the cause of SCIWORA is traumatic flexion-extension injury (3). It is thought that during this injury, a temporary spasm occurs in the arteries supplying the spinal cord, leading to spinal infarction (4).The treatment approach for cases diagnosed with SCIWORA is generally based on conservative medical methods,

including immobilization and corticosteroid use (2). However, surgical intervention may be necessary if there are signs of instability associated with ligamentous injury and/or unresolved spinal cord compression (3). In this case, we aimed to contribute to the literature with an adult patient diagnosed with SCIWORA after a fall due to ischemic stroke.

Case

A 59-year-old female patient presented to the emergency department with numbness and weakness in all extremities, more pronounced in the lower extremities. The patient fell after experiencing vision loss in her left eye, and her symptoms began with this trauma. The patient's medical history included type 2 diabetes mellitus (DM), vision loss in the right eye due to cataracts, and difficulty walking secondary to a previous operation on the right knee. The patient, who had previously been able to walk with a walker, complained of difficulty moving both hands and inability to walk after the fall. The patient's vital signs upon arrival at the emergency department were arterial blood pressure 135/80 mmHg, pulse 86/minute, saturation 99%, and temperature 36.5 °C. Physical examination revealed that the patient was drowsy, oriented, and cooperative. The Glasgow Coma Scale (GCS) was calculated as 14. There was an ecchymosis measuring approximately 2×2 cm above the mandible mentum. Muscle strength was 4/5 in the proximal part of both upper extremities, 3/5 in the distal part, and 2/5 in both lower extremities; the patient was tetraparetic. Deep tendon reflexes (DTR) were diminished, and the bilateral Babinski reflex was absent. The patient's skin test was hypoesthetic below the umbilicus, and no other pathology was observed in the other physical examinations performed. In the patient's radiological imaging, cervical CT showed osteophyte formations at the anterior corners of the vertebral bodies, spondylosis findings, and flattening of the cervical lordosis, with no other pathology observed (Figure-1). The patient's brain CT scan was normal, but diffusion restriction was observed in the right occipital region on brain DWI (Figure-2). A neurology consultation was planned for the patient. However, since the central nervous system imaging studies could not explain a clinical picture such as tetraparesis, spinal cord injury was considered possible in the patient. A neurosurgery consultation was planned, and a cervical-thoracic MRI was performed. Cervical MRI revealed significant anterior compression of the spinal cord at the C4-C5 levels, narrowing of the cervical spinal cord caliber, and increased myelopathic signal intensity (Figure-3). Following the consultations, the patient was admitted to the neurosurgery clinic with diagnoses of SCIWORA and ischemic stroke.

Figure-1: Cervical CT image

Figure-1: Cervical CT image

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Figure-1: Cervical CT image

Figure-2: Diffusion restriction in the right occipital region on brain diffusion MRI

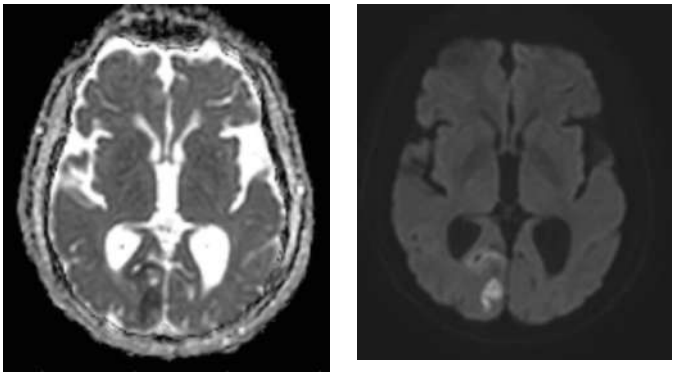
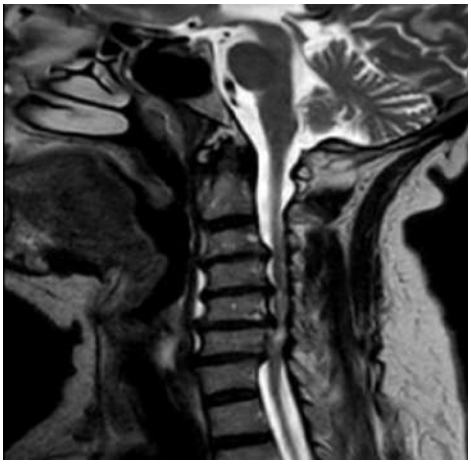


Figure-3: Significant anterior compression of the spinal cord at the C4-C5 levels on cervical



MRI

Discussion

SCIWORA was first described in 1982 by Pang and Wilberger Jr. using radiography and CT imaging (1). This pathology is more commonly seen in the pediatric age group. A report published by Yücesoy and Yüksel stated that the incidence of SCIWORA in the pediatric age group ranged from 3.3% to 32.0% (5). SCIWORA is rarely seen in adults. In a study by Tewari et al., 40 of 472 patients diagnosed with spinal cord injury were found to have SCIWORA due to cervical spine injury. In this study, it is found that 60% of cases are diagnosed with SCIWORA as a result of traffic accidents, 30% as a result of falls from heights, 7.5% as a result of work accidents, and 2.5% as a result of minor falls. Furthermore, the male-to-female ratio in this study was found to be 3.4:1 (6). Our case was a 59-year-old female patient who fell from the same level. According to the literature, spinal cord injury is quite rare in adult patients after minor falls.

SCIWORA affects the cervical spine more than the thoracic and lumbar spine, particularly due to the greater mobility of the cervical spine (3). In a study by Carroll et al. examining 368 pediatric patients with SCIWORA, it was found that 87% of patients had cervical involvement, 9.5% had thoracic involvement, and 1.5% had lumbar involvement (7). In our case, physical examination and imaging revealed spinal cord compression at the C4-C5 level of the cervical spine.

The diagnosis of SCIWORA is made based on physical examination and MRI evaluation. A careful neurological examination is crucial for assessing neurological deficits that may appear even several days after the injury. Therefore, it is important for physicians to be knowledgeable about this clinical picture to help them make the appropriate diagnosis and decide on the management of these rare cases (8). MRI can identify pathologies of nerve tissues, including intervertebral discs, the spinal cord, and nerve roots, and has therefore become the gold standard imaging modality in patients with suspected spinal cord injury (9). This, physical examination revealed tetraparesis, leading to the suspicion of spinal cord injury. After evaluation with MRI, the patient was diagnosed with SCIWORA.

Since SCIWORA does not present with obvious signs of spinal trauma such as fracture and dislocation, non-surgical strategies such as immobilization and corticosteroid treatment form the basis of treatment. However, surgical treatment may be necessary if there are signs of instability accompanied by ligamentous injury and/or unresolved spinal cord compression (3). Of 48 adult SCIWORA patients studied by Martinez-Perez et al., 14 underwent surgical treatment. Good neurological outcomes were seen in 86% of surgically treated patients and 76% of those receiving conservative treatment. However, statistical analyses showed no correlation between treatment method and neurological outcome (10).

Conclusion

SCIWORA is a condition that can be missed in emergency departments and difficult to diagnose due to the absence of any pathological findings on plain radiography and CT imaging. Emergency department physicians should consider advanced imaging methods and spinal MRI for the diagnosis of SCIWORA based on the patient's physical examination findings and pathological findings in the neurological examination.

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To investigate the clinical significance of the HALP score in pediatric patients with acute appendicitis

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Abstract

Aim:
To evaluate the relationship between the HALP score (hemoglobin, albumin, lymphocyte, platelet) and length of hospital stay in pediatric patients with acute appendicitis and to investigate the potential use of this score in predicting clinical course.

Materials and Methods:
This retrospective study includes pediatric patients with acute appendicitis who presented to the emergency department of Malatya Training and Research Hospital between September 15, 2025 and March 15, 2026. A total of 26 patients with sufficient clinical and laboratory data were included in the study. Demographic data, laboratory parameters (WBC, hemoglobin, albumin, neutrophil, lymphocyte, platelet, CRP, creatinine, urea, potassium), and length of hospital stay were obtained from the hospital record system. In statistical analysis, variables with normal distribution were presented as mean ± standard deviation, and non-normally distributed variables were presented as median and IQR. The relationship between HALP score and length of hospital stay was evaluated using Spearman correlation test; $p < 0.05$ was considered statistically significant.

Results:
The study population consisted of 26 patients, and the mean age was determined as 11.9 ± 0.7 years. A significant negative correlation was found between HALP score and length of hospital stay ($r = -0.42$, $p = 0.032$). Although no significant relationship was found when the components of the HALP score were evaluated individually, the composite score was observed to be associated with clinical outcomes. Additionally, a negative correlation was detected between hemoglobin level and length of hospital stay. These findings support that the HALP score has a stronger prognostic potential than single parameters, as it simultaneously reflects inflammation and nutritional status.

Conclusion:
A significant negative relationship was found between HALP score and length of hospital stay in pediatric patients with acute appendicitis. The HALP score may be considered as a simple, inexpensive, and easily applicable biomarker in predicting the clinical course of the disease. However, larger sample-sized, multicenter, and prospective studies are needed to confirm these findings.

Keywords:
Pediatric acute appendicitis, HALP score, Length of hospital stay, Inflammatory markers, Prognostic biomarker

Introduction

Acute appendicitis is one of the most common surgical emergencies in childhood and constitutes an important cause of acute abdomen in the pediatric age group [1,2]. Despite advances in diagnosis and treatment, complications such as perforation, intra-abdominal abscess, and prolonged hospital stay still remain among the significant causes of morbidity [3]. Therefore, reliable biomarkers that can predict disease severity and clinical course are needed in pediatric patients with acute appendicitis [4].

In recent years, the use of various hematological parameters reflecting systemic inflammation and nutritional status as prognostic markers has been increasing. One of these parameters, the HALP score, is a biomarker calculated from hemoglobin, albumin, lymphocyte, and platelet values and evaluates both the inflammatory and nutritional status of the patient simultaneously [5]. The HALP score was first defined as a prognostic marker in oncological diseases, and in recent years, its relationship with clinical outcomes has begun to be investigated in various inflammatory and surgical diseases [6].

The pathophysiology of acute appendicitis includes processes of inflammation, immune response, and tissue damage. Therefore, hematological indices reflecting the inflammatory response are thought to be associated with the clinical course of the disease [7]. In particular, length of hospital stay is an important clinical parameter that reflects both disease severity and the burden on the healthcare system [8].

However, in the current literature, studies evaluating the relationship between HALP score and clinical outcomes in pediatric acute appendicitis patients are quite limited. In particular, data examining the relationship between HALP score and length of hospital stay are insufficient [9]. In this study, it was aimed to evaluate the relationship between HALP score and length of hospital stay in patients treated with a diagnosis of pediatric acute appendicitis. Demonstrating the usability of the HALP score in predicting clinical course may contribute to risk stratification and clinical management of pediatric acute appendicitis patients.

Materials and Methods

This study was conducted retrospectively to evaluate pediatric patients with acute appendicitis who presented to the emergency department of Malatya Training and Research Hospital between September 15, 2025 and March 15, 2026. Patients who presented to the emergency department with abdominal pain and were diagnosed with acute appendicitis within the specified date range were screened through the hospital record system.

A total of 33 pediatric acute appendicitis patients were identified during the study period. Patients diagnosed with acute appendicitis who presented to the emergency department were included in the study. Patients with missing clinical or laboratory data and those whose data could not be accessed were excluded from the study. In line with these criteria, 26 patients were included in the study.

Demographic characteristics of the patients (age, length of hospital stay) and laboratory parameters [C-reactive protein (CRP), albumin, neutrophil, lymphocyte, WBC, platelet, hemoglobin, creatinine, urea, potassium] were obtained from the hospital electronic record system.

Statistical Analysis
The normality of data distribution was evaluated using the Shapiro–Wilk test. Variables with normal distribution were presented as mean ± standard deviation, while non-normally distributed variables were presented as median (interquartile range, IQR). In comparisons between groups according to gender, the Independent Samples t-test was used for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Statistical significance level was accepted as $p < 0.05$.

Results

A total of 26 pediatric acute appendicitis patients were included in the study. The mean age of the patients was found to be 11.9 ± 0.7 years. Whether laboratory values and length of hospital stay showed normal distribution was evaluated by statistical analyses, and p values are summarized in Table 1.

White blood cell (WBC), neutrophil, and CRP values were not normally distributed statistically ($p < 0.001$) and reflect the inflammatory response. The HALP score showed borderline non-normal distribution ($p = 0.032$). Hemoglobin (HB), age, lymphocyte, urea, and length of hospital stay showed normal distribution ($p > 0.05$). Creatinine, albumin, and potassium values were also not normally distributed ($p < 0.05$).

Variables with normal distribution were presented as mean ± standard deviation, and non-normally distributed variables were presented as median and interquartile range (IQR). Descriptive statistics are shown in Table 1.

(Table 1: Demographic and laboratory data of pediatric acute appendicitis patients)

VARİABLES	n	p value	mean	SD	median	IQR	
						25%	75%
Age(years)	26	0,231	11,9	0,7			
WBC(×10³/µL)		<0,001			14,6	11,3	21,3
Hemoglobin(g/dL)		0,315	13,1	0,2			
Neutrophil(×10³/µL)		<0,001			12,3	9,4	18
Lymphocyte(×10³/µL)		0,137	1,3	0,1			
HALP		0,032			0,2	0,1	0,2
Urea(mg/dL)		0,298	27,2	1,3			
Creatinine(mg/dL)		0,006			0,6	0,5	0,6
Albumin(g/dL)		0,001			4,3	3,8	4,6
Platelet(×10³/µL)		0,086	313	11			
CRP(mg/L)		<0,001			2,2	0,3	5,5
Potassium(mmol/L)		0,026			4	3,8	4,3
HOSPITALTY(day)		0,203	3,7	0,3			

When the relationship between length of hospital stay and our quantitative data was examined, a negative but statistically non-significant relationship was found with lymphocyte, albumin, and platelet (p and r values respectively; p=0.310 r= -0.207; p=0.797 r= -0.053; p=0.877 r= -0.032).

A statistically significant negative relationship was found between HALP score and hemoglobin with length of hospital stay (p and r values respectively; p=0.0002 r= -0.579; p=0.0001 r= -0.480).

Discussion

Acute appendicitis is one of the most common surgical emergencies in childhood and constitutes an important cause of acute abdomen in the pediatric age group [1,2]. The mean age of the 26 pediatric appendicitis cases examined in our study was 11.9 ± 0.7, which is consistent with the literature [10]. This situation is explained by the atypical course of clinical findings and communication difficulties in younger children.

Our findings also support these data, and it was observed that the frequency of admission increases especially in the older age group. This may be associated with the development of lymphoid tissue with age and increased susceptibility to obstruction.

In our study, the relationship between HALP score and length of hospital stay was evaluated in patients diagnosed with pediatric acute appendicitis, and a significant negative correlation was found between HALP score and length of hospital stay. In previous studies, negative

relationships have also been observed between HALP score and hospital stay, mortality, and morbidity [11]. This suggests that patients with higher HALP scores may have a milder clinical course and shorter hospital stays.

Acute appendicitis is a clinical condition characterized by a prominent inflammatory response, and there is a close relationship between disease severity and systemic inflammation level. Therefore, in recent years, the use of hematological parameters reflecting inflammation in diagnosis and prognosis determination has increased [12]. In previous studies, parameters such as neutrophil, lymphocyte, and CRP have been shown to be associated with disease severity. In our study, the non-normal distribution and high variability of WBC, neutrophil, and CRP values support the prominence of the acute inflammatory process.

The HALP score, as it includes hemoglobin, albumin, lymphocyte, and platelet values, is a comprehensive biomarker that reflects not only inflammation but also the nutritional and immune status of the patient [13]. Low hemoglobin and albumin levels indicate chronic inflammation and poor nutritional status; a decrease in lymphocyte count reflects immunosuppression, while changes in platelet count may reflect inflammatory activity. In this respect, the HALP score is thought to be a stronger prognostic indicator than single parameters.

The finding of a negative correlation between HALP score and length of hospital stay in our study is consistent with findings reported in other inflammatory diseases in the literature. The association of higher HALP scores with better clinical condition indicates that this score may indirectly reflect disease severity [14]. Especially in the pediatric patient group, where clinical evaluation may be difficult, the HALP score may be used as an objective marker.

An important finding in our study is that although there was no statistically significant relationship between albumin, lymphocyte, and platelet—components of the HALP score—and length of hospital stay, the HALP score, which includes these parameters, was found to be significantly associated with length of hospital stay. This suggests that individual biomarkers may be insufficient to reflect the complex pathophysiology of the disease, whereas composite indices containing multiple parameters may be more powerful and sensitive in predicting clinical course. The simultaneous evaluation of inflammation, immune response, and nutritional status by the HALP score may be the main reason for this superiority.

In conclusion, a significant negative relationship was found between HALP score and length of hospital stay in pediatric acute appendicitis patients. The HALP score can be used as a simple, inexpensive, and easily applicable biomarker in predicting the clinical course of the disease. However, larger sample-sized and prospective studies are needed to confirm these findings.

Limitations

This study has some limitations. First, it was conducted as a single-center study and included a limited number of patients (n=26). This may limit the generalizability of the findings. In addition, the retrospective design of the study and the use of hospital record data may introduce potential biases such as missing data and recording errors.

Moreover, clinical parameters that could directly reflect the severity of acute appendicitis, such as perforation status, stage of appendicitis, and postoperative complications, were not included in the analysis. In addition, factors that may affect the components of the HALP score, such as nutritional status, comorbidities, and chronic inflammatory conditions, were not evaluated in detail.

Finally, the cross-sectional evaluation of the relationship between HALP score and length of hospital stay limits the ability to establish a causal relationship. Therefore, larger sample-sized, multicenter, and prospective studies are needed to confirm these findings.

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Rapid Diagnosis of Sudden Onset Chest Pain with bedside ultrasonography: A Case Report

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ABSTRACT

In our case, we presented a 47-year-old male patient with a hyperacute aortic dissection. We evaluated the role of bedside ultrasonography in the diagnosis of dissection and the diagnosis and treatment processes of these patients. The 47-year-old male patient, who was brought to the emergency department by emergency medical services (112) with sudden-onset back and chest pain. The patient stated that after eating, he suddenly felt as if a bomb had exploded in his epigastric region, followed by back and chest pain, which then radiated to his entire body. He noted that the pain decreased upon standing and increased when lying down. He had no medical history other than gastritis and was not on any regular medication. On physical examination, his abdomen was relaxed, breath sounds were normal,

and the neurological examination was unremarkable. The patient expressed that when he turned prone during the examination, the pain radiated to his chest. The patient's electrocardiogram showed a normal sinus rhythm, and on bedside ultrasonography, the aortic valve was measured at 60.4 mm at the valve level, and a bicuspid aortic valve with surrounding thrombus was observed. The patient's contrast-enhanced computed tomography revealed a dissection flap in the ascending aorta, aortic arch, descending thoracic aorta, abdominal aorta, and both main iliac arteries. The patient was consulted for cardiovascular surgery. The patient died during the intensive care unit follow-up after the aortic operation. Aortic dissection is a critical diagnosis in patients presenting with chest pain, carrying a high risk of death, and bedside ultrasonography is crucial for rapid diagnosis.

Key words: Aortic dissection, Chest pain, Ultrasonography.

INTRODUCTION

Chest pain is one of the most common reasons for presentation to the emergency department (1). There are many critical diagnoses in the differential diagnosis of chest pain. Aortic dissection is a cause of chest pain with high mortality. In our case, we presented a 47-year-old male patient with a hyperacute aortic dissection. We evaluated the role of bedside ultrasonography (USG) in the diagnosis of dissection and the diagnosis and treatment processes of these patients.

CASE

The 47-year-old male patient, who was brought to the emergency department by emergency medical services (112) with sudden-onset back and chest pain. His vital signs of the was measured as follows: blood pressure 160/90 mmHg in the right arm and 150/90 mmHg in the left arm, heart rate 76 beats/min, and oxygen saturation 97%. The patient stated that after eating, he suddenly felt as if a bomb had exploded in his epigastric region, followed by back and chest pain, which then radiated to his entire body. He noted that the pain decreased upon standing and increased when lying down. He had no medical history other than gastritis and was not on any regular medication. On physical examination, his abdomen was relaxed, breath sounds were normal, and the neurological examination was unremarkable. The patient expressed that when he turned prone during the examination, the pain radiated to his chest. The patient's electrocardiogram (ECG) showed a normal sinus rhythm, and on bedside USG, the aortic valve was measured at 60.4 mm at the valve level, and a bicuspid aortic valve with surrounding thrombus was observed (Figure 1). With a preliminary diagnosis of aortic dissection, a contrast-enhanced computed tomography (CT) scan was planned. The patient's contrast-enhanced CT revealed a dissection flap in the ascending aorta, aortic arch, descending thoracic aorta, abdominal aorta, and both main iliac

arteries. The aorta was measured as 60.6 mm at the valve level (Figure 2). Analgesia was administered to the monitored patient, and he was followed up by keeping his blood pressure within the target ranges. The patient was consulted for cardiovascular surgery. The patient died during the intensive care unit follow-up after the aortic operation.

DISCUSSION

Aortic dissection is a disease that we may encounter in the emergency department, which has a low in-hospital survival despite advancing medicine, and is often diagnosed postmortem (2). Currently, approximately one in every five patients dies despite receiving diagnosis and treatment (3). Aortic dissection occurs when blood passes through an intimal tear in the aorta into the aortic media, creating a false lumen. This condition causes longitudinal cleavage of the intima, creating a dissection flap that separates the true and false lumens (2,3). There are classifications based on the location and extension of the dissection. According to the Stanford classification, which is one of the most frequently used classifications, all aortic dissections involving the ascending aorta are classified as type A, and dissections originating distal to the innominate artery are classified as type B. Mortality rates are higher in type A patients (4). There are many factors in the etiology of aortic dissection, including hypertension, connective tissue diseases, congenital anomalies (most commonly a bicuspid aortic valve), and aortic aneurysms (5). Early treatment is crucial for aortic dissection; when clinical suspicion is high, treatment should be initiated before diagnosis is confirmed (6).

Although hospitalizations have been reported with a wide variety of symptoms, the most common symptom is pain, which is very severe and has a sudden onset. USG is an imaging modality used to diagnose aortic dissection. Bedside echocardiography performed on the patient can be supportive in the differential diagnosis of chest pain and provide rapid information about the aortic valve structure. However, its limited ability to visualize the entire aorta may not be sufficient for a definitive diagnosis. CT is more valuable for diagnosis due to its ease of access and high sensitivity (7).

In the emergency department management of patients with dissection, the control of pain, heart rate, and blood pressure is of great importance. The patients' heart rate should be maintained at around 60 beats/min, and the systolic blood pressure at 100-120 mmHg. The mortality of these patients increases by 1-2% with each passing hour (3).

In our case, a bicuspid aorta was present, and the diagnosis was made in our emergency department along with the diagnosis of dissection. Our patient's Stanford type A aneurysm was diagnosed using bedside USG and CT. Treatment was initiated directly upon

clinical suspicion, and diagnostic tests were performed as rapidly as possible. Although the patient was operated on the same day, he died.

CONCLUSION

Aortic dissection is a critical diagnosis in patients presenting with chest pain, carrying a high risk of death, and bedside USG is crucial for rapid diagnosis.

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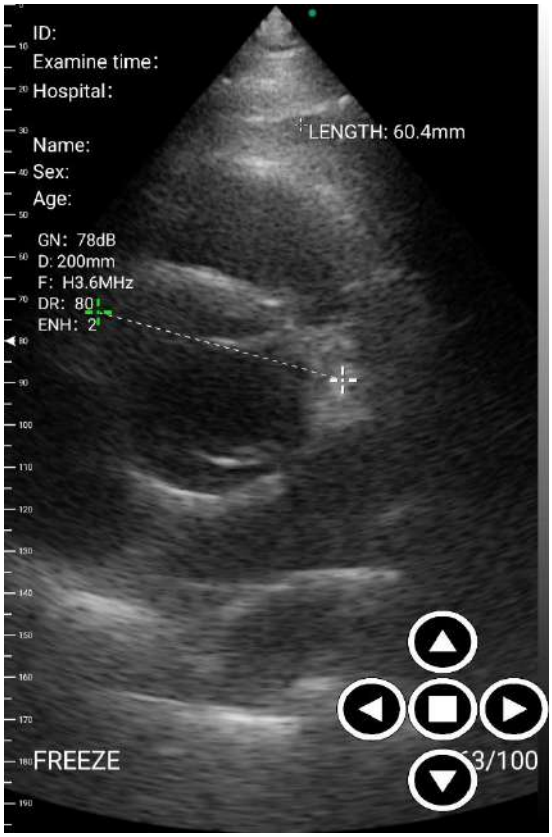

Figure 1. Thrombus appearance at the aortic valve level with TTE.

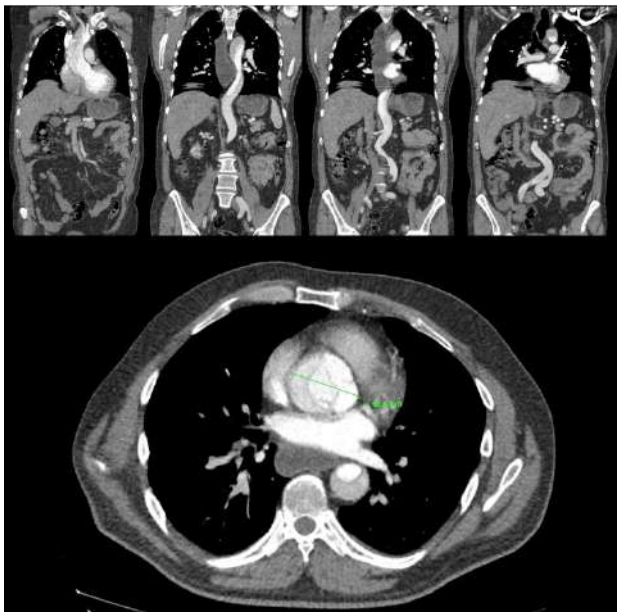

Figure 2. Dissection flap extending from the ascending aorta to the iliac arteries on contrast-enhanced CT.

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Myopericarditis in the Emergency Department: Navigating the Spectrum Between Pericarditis and Myocarditis

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INTRODUCTION

Acute pericarditis is characterized by inflammation of the pericardium and most commonly develops following viral infections (1). Clinically, the most prominent finding is positional chest pain. Worsening of pain while lying down and relief with sitting up or leaning forward are typical features (2).

Electrocardiography may reveal diffuse ST-segment elevations and PR-segment changes, which are helpful in diagnosis. However, in some patients, ECG findings may be minimal (2,3).

Cases accompanied by myocardial injury are referred to as myopericarditis and are typically indicated by elevated troponin levels (4). Myopericarditis represents a clinical spectrum between pericarditis and myocarditis, and distinguishing these conditions is important for accurate diagnosis and appropriate treatment.

CASE PRESENTATION

A 28-year-old male patient presented with chest pain that developed following upper respiratory tract infection symptoms persisting for approximately one week. The pain was sharp in nature and had positional characteristics; it decreased when leaning forward, worsened while lying down, and improved when sitting upright. The pain was not clearly associated with exertion.

On physical examination, vital signs were stable, and no significant pathological findings were detected on cardiovascular examination.

Electrocardiography demonstrated sinus rhythm with minimal ST-segment elevation in the lateral leads.

Laboratory investigations revealed elevated troponin levels of 738 ng/L and C-reactive protein (CRP) levels of 133 mg/L.

Echocardiographic evaluation showed a left ventricular ejection fraction of 60%, with no regional wall motion abnormalities or pericardial effusion. Valvular structures were assessed as normal.

Based on these findings, the patient was hospitalized in the cardiology department with a preliminary diagnosis of myopericarditis. Nonsteroidal anti-inflammatory therapy and colchicine were initiated. Due to the possibility of myocardial involvement, physical activity restriction and rest were recommended.

During hospitalization, the patient remained clinically stable, significant improvement in chest pain was observed, and no hemodynamic deterioration occurred. As no complications developed during follow-up, the patient was discharged on the second day of hospitalization with recommendations.

DISCUSSION

Acute pericarditis and myopericarditis are important causes of chest pain, particularly in young patients, and they frequently develop following viral infections (1). A history of upper respiratory tract infection is an important clinical clue in this context.

The most characteristic finding of pericarditis is positional chest pain. Pain that worsens while lying down and improves when leaning forward is explained by changes in pericardial friction related to pericardial inflammation (2,3). Similar clinical features in this case supported the diagnosis.

Although diffuse ST-segment elevations may be observed in pericarditis, ECG changes can be limited in some cases. In this patient, minimal ST-segment elevation in the lateral leads was interpreted in conjunction with clinical findings (2,3).

Elevated troponin levels indicate myocardial involvement, which is defined as myopericarditis (4,5). In cases of myopericarditis, left ventricular function is generally preserved and prognosis is usually favorable. In this patient, normal ejection fraction and the absence of regional wall motion abnormalities supported this observation.

Acute coronary syndrome must always be excluded in the differential diagnosis. Young age, positional chest pain, non-territorial ST changes, and normal echocardiographic findings favor the diagnosis of myopericarditis (1–4).

Nonsteroidal anti-inflammatory drugs and colchicine constitute the mainstay of treatment. Additionally, physical activity restriction and rest are important in patients with suspected myocardial involvement. Although prognosis is favorable in most cases, close follow-up is necessary since arrhythmias and cardiac dysfunction may rarely develop (2,4,5).

This case highlights that inflammatory causes should not be overlooked in the evaluation of chest pain in young patients.

CONCLUSION

In the evaluation of chest pain in young patients, not only acute coronary syndrome but also inflammatory causes such as pericarditis and myopericarditis should be considered. The combined evaluation of clinical, electrocardiographic, and biochemical findings is critical for accurate diagnosis and appropriate treatment.

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An Unexpected Peril in Treatment-Resistant Schizophrenia: Management of Late-Onset Clozapine-Induced Agranulocytosis and Septic Shock

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Introduction

Schizophrenia is a chronic and disabling disease for which antipsychotic drugs are the evidence-based first-line treatment. Clozapine, discovered over 65 years ago, is a second-generation antipsychotic and remains the only approved drug for treatment-resistant schizophrenia, significantly reducing mortality and suicide risk. However, many clinicians remain hesitant to prescribe clozapine due to its poorly understood mechanism of action and the potential for serious adverse reactions (1).

Despite its therapeutic advantages, clozapine use is strictly regulated because of life-threatening side effects, most notably idiosyncratic drug-induced agranulocytosis (2). The incidence of clozapine-induced agranulocytosis (CIA) ranges between 0.8% and 1.0% (1). Agranulocytosis is defined as a profound reduction in the absolute neutrophil count (ANC) to less than 500/mm³ (2). This condition leaves the host virtually defenseless against bacterial and fungal pathogens, often progressing rapidly from asymptomatic neutropenia to severe infections, pneumonia, and septic shock (3).

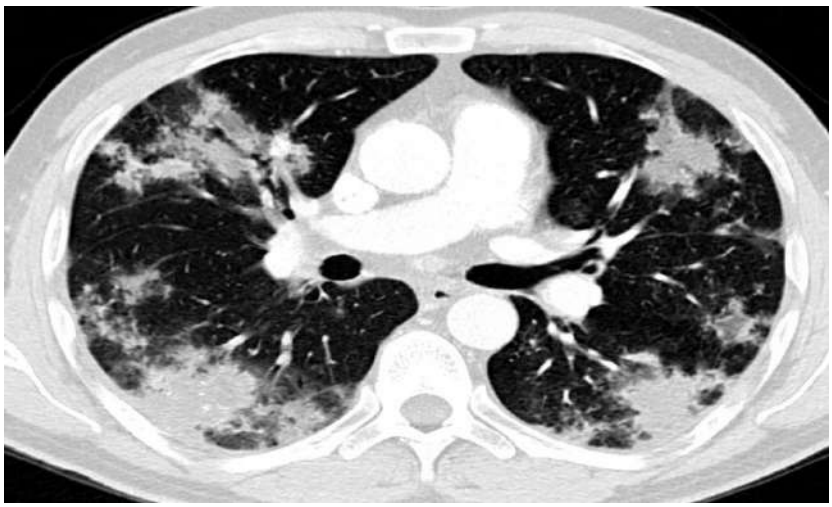
Patients typically present with "neutropenic fever," characterized by high temperatures, rigors, and severe oropharyngeal ulcerations, which are frequently the first clinical signs of impending septicemia (2). The management of these patients involves the immediate cessation of clozapine, aggressive fluid resuscitation, and the administration of empirical broad-spectrum antibiotics within the "golden hour" of sepsis management (4). Furthermore, hematopoietic growth factors, such as granulocyte colony-stimulating factor (G-CSF), have been shown to significantly shorten the duration of neutropenia and improve clinical outcomes (5).

Case Presentation

A 45-year-old male patient with a diagnosis of schizophrenia and an eight-month history of clozapine therapy presented to the emergency department with a three-day history of worsening fever, malaise, and sore throat. Medical history revealed that the patient had neglected his routine hematological monitoring for the past two months, developed anorexia over the last week, and experienced rigors within the 24 hours prior to admission.

Upon initial physical examination, the patient appeared toxic with a body temperature of 39.4°C, blood pressure of 84/52 mmHg, and a heart rate of 128 beats/minute. Oropharyngeal examination demonstrated widespread ulcerative lesions. Laboratory investigations were significant for a white blood cell count of 483/mm³, an absolute neutrophil count of 80/mm³, C-reactive protein of 281 mg/L, a lactate level of 4.5 mmol/L, and a procalcitonin level of 22 ng/mL. Thoracic computed tomography (CT) revealed patchy consolidation areas and peripheral infiltrates in both lungs (Figure-1), consistent with neutropenic infection.

Figure-1: Thoracic CT imaging: Bilateral infiltrates consistent with neutropenic infection



Based on these findings, the patient was admitted to the intensive care unit with diagnoses of clozapine-induced agranulocytosis and secondary septic shock. The offending agent was discontinued, and management was initiated with broad-spectrum antibiotics (carbapenem and vancomycin) along with granulocyte colony-stimulating factor (G-CSF) support. Following 16 days of aggressive treatment and close clinical monitoring, significant clinical and laboratory improvement was achieved, and the patient was discharged in stable condition.

Discussion

The management of CIA complicated by septic shock represents one of the most complex challenges in emergency medicine and critical care psychiatry (3). As demonstrated in the case of this 45-year-old male, the absence of protective neutrophils allows for a rapid and overwhelming systemic inflammatory response, often originating from the oral cavity or respiratory tract (6). This clinical course underscores several vital themes: the unpredictability of late-onset agranulocytosis, the diagnostic utility of biochemical markers in neutropenic sepsis, and the life-saving role of early G-CSF and intensive antimicrobial therapy (7).

A significant aspect of this case is that the patient developed agranulocytosis eight months after initiating clozapine. While classic epidemiological studies by Alvir et al. established that the vast majority of cases occur within the first eighteen weeks, this case reinforces that the risk, though diminished, never reaches zero (8). After the first year of treatment, the residual risk is estimated at 0.39 per 1,000 patient-years (9). Notably, elderly patients (aged 65 and older) have a significantly higher incidence, reported up to 7%, with a higher case-fatality rate (2).

The most critical step in managing CIA is the immediate and permanent discontinuation of the drug (4). Most patients show hematological recovery within one to three weeks after withdrawal (5). Continued use during the neutropenic phase is associated with a dramatic increase in fatal infections (3). In accordance with the Surviving Sepsis Campaign and IDSA

guidelines, our patient received intravenous carbapenem and vancomycin within the first hour (10). The administration of G-CSF was a key intervention, stimulating the survival and differentiation of neutrophil precursors and shortening the duration of severe neutropenia by three to five days (7). While its impact on overall mortality is still debated, G-CSF is strongly indicated for patients with an ANC < 100/mm³ or those presenting with sepsis and multiorgan failure (11).

Conclusion

Clozapine is a uniquely effective treatment for resistant schizophrenia, yet its association with life-threatening agranulocytosis necessitates constant clinical vigilance. This case is particularly significant because it demonstrates that severe agranulocytosis can occur as a late-onset complication, appearing eight months after the initiation of therapy. The patient's rapid progression to secondary septic shock following a lapse in routine blood monitoring underscores the vital importance of strict, long-term hematological surveillance. Ultimately, this case highlights that early diagnosis, the immediate and permanent discontinuation of clozapine, and the rapid administration of G-CSF alongside intensive antimicrobial therapy are life-saving interventions in the management of this rare but dangerous condition.

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A Silent but Dangerous Complication: Retroperitoneal Hemorrhage After Bone Marrow Biopsy

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INTRODUCTION

Bone marrow aspiration and biopsy are commonly used invasive diagnostic procedures in the diagnosis and follow-up of hematological disorders. The posterior iliac crest is the most frequently preferred site for this procedure due to its superficial location and ease of access to the bone marrow (1). Although bone marrow biopsy is generally considered safe, it carries a complication risk of approximately 0.05–0.12% (2).

The most common complications include local pain and minor hematomas, whereas serious vascular complications are extremely rare (2,3). However, these rare complications may lead to massive bleeding and hemodynamic instability, posing a life-threatening risk (3). In particular, injury to pelvic vessels may result in retroperitoneal hematoma (4).

In this report, we present a case of distal branch injury of the internal iliac artery during bone marrow biopsy, which was successfully managed with arterial embolization. The aim of this report is to emphasize the importance of early diagnosis and prompt intervention in reducing mortality associated with life-threatening complications following interventional procedures (3,5).

CASE PRESENTATION

A 69-year-old female patient was brought to the emergency department by ambulance after experiencing sudden onset groin pain and near-syncope at home following a bone marrow biopsy performed earlier that day.

On initial evaluation in the emergency department, the patient was somnolent but arousable, with a Glasgow Coma Scale (GCS) score of 14; cooperation and orientation were intact. Vital signs were as follows: blood pressure 90/56 mmHg, heart rate 45 beats/min, oxygen saturation 99% on room air, and capillary blood glucose 120 mg/dL.

Physical examination revealed tenderness in the lower abdominal quadrants and right iliac region. Distal pulses in both upper and lower extremities were palpable; however, capillary refill time was prolonged (4 seconds). No focal neurological deficits were observed. Other system examinations were unremarkable.

The patient's medical history included hypertension and atrial fibrillation. She also had a history of breast cancer, appendectomy, total abdominal hysterectomy, and bilateral salpingo-oophorectomy. Her regular medications included rivaroxaban, digoxin, bisoprolol, escitalopram, and trazodone. She was undergoing further evaluation for a preliminary diagnosis of polycythemia vera.

For differential diagnosis, electrocardiography (ECG), arterial blood gas analysis, complete blood count, liver and renal function tests, electrolytes, cardiac enzymes, and coagulation parameters were obtained. ECG revealed sinus bradycardia (heart rate: 45 bpm) without additional abnormalities. Initial hemoglobin was 13.5 g/dL and INR was 1.07. Follow-up hemogram showed a significant drop in hemoglobin (Hb: 9.9 g/dL). Other laboratory findings were within normal limits.

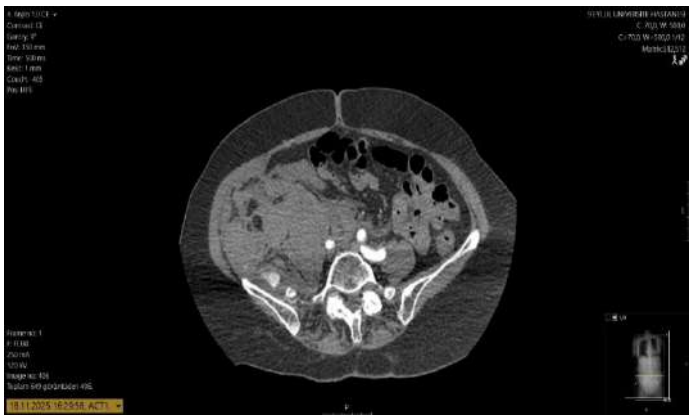
Focused bedside ultrasonography demonstrated a preserved ejection fraction (60%), inferior vena cava (IVC) diameter of 18 mm with normal collapsibility (40%), an aortic diameter of 28 mm, and no pericardial effusion. Free intraperitoneal fluid was detected in Morison's pouch.

After appropriate resuscitation and stabilization, computed tomography angiography (CTA) was performed to identify the bleeding source. Imaging revealed a large retroperitoneal hematoma at the level of the right iliopsoas muscle, along with active contrast extravasation originating from distal branches of the internal iliac artery. Additionally, a non-displaced fracture line was observed in the posterior aspect of the right iliac bone.

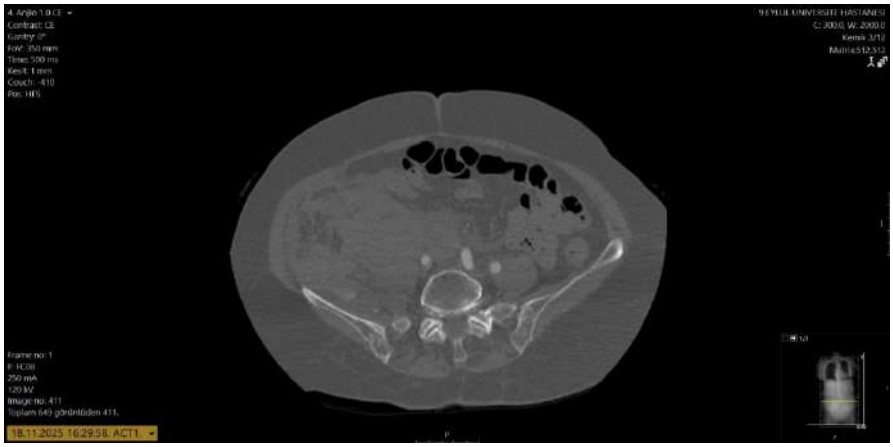
The patient was urgently consulted with cardiovascular surgery, orthopedics and traumatology, general surgery, and interventional radiology. Tranexamic acid was administered in the emergency department, and 3 units of packed red blood cells were transfused. Hemorrhage was successfully controlled via arterial embolization performed by interventional radiology.

Subsequently, the patient achieved hemodynamic stability. She was admitted to the hematology ward for monitoring and was discharged without any complications.

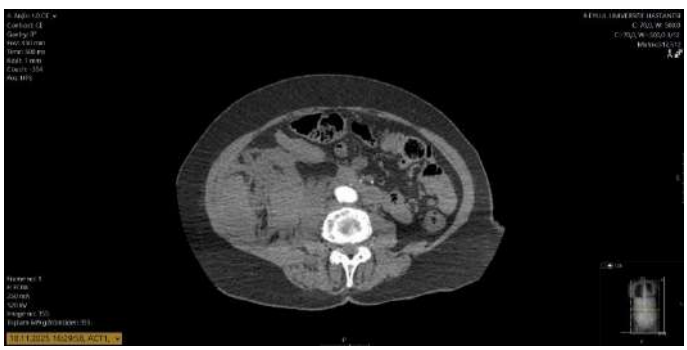
Resim-1: İliolumbar arterde gördüğümüz ekstravazasyon.



Resim-2: Sağ iliak kanatta gördüğümüz kırık hattı.



Resim-3: Retroperitoneal bölgede gördüğümüz yüksek dansiteli kanama ile uyumlu hematom.



DISCUSSION

Bone marrow aspiration and biopsy are widely used invasive procedures in the diagnosis and follow-up of hematological diseases and are generally considered safe (1,6). The posterior iliac crest is the most commonly preferred site due to its anatomical accessibility and low complication rate (1,6).

However, major complications, although rare, may occur, with an incidence reported between 0.05–0.12% (2). While common complications include local pain, minor bleeding, and small hematomas, retroperitoneal hemorrhage due to pelvic vessel injury is a rare but potentially fatal complication (3,4).

The posterior iliac crest is anatomically adjacent to branches of the internal iliac artery, including the superior gluteal and iliolumbar arteries. Therefore, deep or misdirected needle insertion may result in vascular injury (5,7). Previous reports have described complications such as pseudoaneurysm, arteriovenous fistula, and retroperitoneal hematoma following bone marrow biopsy (5,8).

In the present case, retroperitoneal hemorrhage due to distal internal iliac artery branch injury developed, with notably nonspecific initial symptoms. Symptoms such as sudden groin pain, syncope, or near-syncope may be misinterpreted as benign post-procedural discomfort, potentially delaying diagnosis (4,9). Therefore, in the presence of hypotension, unexplained hemoglobin drop, or new-onset pelvic pain following the procedure, occult retroperitoneal bleeding should be strongly suspected (9,10).

Another factor complicating clinical evaluation in this case was the patient's use of beta-blockers. The expected compensatory tachycardia in hypovolemic shock may be blunted, potentially delaying recognition of hemodynamic instability (11). Additionally, rivaroxaban use represented a significant risk factor for increased bleeding severity. It is important to note that normal INR and coagulation parameters do not exclude bleeding risk in patients receiving direct oral anticoagulants (12).

In terms of diagnostic approach, bedside ultrasonography is valuable for rapid assessment in the emergency setting; however, it has limited sensitivity for detecting retroperitoneal hemorrhage (13). Therefore, when clinical suspicion persists, computed tomography angiography remains the gold standard imaging modality for identifying active bleeding sources (10,13).

From a therapeutic perspective, in hemodynamically stable or stabilized patients, endovascular embolization is a less invasive and highly effective alternative to surgical

intervention (5,14). Selective arterial embolization enables rapid control of bleeding while reducing surgical morbidity (14,15).

In this case, hemorrhage was successfully controlled by embolization performed by interventional radiology, and no additional surgical intervention was required.

In conclusion, although bone marrow biopsy is generally safe, it may rarely result in life-threatening vascular complications. The possibility of retroperitoneal hemorrhage should be considered in patients presenting with new-onset pain, syncope, hypotension, or unexplained hemoglobin decrease after the procedure. Early clinical suspicion, appropriate imaging, and a multidisciplinary approach are crucial in reducing morbidity and mortality.

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Massive Pulmonary Embolism Mimicking Supraventricular Tachycardia: A Case Presenting with Syncope

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INTRODUCTION:

Syncope is defined as a transient loss of consciousness accompanied by loss of postural tone, characterized by a short duration and complete spontaneous recovery. It represents one of the clinical conditions frequently encountered in emergency department (ED) practice that requires careful evaluation in terms of both the diagnostic approach and the diversity of underlying causes [1,2]. This clinical condition is of particular importance in emergency medicine practice, especially because it requires rapid differentiation between benign and life-threatening underlying etiologies. Cardiac causes occupy an important and clinically high-risk etiology of syncope. These causes include both rhythm disturbances and structural heart diseases such as myocardial infarction or pulmonary embolism, as well as pathologies that impair circulatory outflow and lead to hemodynamic instability [1,2]. Cases of cardiac syncope are considered among the clinical conditions that require early diagnosis and rapid management because of the risk of sudden deterioration and mortality. Pulmonary embolism is not only a clinical entity that may cause syncope but also an important emergency condition that may present with various tachyarrhythmias and atrial rhythm disturbances, particularly sinus tachycardia on electrocardiography (ECG), and may therefore mimic different cardiac conditions during the initial evaluation [3,4]. In addition, the highly variable nature of its clinical presentation may lead to pulmonary embolism being overlooked during the diagnostic process. In this case report, we aimed to draw attention to the fact that pulmonary embolism may be the underlying primary etiology in a patient with syncope in whom a rhythm disorder was initially considered predominant, and to emphasize the importance of performing a comprehensive differential diagnosis in similar clinical presentations.

CASE PRESENTATION

A 26-year-old male patient who was referred to our hospital from an external center with a preliminary diagnosis of supraventricular tachycardia. The heart rate of the patients could not be controlled. It was learned that the patient had experienced syncope at home and subsequently presented to a district state hospital for initial evaluation. Detailed history revealed that he had experienced progressively increasing dyspnea for approximately one week, a marked decrease in exercise capacity, a gradually shortened walking distance, and

easier fatigability compared with his previous baseline. The vital signs of the patient are presented in Table 1. The initial ECG tracing is shown in Figure 1. After sinus tachycardia was detected on ECG, bedside echocardiography was performed for further evaluation of possible right heart strain and right ventricular involvement, and a D-sign was observed. When evaluated together with the existing clinical picture, this finding was considered an important sign supporting right ventricular pressure overload. As the preliminary diagnosis of pulmonary embolism became stronger with these findings, urgent computed tomography pulmonary angiography (CTPA) was performed. On CTPA, extensive filling defects extending into the lobar and segmental branches of both main pulmonary arteries were observed. These radiological findings were considered consistent with massive pulmonary embolism. In addition, the widespread distribution of thrombus burden was noteworthy as an important radiological feature indicating high clinical severity. A section from the CTPA scan is presented in Figure 2. The patient was consulted with the pulmonology department. After thrombolytic therapy was administered in the ED, the patient was admitted to the pulmonology intensive care unit for further monitoring and treatment.

DISCUSSION AND CONCLUSION

This case clearly demonstrates that, in patients presenting to the ED with similar symptoms and findings, different etiologies that may follow an atypical course and be life-threatening may also be present in addition to the diagnoses more commonly considered at first glance [1-4]. In the evaluation of relatively nonspecific clinical presentations such as syncope, tachycardia, and dyspnea, clinicians should not focus solely on the most prominent diagnosis, other critical causes that may explain the clinical picture should be systematically considered [1-4]. This approach is also important in terms of evaluating serious diagnoses without excluding them, particularly in young patients [1-4]. In conclusion, a systematic approach supported by detailed history-taking, careful physical examination, and appropriate imaging modalities is critically important for reaching the correct diagnosis in a timely manner, initiating appropriate treatment without delay, and preventing possible adverse clinical outcomes [1-4].

Table 1. The patient's vital signs on arrival

Blood Pressure	Heart Rate	Body Temperature	Fingertip Oxygen Saturation	Respiratory Rate
168/83 mmHg	135 beats/min	36.1 °C	99 %	24 breaths/min

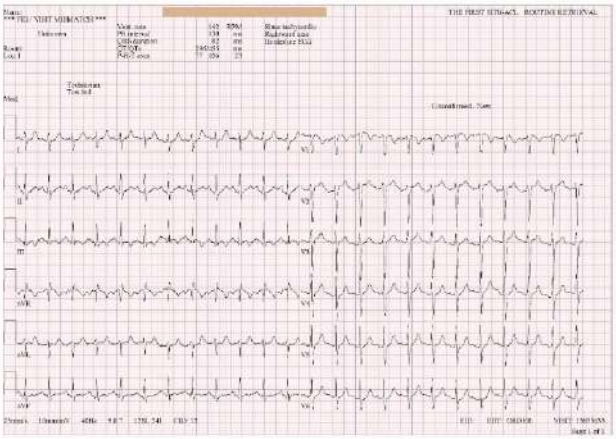

Figure 1. Electrocardiogram of the patient on presentation

Figure 2. Computed tomography pulmonary angiography of the patient

KEYWORDS: Differential diagnosis, emergency department, pulmonary embolism, sinus tachycardia, syncope

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Rectus Sheath Hematoma Mimicking Acute Abdomen in an Elderly Patient on Anticoagulation: A Case Report

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Introduction

Abdominal pain is one of the most common reasons for emergency department admissions and has a broad differential diagnosis spectrum (1). Although the majority of cases can be explained by gastrointestinal, hepatobiliary, or genitourinary system pathologies, less common musculoskeletal causes may also lead to this clinical presentation (1).

Rectus sheath hematoma (RSH) is a rare but clinically significant condition resulting from bleeding within the rectus abdominis muscle or its sheath (1,2). Due to its potential to lead to unnecessary surgical interventions when misdiagnosed, it should be considered in the differential diagnosis of acute abdomen (4).

RSH most commonly develops as a result of rupture of the epigastric vessels (1). The condition is frequently observed in elderly patients receiving anticoagulant therapy (3,5). However, trauma, severe coughing, sudden physical exertion, pregnancy, and previous abdominal surgery are also among the etiological factors (1,5).

Although the clinical presentation may vary, the most common symptom is abdominal pain. This may be accompanied by localized tenderness, abdominal wall masses, and occasionally skin ecchymosis (1,2). However, these findings may mimic acute abdominal conditions such as acute appendicitis, diverticulitis, or ruptured abdominal aortic aneurysm (6).

Imaging modalities play a crucial role in diagnosis. Ultrasonography is a rapid and accessible method; however, it may be limited in evaluating the extent of the hematoma (7). Contrast-enhanced computed tomography (CT) is considered the most reliable imaging modality for assessing the localization, size, and presence of active bleeding (4,9).

Case Presentation

An 80-year-old female patient presented to the emergency department with a complaint of abdominal pain persisting for approximately one week, predominantly localized to the right middle quadrant. The pain had progressively increased in recent days. There was no history of trauma, sudden physical exertion, or severe coughing.

The patient's medical history included hypertension, atrial fibrillation, vertigo, and a previous cerebrovascular event. She had been using rivaroxaban regularly due to atrial fibrillation.

Vital signs were as follows: heart rate 82 beats/min, blood pressure 110/60 mmHg, body temperature 37°C, respiratory rate 16 breaths/min, and oxygen saturation (SpO_2) 100%. The patient was in good general condition, conscious, oriented, and cooperative.

Abdominal examination revealed marked localized tenderness in the right middle and upper quadrants. Carnett's sign was positive. There were no signs of peritoneal irritation, and no palpable mass was detected.

Laboratory evaluation showed a hemoglobin level of 10.9 g/dL, and no significant decrease was observed in serial measurements.

Contrast-enhanced abdominal CT revealed a heterogeneous hematoma measuring 74 × 110 × 166 mm within the right rectus muscle. Additionally, a hematoma measuring 74 × 38 × 95 mm was observed within the right internal oblique muscle. The lesion extended from the level of the hepatic veins to the pelvis. A vascular structure consistent with active contrast extravasation was noted within the right rectus muscle.

These findings were evaluated as consistent with a Type II rectus sheath hematoma according to the Berna classification (Figure 1A–D).

The patient was consulted with general surgery, cardiology, and neurology departments. Conservative management was planned due to hemodynamic stability, and the patient was followed under observation.

Discussion

Rectus sheath hematoma is a rare but important clinical condition that can mimic acute abdomen (1,2). It has been reported that unnecessary surgical interventions may be performed in patients with misdiagnosis (4).

Risk factors for RSH include trauma, anticoagulant therapy, coughing, sudden physical exertion, and advanced age (1,5). In the literature, approximately 60–70% of patients diagnosed with RSH are reported to be receiving anticoagulant therapy (3).

Rectus sheath hematoma remains underdiagnosed in emergency settings due to its ability to mimic intra-abdominal pathology, particularly in elderly patients receiving anticoagulant therapy (1,5).

RSH may be confused with various conditions causing acute abdominal pain. The differential diagnosis should include acute appendicitis, diverticulitis, abdominal wall abscess, incarcerated hernia, and ruptured abdominal aortic aneurysm (6).

A positive Carnett's sign on physical examination is an important indicator suggesting abdominal wall-origin pain (1).

Contrast-enhanced CT is the gold standard for diagnosis and has high sensitivity in demonstrating the extent of the hematoma and active bleeding (4,9).

According to the Berna classification, Type I hematomas are small and confined within the muscle, Type II hematomas are intramuscular but extend along the fascia, and Type III hematomas are large and extend into the preperitoneal space (9).

The treatment approach depends on the patient's hemodynamic status. Conservative management is usually sufficient in stable patients, whereas interventional radiology or surgical treatment may be required in cases of active bleeding or hemodynamic instability (5,8).

Early recognition of rectus sheath hematoma is crucial for emergency physicians. This condition should always be considered in the differential diagnosis, especially in elderly patients on anticoagulant therapy presenting with localized abdominal pain (1).

Conclusion

Rectus sheath hematoma is a rare but significant clinical condition that can mimic acute abdomen (1). It should be considered in the differential diagnosis, particularly in elderly patients receiving anticoagulant therapy who present with sudden or progressively worsening abdominal pain. Early diagnosis with contrast-enhanced CT plays a critical role in patient management by preventing unnecessary surgical interventions (4).

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FİGURE 1 (A-B)

FİGURE 1 (C-D)

Figure 1 (A–D)

In contrast-enhanced abdominal CT sections, a large hematoma is observed within the right rectus abdominis muscle. The hematoma extends inferiorly along the abdominal wall, and active contrast extravasation is noted in some sections. The findings are consistent with a Type II rectus sheath hematoma according to the Berna classification.

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Palatal Necrosis: A Silent-Onset Fatal Infection

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Abstract

Objective

Mucormycosis is a rapidly progressive, angioinvasive fungal infection with high mortality, particularly in the setting of uncontrolled diabetes (1,2). Early clinical findings are often nonspecific, leading to delays in diagnosis. In this case report, a patient with rhino-orbito-palatal mucormycosis presenting with palatal necrosis, cranial neuropathy, and orbital involvement is described.

Case

A 51-year-old female patient with a 10-year history of type 2 diabetes mellitus and severely uncontrolled metabolic status (HbA1c 15.7%) presented to the emergency department with a two-week history of right hemifacial pain, ulcerative lesion on the hard palate, and recent onset of ptosis and decreased vision. Physical examination revealed palatal necrosis, right hemifacial hypoesthesia, and severe ophthalmoplegia. Laboratory findings included marked hyperglycemia (387 mg/dL), ketonuria (>160), leukocytosis ($16.2\times10^3/mm^3$), and thrombocytosis ($451\times10^3/mm^3$). Contrast-enhanced maxillofacial and orbital computed tomography demonstrated invasive soft tissue involvement extending from the nasal cavity and maxillary sinus to the medial extraconal orbital fat. Histopathological examination of palatal and nasal biopsies revealed broad, non-septate fungal hyphae on PAS and GMS staining, confirming invasive mucormycosis. Liposomal amphotericin B therapy was initiated. The patient was referred to a tertiary care center for advanced surgical evaluation, where treatment was continued.

Conclusion

In patients with uncontrolled diabetes, the coexistence of palatal necrosis, cranial neuropathy, and ophthalmoplegia is a critical warning sign for invasive mucormycosis. Early recognition in the emergency department, rapid imaging, histopathological confirmation, and prompt initiation of antifungal therapy are key determinants of prognosis.

Keywords

Mucormycosis, Rhino-orbital infection, Palatal necrosis, Cranial neuropathy, Diabetes

Introduction

Mucormycosis is an angioinvasive fungal infection caused by filamentous fungi of the order *Mucorales*, characterized by rapid progression and high mortality, particularly in patients with uncontrolled diabetes and diabetic ketoacidosis (1–3). The most common clinical form is rhino-orbito-cerebral mucormycosis, in which infection typically originates in the nasal cavity or paranasal sinuses and may extend to the orbit, cranial nerves, and intracranial structures (5,6). Vascular invasion leads to thrombosis and tissue ischemia, which are responsible for the characteristic necrotic clinical findings (2,3).

Early clinical findings are often nonspecific and may be mistaken for bacterial sinusitis or odontogenic infections. However, the presence of necrotic ulceration of the hard palate, necrotic nasal turbinates, facial sensory loss, and ophthalmoplegia are considered strong clinical indicators of invasive fungal infection (2,5).

The aim of this case report is to present a patient with uncontrolled diabetes who presented with palatal necrosis, cranial neuropathy, and orbital involvement due to rhino-orbito-palatal mucormycosis, and to emphasize early clinical warning signs and the importance of prompt recognition of this rare but fatal infection in the emergency department.

Case Presentation

A 51-year-old female patient with a 10-year history of type 2 diabetes mellitus presented to the emergency department with a two-week history of right hemifacial pain and swelling, painful lesion on the hard palate, and recent onset of nausea and vomiting. It was learned that she had previously received antibiotic therapy at an external center with a preliminary diagnosis of dental abscess; however, she had used the treatment irregularly and her symptoms had progressed.

On admission, the patient was conscious and cooperative. Vital signs included temperature 37°C, blood pressure 130/80 mmHg, respiratory rate 18 breaths/min, and heart rate 75 beats/min. She was hemodynamically stable.

Laboratory investigations revealed blood glucose 387 mg/dL, HbA1c 15.7%, hemoglobin 12.7 g/dL, platelet count $451\times10^3/mm^3$, and leukocyte count $16.2\times10^3/mm^3$. Urinalysis demonstrated significant ketonuria (>160). Arterial blood gas analysis showed pH 7.38, pCO_2 32 mmHg, and HCO_3^- 19 mmol/L.

Oral examination revealed an approximately 3×2 cm irregularly bordered necrotic ulcerative lesion with crusting and areas of black eschar extending from the right hard palate to the junction of the hard and soft palate. Examination of the right nasal passage demonstrated pale and necrotic mucosa in the inferior and middle turbinates. Facial examination revealed right hemifacial edema, flattening of the nasolabial fold, and hypoesthesia on the right side of the face.

Ophthalmological evaluation showed ptosis and proptosis in the right eye, a fixed and dilated right pupil, and severe ophthalmoplegia in all directions. Decreased visual acuity was noted. These findings were consistent with orbital apex syndrome.

Contrast-enhanced maxillofacial and orbital computed tomography revealed heterogeneous soft tissue densities filling the right nasal cavity and maxillary sinus, extension into the ethmoid cells, infiltration of periantral fat planes, and involvement of the medial extraconal orbital fat. These findings were consistent with invasive fungal rhinosinusitis.

Biopsies were obtained from the necrotic inferior turbinate and palatal ulcer by the otorhinolaryngology department. Histopathological examination demonstrated broad, non-septate fungal hyphae on PAS and GMS staining, confirming invasive mucormycosis.

Insulin therapy was initiated for metabolic control, and liposomal amphotericin B (5 mg/kg/day intravenously) was started following infectious disease consultation. The patient was referred to a tertiary care center for advanced surgical evaluation, where treatment was continued.

Discussion

Mucormycosis is an invasive fungal infection with rapid clinical progression, and delays in treatment dramatically increase mortality (1,2). The rhino-orbito-cerebral form associated with uncontrolled diabetes is one of the most critical infections requiring early recognition in emergency medicine practice (5).

The key feature of the disease is its angioinvasive nature. Fungal hyphae invade the vascular wall, leading to thrombosis, tissue ischemia, and necrosis (2,3). This pathophysiological process results in characteristic clinical findings such as black eschar, palatal necrosis, and cranial nerve involvement.

In this case, one of the most striking findings was the necrotic ulceration of the hard palate. Palatal necrosis results from thrombosis of the palatine arteries and subsequent local ischemia and is considered a highly specific clinical finding for rhino-orbito-cerebral mucormycosis in the literature (2,4).

However, in clinical practice, it is often misdiagnosed as a dental abscess or odontogenic infection, leading to delays in diagnosis. Therefore, the presence of palatal necrosis in patients with uncontrolled diabetes should be regarded as a strong warning sign for invasive fungal infection.

The right hemifacial hypoesthesia observed in this case suggests involvement of the maxillary branch of the trigeminal nerve. Additionally, ptosis, fixed-dilated pupil, and severe ophthalmoplegia are consistent with orbital apex syndrome. Orbital apex involvement indicates the spread of infection to neurovascular structures within the orbit and represents a critical stage associated with the risk of intracranial complications (5,6).

Imaging modalities provide important clues in the diagnosis of mucormycosis. Computed tomography findings such as sinus opacification, infiltration of periantral fat planes, and heterogeneity of orbital fat are characteristic of invasive fungal rhinosinusitis (6).

However, since bone destruction may not always be evident in early stages, careful evaluation of soft tissue planes is essential. Therefore, in the presence of clinical suspicion, imaging findings must be interpreted cautiously and the diagnosis should be confirmed by biopsy.

The cornerstone of mucormycosis treatment includes rapid correction of underlying metabolic abnormalities, systemic antifungal therapy, and surgical debridement. Current guidelines recommend liposomal amphotericin B as first-line antifungal therapy (1). However, antifungal therapy alone is often insufficient, and surgical debridement is one of the most critical determinants of prognosis (1,2).

Conclusion

The combination of palatal necrosis, cranial neuropathy, and ophthalmoplegia should be considered a critical clinical warning sign for invasive mucormycosis in patients with uncontrolled diabetes. Early recognition of these findings in the emergency department and prompt initiation of antifungal therapy are essential for improving patient outcomes (1).

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Figure 1: Ptosis and facial asymmetry of the right eye; clinical appearance suggestive of orbital involvement and cranial nerve dysfunction.

Figure 2: Irregularly bordered, crusted necrotic ulcerative lesion at the junction of the hard and soft palate.

Figure 3: Inferior view of palatal necrosis demonstrating a characteristic black eschar typical of invasive mucormycosis.

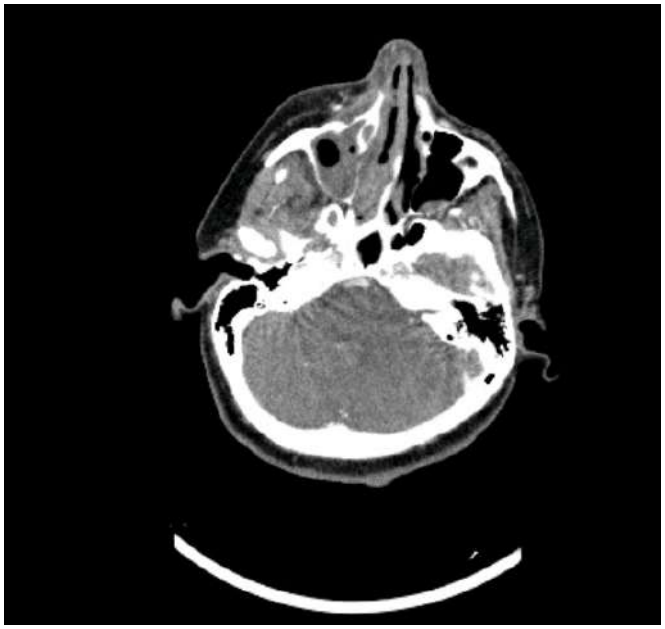

Figure 4A-B: Contrast-enhanced CT images show invasive sinonasal soft tissue disease extending from the maxillary sinus into the orbit through periantral fat infiltration, without early bone destruction. This pattern is characteristic of angioinvasive fungal infection such as mucormycosis, where soft tissue invasion precedes osseous involvement.

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Determinants of mortality in methanol intoxication cases and evaluation of a novel prognostic score

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Abstract:

Objective: Methanol intoxication is a toxicological emergency characterized by multisystemic involvement, where delays in diagnosis and treatment can lead to fatal outcomes. In this study, clinical, biochemical, and radiological parameters predictive of mortality in cases

presenting to the emergency department due to methanol poisoning were evaluated in detail; additionally, the predictive performance and clinical applicability of the developed Methanol Prognostic Mortality Score (MPMS) were examined.

Materials and Methods: This retrospective, single-center cohort study included a total of 92 patients evaluated in the emergency department following methanol ingestion between 2020 and 2024. Patients' demographic characteristics (age, sex), clinical findings at presentation (level of consciousness, visual symptoms), laboratory parameters, inflammatory indices, coagulation profile, and neuroimaging findings were systematically recorded. Scores used in intensive care triage were examined, and the MPMS was developed. Cutoff values were determined using ROC curves.

Results: A male predominance (94.6%) and advanced age were found to be significantly associated with mortality in the study population. Altered level of consciousness and visual disturbances emerged as primary predictors in determining prognosis. A low Glasgow Coma Scale (GCS) score was identified as the most reliable clinical indicator of central nervous system involvement. In the mortality group, Acute Physiology and Chronic Health Evaluation (APACHE II) and Sequential Organ Failure Assessment (SOFA) scores were found to be significantly elevated; severe acidosis (pH <7.0), low bicarbonate levels, and high lactate levels showed a strong correlation with survival. Elevations in creatinine, troponin, amylase, and liver function tests were detected. Among inflammatory indices, high neutrophil-to-lymphocyte ratio (NLR) and low platelet-to-lymphocyte ratio (PLR) values were found to be strongly associated with mortality, and coagulation parameter abnormalities observed in severe cases were found to be associated with poor prognosis. Radiologically, putamen involvement was defined as the most characteristic finding specific to methanol intoxication and was evaluated as a guiding factor in determining mortality risk. The MPMS demonstrated high sensitivity and specificity by integrating clinical, biochemical, and radiological parameters. The AUC value of the score for predicting mortality was determined to be 0.939 (95% CI: 0.891–0.987; $p < 0.001$), and the optimal cutoff value was established as 2.5.

Conclusion: In methanol poisoning, advanced age, male gender, altered consciousness, visual disturbances, low GCS, severe acidosis, high lactate levels, radiological putamen involvement, and inflammatory indices are critical prognostic factors for predicting mortality. The developed MPMS can be used as a decision-support tool in patient management due to its ease of application in clinical practice, high predictive power, and integration into multidisciplinary decision-making processes.

Keywords: Methanol intoxication, Mortality, Score.

1. Introduction

Methanol intoxication is a toxicological emergency characterized by high mortality due to delays in diagnosis and treatment (1,2). Formaldehyde and formic acid, the toxic metabolites of methanol, cause multisystemic involvement and severe damage, particularly to the central nervous system and the visual system (3,4). Clinical findings are often nonspecific and may mimic alcohol intoxication, potentially leading to diagnostic delays (5). This situation makes the application of early diagnosis and appropriate treatment algorithms in the emergency department critical (6).

In recent years, methanol-related poisonings have been reported as mass outbreaks both in our country and worldwide, with high mortality rates drawing attention (7,8). In Turkey, the consumption of cologne and low-cost alcohol products is among the primary causes of methanol poisoning (8). Methanol intoxication is characterized by gastrointestinal, neurological, and ophthalmological findings that emerge after a latent clinical period. Visual disturbances and changes in consciousness stand out as critical prognostic indicators (9,10).

Pathophysiologically, formic acid—a metabolite of methanol—exerts toxic effects on the retina and basal ganglia, leading to blindness and neurological sequelae (11). Additionally, high anion gap metabolic acidosis and elevated lactate levels are strongly associated with mortality (12). Therefore, the early assessment of clinical and laboratory parameters is of vital importance in patient management.

Clinical and laboratory parameters predictive of mortality have been examined in only a limited number of studies in the literature, and there is a lack of data regarding the clinical applicability of prognostic scores in particular (12). In this context, the development of new scoring systems that can be used to predict mortality in cases of methanol intoxication could provide significant contributions as decision-support tools in clinical practice.

In this study, clinical, laboratory, and radiological parameters predictive of mortality were evaluated in cases presenting to the emergency department following methanol ingestion; additionally, the predictive power and clinical applicability of the developed MPMS were investigated.

2. Materials and Methods

2.1 Study Design and Data Set

This study was conducted using a retrospective, single-center cohort design. A total of 92 patients who presented to the emergency department of Konya City Hospital, Health Sciences University, following methanol ingestion between 2020 and 2024 were evaluated. Inclusion criteria included a history of methanol ingestion, clinical findings documented in

the emergency department, and the availability of laboratory and imaging data. Cases involving patients under 18 years of age, those with incomplete medical records, and those with confirmed ingestion of toxic alcohol other than methanol were excluded from the study.

A patient follow-up form was created for the patients included in the study. The form included the patient's name, surname, medical record number, age, gender, chief complaint, history, vital signs upon presentation, clinical findings at the time of presentation (level of consciousness, visual symptoms, GCS), laboratory parameters (blood gas, biochemistry, inflammatory markers, coagulation profile), and neuroimaging findings were systematically recorded. Additionally, the APACHE II and SOFA scores used in intensive care triage were examined and integrated with clinical and laboratory data to create the newly developed MPMS. Laboratory parameters included pH, bicarbonate, lactate, creatinine, liver function tests, troponin, amylase, and inflammatory indices NLR and PLR. The coagulation profile was evaluated based on activated partial thromboplastin time (APTT), prothrombin time (PT), and international normalized ratio (INR) values. In radiological examinations, putamen involvement and basal ganglia lesions were recorded as findings associated with mortality.

For this study, ethical approval was obtained from the Karatay University Faculty of Medicine Ethics Committee for Non-Pharmaceutical and Non-Medical Device Research under document number 96817 and decision number 2024/034. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki.

2.1. Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). The distribution of quantitative variables was assessed using the Kolmogorov–Smirnov test. Variables with a normal distribution were presented as mean ± standard deviation, while those without a normal distribution were presented as median (minimum–maximum). Categorical variables were summarized using frequency and percentage. For comparisons of quantitative variables, the independent samples t-test or the Mann–Whitney U test was used, depending on the distribution characteristics. For comparisons involving more than two groups, ANOVA was applied when assumptions were met, and the Kruskal–Wallis H test was used when they were not. When significant differences were found between groups, post-hoc analyses were used to identify the differing groups. Relationships between categorical variables were assessed using the chi-square test or, when appropriate, the Fisher's exact test. Relationships between variables were examined using Pearson or Spearman correlation analyses. For quantitative parameters with a significant association with mortality, ROC analysis was performed; discriminatory power was assessed using the AUC, and optimal cutoff values were determined using the Youden index. The predictive performance of the mortality score developed based on these cutoff

values was also evaluated using ROC analysis. In all analyses, a p-value of <0.05 was considered statistically significant.

3. Results

When examining the demographic characteristics of the patients in our study, the mean age of the 92 patients was 46.6 ± 14.77 years, and the time elapsed between exposure and presentation was found to be 25.4 ± 27.37 hours on average. Regarding gender distribution, 87 patients (94.6%) were male and 5 (5.4%) were female. The number of patients without a history of trauma was 90 (97.8%), while 2 (2.2%) had a history of trauma. There were 71 patients (77.2%) who had not consumed ethanol and 21 (22.8%) who had. Regarding chronic alcohol use, 86 patients (93.5%) had a history of chronic alcohol use, while 6 (6.5%) did not. Regarding the mode of presentation: 2 patients (2.2%) presented as outpatients, 40 patients (44.4%) were brought directly by ambulance, and 48 patients (53.3%) were transferred by ambulance. Among the presenting complaints, altered consciousness was the most common, reported by 34 patients (37.0%), blurred vision in 25 patients (27.2%), nausea and altered consciousness in 14 patients (15.2%), nausea and blurred vision in 10 patients (10.9%), and nausea and vomiting in 9 patients (9.8%). In the general condition assessment, 49 (53.3%) patients were recorded as good, 9 (9.8%) as moderate, and 34 (37.0%) as poor. Detailed data on the demographic parameters in our study are presented in Tables 1a and 1b.

Tables 1a and 1b. Descriptive statistics of patients' demographic parameters

a) Quantitative parameters

Variable	N	Mean	Std. Deviation	Median	Min.	Max.	p*
Age (years)	92	46.6	14.77	49	19	75	0.200
Time elapsed between exposure and presentation (hours)	89	25.4	27.37	18	1	168	<0.001

*: Kolmogorov-Smirnov test p-value

a) Qualitative parameters

Parameters	n	%

Gender	Male	87	94.60
	Female	5	5.40
History of trauma	None	90	97.80
	Present	2	2.20
Ethanol purchase	None	71	77.20
	Available	21	22.80
Chronic alcohol consumption	None	6	6.50
	Present	86	93.50
Application method	Outpatient	2	2.20
	Direct ambulance	40	44.40
	Ambulance dispatch	48	53.30
Complaint submission	Altered mental status	34	37.00
	Blurred vision	25	27.20
	Nausea + altered consciousness	14	15.20
	Nausea + vomiting	9	9.80
	Nausea + blurred vision	10	10.90
General condition	Poor	34	37.00
	Moderate	9	9.80
	Good	49	53.30

In our study, when patients' admission scores were evaluated, the mean GCS was 10.63 ± 5.45, and the median value was 15 (3–15). The mean APACHE II score was 15.1 ± 10.3, and the median value was 12 (1–44). The mean SOFA score was 4.65 ± 5.34, and the

median was 1 (0–19). When the length of stay in the intensive care unit was examined, the mean was 10.04 ± 38.01 days, and the median was 4 days (1–364).

In our study, when the patients' imaging findings were examined, computed tomography (CT) results showed normal findings in 77 patients (91.7%), while positive findings were observed in 7 patients (8.3%). When magnetic resonance (MR) findings were examined, normal findings were observed in 54 patients (65.1%), putamen involvement in 13 patients (15.7%), and other findings (basal ganglia outside the putamen, cerebellum, external capsule, parietal region, and diffuse involvement) were detected in 16 patients (19.3%).

Table 2. Descriptive statistics of imaging parameters for patients

Parameters		**n**	**%**
CT findings	Normal	77	91.70
	Involvement present	7	8.30
MRI findings	Normal	54	65.10
	Putamen involvement	13	15.70
	Other lesions*	16	19.30

CT: Computed tomography, MRI: Magnetic resonance imaging

Other involvement: basal ganglia other than the putamen, cerebellum, external capsule, parietal region, widespread involvement

When parameters associated with mortality were examined in our study, the strongest predictors were presenting symptoms, general condition, imaging findings, and treatment methods. The mortality rate was 61.8% in patients presenting with altered consciousness, whereas no mortality occurred in those presenting with nausea or blurred vision ($p<0.001$). Mortality was 73.5% in patients with poor general condition, whereas no mortality was observed in those with good general condition ($p < 0.001$). Mortality was significantly higher at 69.2% in patients with putamen involvement on MRI findings ($p < 0.001$).

Table 3. Binary comparisons of qualitative admission parameters according to mortality status

Variable	Category	Outcome		p*
		Mortality(-)	Mortality(+)	
		n(%)	n(%)	
Gender	Male	62 (71.3)	25 (28.7)	0.163*
	Female	2(40)	3(60)	
Ethanol intake	None	46 (64.8)	25(35.2)	0.067
	Present	18 (85.7)	3 (14.3)	
Type of application	Outpatient	2(100)	0(0)	0.238*
	Ambulance (primary)	31(77.5)	9(22.5)	
	Ambulance dispatch	30 (62.5)	18 (37.5)	
Chronic alcohol use	None	6 (100)	0(0)	0.172*
	Present	58(67.4)	28(32.6)	
Reason for visit	Nausea + vomiting	9(100)	0(0)	**<0.001***
	Blurred vision	25(100)	0(0)	
	Altered consciousness	13 (38.2)	21 (61.8)	
	Nausea + altered consciousness	9 (64.3)	5 (35.7)	
	Nausea + blurred vision	8 (80)	2 (20)	
General Condition	Poor	9(26.5)	25(73.5)	**<0.001***
	Moderate	6(66.7)	3(33.3)	
	Good	49(1)	0(0)	
CT findings	Normal	55 (71.4)	22(28.6)	0.195*

	Involvement present	3(42.9)	4(57.1)	
MRI findings	Normal	48 (88.9)	6 (11.1)	**<0.001***
	Putamen	4(30.8)	9(69.2)	
	Other	6 (37.5)	10(62.5)	

*: Fisher's exact test

CT: Computed tomography, MRI: Magnetic resonance imaging

When laboratory parameters were examined, the mean pH was 6.87 in the mortality group and 7.3 in the survivor group. Bicarbonate levels were 7.3 ± 3.29 mmol/L in the mortality group and 17.36 ± 6.97 mmol/L in the survivor group. Lactate levels were 8.7 mmol/L in the mortality group and 2.5 mmol/L in the survivor group; creatinine was 1.66 ± 1.34 mg/dL in the mortality group and 0.93 ± 0.32 mg/dL in the survivor group. Troponin levels were found to be significantly higher in the mortality group at 19.9 ng/mL, Amylase levels were 159.93 ± 118.52 U/L in the mortality group and 79.34 ± 56.15 U/L in the survivor group. In liver function tests, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels were significantly higher in the mortality group (AST: 71 U/L, ALT: 44 U/L)

Among inflammatory indices, the NLR was 3.23 in the mortality group and 3.91 in the survival group; the PLR was 100 in the mortality group and 164 in the survival group. When coagulation parameters were examined, the INR was 1.52 in the mortality group, 1.05 in the survivor group; APTT values were 48.5 seconds in the mortality group and 26.2 seconds in the survivor group.

The overall mortality rate was found to be 27% (n=25). Parameters significantly associated with mortality included: low GCS (<7), altered consciousness and visual disturbances, pH <7.06, bicarbonate <9.15 mmol/L, lactate >7.65 mmol/L, high NLR, low PLR, coagulation abnormalities (INR >1.29, APTT >50 s), and putamen involvement.

Tables 4a and 4b. Binary comparisons of quantitative admission parameters according to mortality status

Table 4a. Results of t-tests for parameters showing a normal distribution

Variable	Outcome				p
	Mortality (-)		Mortality (+)		
	n	Mean ± SD	n	Mean ± SD	
Age (years)	**64**	**43.86±15.62**	**28**	**52.86 ± 10.38**	**0.002**
Pulse	64	88.8 ± 14.37	28	85.54 ± 18.72	0.365
pCO2	64	36.61 ± 11.82	28	38.01 ± 16.68	0.69
HCO_3	**64**	**17.36 ± 6.97**	**28**	**7.3 ± 3.29**	**<0.001**
Anion Gap	**64**	**18.23 ± 7.74**	**27**	**27.76 ± 5.43**	**<0.001**
WBC	64	12.37 ± 4.99	28	13.97 ± 5.95	0.186
PLT	64	251.33 ± 91.25	28	241.86 ± 117.24	0.676
RBC	**64**	**4.91 ± 0.85**	**28**	**4.55 ± 0.61**	**0.049**
MCV	**64**	**93.26 ± 10.03**	**28**	**104.26 ± 9.15**	**<0.001**
LYM%	64	19 ± 11.98	28	23.5±13.19	0.111
NEU%	64	72.47 ± 13.5	28	69.97 ± 14.26	0.425
PLR	**64**	**164.68 ± 131.83**	**28**	**100.41 ± 61.3**	**0.002**
MPV	64	10.29 ± 1.05	28	10.28 ± 0.85	0.963
Creatinine	**64**	**0.93 ± 0.32**	**28**	**1.66 ± 1.34**	**0.009**
Amylase	**64**	**79.34 ± 56.15**	**27**	**159.93 ± 118.52**	**0.002**
K	**64**	**4.49 ± 0.88**	**28**	**5.72 ± 0.93**	**<0.001**
Ca	34	8.77 ± 0.59	5	8.8 ± 0.12	0.922

pCO2: Partial carbon dioxide, HCO3: bicarbonate, WBC: white blood cells, PLT: platelet count, RBC: red blood cells, MCV: mean corpuscular volume, LYM%: lymphocyte percentage, NEU%: neutrophil percentage, PLR: platelet-to-lymphocyte ratio, MPV: mean platelet volume, K: Potassium, Ca: Calcium, SS: standard deviation

Table 4b. Results of the Mann-Whitney U test for parameters not following a normal distribution

Variable	Outcome				p*
	Mortality (-)		Mortality (+)		
	n	Median [Min;Max]	n	Median [Min;Max]	
Time between exposure and presentation (hours)	62	18[1;168]	27	12[1;48]	0.373
GKS	**64**	**15[3;15]**	**28**	**3[3;13]**	**<0.001**
APACHE	**64**	**9.5[1;36]**	**28**	**26[11;44]**	**<0.001**
SOFA	**64**	**0.5[0;12]**	**28**	**12[2;19]**	**<0.001**
Length of stay in the intensive care unit	64	5[1;364]	28	3[1;60]	0.341
Duration (days)					
Systolic blood pressure	**64**	**130[30;195]**	**28**	**109[40;210]**	**<0.001**
Diastolic blood pressure	**64**	**80[68;110]**	**28**	**70[25;100]**	**0.017**
Temperature (°C)	**64**	**36.7[36.2;37.2]**	**28**	**36.9[36.3;37.3]**	**0.019**
Oxygen saturation (%)	64	98[90;100]	28	98.5[84;100]	0.166
pH	**64**	**7.3[6.75;7.54]**	**28**	**6.87[6.6;7.31]**	**<0.001**
Lactate	**64**	**2.5[0.9;14.8]**	**25**	**8.7[1.4;12.3]**	**<0.001**
Base Deficit	**63**	**-4[-33.2;4.4]**	**28**	**-27.5[-31.9;-11.9]**	**<0.001**
Lactate/HCT	**64**	**0.05[0.01;0.38]**	**28**	**0.17[0.02;0.33]**	**<0.001**
Glucose	**64**	**106.5 [49;468]**	**28**	**200[45;512]**	**<0.001**
GFR	**64**	**100[11;170]**	**28**	**57.5[8;93]**	**<0.001**
BUN	64	11.5[4;50]	28	12[6;81]	0.571
AST	**64**	**31.5[14;5118]**	**27**	**71[10;589]**	**<0.001**
ALT	**64**	**25[7;1023]**	**27**	**44[5;221]**	**0.002**
GGT	**56**	**37.5[7;870]**	**25**	**99[15;2272]**	**0.005**

Total Bilirubin	64	0.36[0.1;2.7]	27	0.3 [0.14; 2.47]	0.489
Direct Bilirubin	64	0.11[0.09;1.7]	27	0.16 [0.09; 1.39]	0.215
CK	63	163[51;12478]	25	152[47;1247]	0.410
LDH	**48**	**279[176;1683]**	**19**	**355[157;2338]**	**0.016**
Lipase	**64**	**39[15;475]**	**26**	**83.5[23;624]**	**<0.001**
CRP	64	2.6[0.6;94]	28	2.08[0.6;29.5]	0.587
Cl	64	103[81;117]	28	102[96;109]	0.548
Na	64	138[120;146]	28	137.5[128;148]	0.670
Troponin	**64**	**8.48[3;116]**	**27**	**19.9[4;628]**	**<0.001**
CKMB	**63**	**3[0.3;18]**	**27**	**4[1;24]**	**0.045**
Osmolality	64	289.65[256.1;379.6]	28	292.95[196.6;377.8]	0.230
HBG	64	15.25[7.7;19.8]	28	14.9[9.1;17.8]	0.408
HCT	64	46.35[25.3;66.9]	28	49.25 [34.2; 58.9]	0.242
MO%	**64**	**6.4[0.3;18.8]**	**28**	**4.85[0.4;12.5]**	**0.029**
EOS%	64	0.3[0;4.6]	28	0.4[0;3]	0.366
NEU#	64	8.35[2.71;28.4]	28	9.06[1.65;22.2]	0.488
LY#	**64**	**1.95[0.2;8.2]**	**28**	**3.2[0.58;5.62]**	**0.017**
MO#	64	0.74[0.24;2.4]	28	0.61[0.07;2.2]	0.272
NLR	64	3.91[0.93;41]	28	3.23[0.45;16.78]	0.205
SII	64	914.11[151.28;10593.2]	28	838.04[26.01;5153.75]	0.263
PIV	64	718.63[79.39;12394.04]	28	553.35[11.38;3092.25]	0.109
APTT	**60**	**26.2[18.5;83]**	**25**	**48.5 [27.2; 198.3]**	**<0.001**
PTZ	**60**	**12.55[10.6;18.3]**	**25**	**16.9[12.7;30.3]**	**<0.001**
INR	**60**	**1.05[0.9;1.72]**	**25**	**1.52[1;2.87]**	**<0.001**
Ethanol	64	10[10;377]	28	10[10;524]	0.055

* Mann-Whitney U test p-value

GCS: Glasgow Coma Scale, SOFA: Sequential Organ Failure Assessment, APACHE: Acute Physiology and Chronic Health Evaluation, GFR: glomerular filtration rate, BUN: blood urea nitrogen, AST: aspartate aminotransferase, ALT: alanine aminotransferase, GGT: gamma-glutamyl transferase, CK: creatine kinase, LDH: lactate dehydrogenase, CRP: C-reactive protein, Cl: Chloride, Na: Sodium,

CKMB: Creatine kinase myocardial band, HGB: Hemoglobin, HCT: Hematocrit, MO%: Monocyte percentage, EOS%: Eosinophil percentage, NEU#: absolute neutrophil count, LY#: absolute lymphocyte count, MO#: absolute monocyte count, NLR: neutrophil-to-lymphocyte ratio, SII: Systemic Immune-Inflammation Index, PIV: Pan-Immune-Inflammation Value, APTT: activated partial thromboplastin time, PTZ: prothrombin time, INR: international normalized ratio

In our study, numerous biochemical and clinical parameters were evaluated for their potential use in predicting mortality risk; however, based on statistical analyses, 6 parameters with clinically significant cut-off values were selected. This selection took into account both variables found to be significant in correlation analyses and parameters with prognostic value in clinical practice. The selected parameters are: lactate >7.65 mmol/L, anion gap >23 mmol/L, INR >1.29, creatinine >1.19 mg/dL, GCS <13, pH <7.06. Exceeding the cut-off value for each of these parameters was assigned 1 point, and an increase in the total score was observed to correlate with an elevated risk of mortality.

The MPMS developed in this study demonstrated high predictive performance by integrating clinical, biochemical, and radiological parameters. As a result of the ROC analysis, the AUC value of the MPMS for predicting mortality was calculated as 0.939 (95% CI: 0.891–0.987; p<0.001), and the optimal cutoff value was found to be 2.5. This score provided high sensitivity (91%) and specificity (88%) in predicting mortality and has been proven to be applicable in clinical practice.

Table 5. MPMS mortality risk scoring according to cut-off values

Variable	Cut-off value	High-risk condition (1 point)	Non-risk condition (0 points)
Lactate	> 7.65	> 7.65	≤ 7.65
Anion gap	> 23	> 23	≤ 23
INR	> 1.29	> 1.29	≤ 1.29
Creatinine	> 1.19	> 1.19	≤ 1.19
GCS	<13	<13	≥ 13
pH	<7.06	<7.06	≥ 7.06

INR: international normalized ratio, GCS: Glasgow Coma Scale

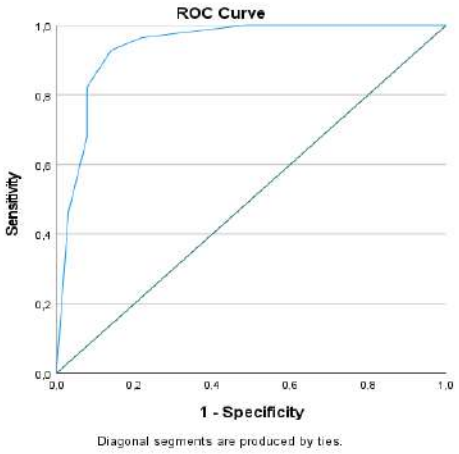

Figure 1. ROC analysis of the MPMS mortality score based on the presence of mortality

4. Discussion

In this study, the clinical, laboratory, and radiological findings of 92 patients presenting to the emergency department following methanol ingestion were examined in detail, and parameters associated with mortality were evaluated. Our findings once again demonstrate that methanol intoxication is a critical toxicological emergency characterized by multisystemic involvement, leading to high mortality if not diagnosed and treated early.

In our study, male gender (94.6%) and advanced age showed a significant association with mortality. This finding is consistent with epidemiological data reported in Turkey and other countries (7,8). The literature indicates that illicit alcohol production and consumption are more common among men, and that socioeconomic factors in particular contribute to increased risk in this group (13). The association between advanced age and mortality can be explained by declining physiological reserves and accompanying comorbidities with advancing age (11).

The most common symptoms at presentation were altered consciousness (37%) and visual disturbances (27.2%). These findings showed a strong correlation with mortality. In particular, a low GCS (mean 10.63 ± 5.45) was found to be strongly associated with mortality. Similarly, the literature emphasizes that a low GCS is a critical parameter in predicting central nervous system involvement and poor prognosis (9,14). Visual disturbances reflect the toxic effects of methanol on the retina and optic nerve, and it has been noted in the literature that the visual distortion described as the “snowstorm” phenomenon is specific to methanol toxicity (10).

In our study, critical thresholds for predicting severe acidosis in the mortality group are reported (12).

Upon examination of biochemical parameters, significant elevations were detected in creatinine, troponin, amylase, and liver function tests. This situation highlights methanol's multisystemic toxicity and, in particular, renal, cardiac, and hepatic organ dysfunction (15,16). Among inflammatory indices, high NLR (6.54 ± 7.19) and low PLR (145.12 ± 118.45) values showed a strong correlation with mortality. This finding indicates that the systemic inflammatory response plays a significant role in determining prognosis. The literature also emphasizes that the NLR is a reliable marker for prognosis in sepsis and toxicological conditions (4).

Among coagulation parameters, INR >2 and APTT >50 s showed a significant association with mortality. This result indicates that the development of coagulopathy in severe cases is associated with a poor prognosis. The literature also reports that coagulation abnormalities are associated with mortality in methanol intoxication (17).

The most striking finding in neuroimaging was putamen involvement. Putamen lesions were observed in 69.2% of the mortality group, whereas this rate was 30.8% in the survivor group. This finding is frequently highlighted in the literature as a pathognomonic feature specific to methanol intoxication (3,10). Putamen involvement reflects methanol's toxic effect on the basal ganglia and serves as a guide in predicting mortality risk.

In our study, the total mortality score obtained using a binary (0–1) scoring method based on cutoff values was comprehensively evaluated via ROC analysis and demonstrated strong performance in predicting mortality. The AUC value obtained from the ROC analysis was 93.9%, indicating that this score is superior to individual parameters in distinguishing mortality. The optimal cutoff value was determined to be 2.5, and patients were classified into different mortality risk groups based on this threshold. According to this scoring system, a significant increase in mortality was observed in groups scoring 3 points or higher. This approach demonstrates that the developed MPMS system can be used as an easily applicable and interpretable risk classification tool in clinical practice.

In the literature, ROC analyses are frequently used to predict mortality in methanol poisoning, and the prognostic value of individual parameters has been examined. It has been reported that threshold values of pH <7.0 and GCS ≤12 strongly predict mortality (18). In one study, ROC analyses were performed for pH and anion gap, and it was reported that they provided high sensitivity and specificity in predicting mortality (19). In another study, it was reported that bicarbonate (HCO_3^-) levels were strongly associated with mortality in ROC analysis (20). In yet another study, it was reported that creatinine and HCO_3^- levels served as independent predictors of mortality in ROC analysis (21). A further study emphasized that GCS ≤12 and elevated lactate levels were strong predictors of mortality in ROC analysis (22).

In our study, however, these parameters were not used individually but were combined to create a new scoring system, yielding a higher AUC value in ROC analysis compared to individual parameters. This finding supports the superiority of a multi-parameter approach in mortality prediction and suggests that the MPMS score could serve as a guide in clinical practice for early risk stratification and the determination of treatment strategies.

The MPMS developed in our study stands out as one of the most comprehensive and original initiatives in the literature aimed at predicting mortality in methanol poisoning. The most significant feature of this scoring system is that it combines the prognostic power of individual parameters to create a multidimensional risk classification system. The selection of critical parameters—both biochemical and clinical—such as lactate, anion gap, INR, creatinine, GCS, and pH enhances the score's applicability in both laboratory and clinical settings. Validating the cut-off values determined for each parameter via ROC analysis has reinforced the score's statistical robustness.

One of the strengths of this scoring system is that it achieves a higher AUC value in predicting mortality compared to individual parameters. This finding demonstrates the superiority of the multi-parameter approach in mortality prediction and indicates that it provides a reliable tool for early risk classification in clinical practice. Additionally, the ease of interpretation and application of the score in clinical practice, thanks to the established cutoff value, provides a practical advantage for intensive care physicians.

There are studies in the literature where various parameters aimed at predicting mortality are evaluated individually. However, a common feature of these studies is that they evaluate parameters individually and do not offer a combined scoring system. Our study addresses this shortcoming by integrating multiple parameters into a single scoring system, achieving higher accuracy in mortality prediction, and developing a unique tool suitable for clinical practice.

Given these features of the MPMS, it can be tested not only in retrospective analyses but also in prospective, multicenter studies, validated across different populations, and integrated into treatment algorithms. In particular, integrating MPMS into clinical decision support systems at critical decision points—such as antidote timing, hemodialysis indication, and intensive care prioritization—could provide a strategic contribution toward reducing mortality. In this context, MPMS has the potential to usher in a new era in the field of methanol poisoning, both scientifically and clinically. In terms of future studies, MPMS, in this regard, is not merely a product of our current study but also a model that could lead the way for future research.

Our results demonstrate that clinical (altered consciousness, low GCS), laboratory (pH, bicarbonate, lactate, NLR, PLR, coagulation abnormalities), and radiological (putamen involvement) parameters are of critical importance in predicting mortality in methanol

intoxication. Early assessment of these parameters may contribute to the development of risk stratification in the emergency department and the reduction of mortality rates.

5. Limitations

- First, since the study is single-center and retrospective in design, the generalizability of the results to different geographic regions or healthcare systems is limited.
- Due to the retrospective data collection method, there is a possibility of missing or erroneous information in medical records, which has limited the detailed analysis of certain clinical variables.
- The relatively low number of patients included in the study (n=92) reduced its statistical power, particularly in comparisons between subgroups. Additionally, since serum methanol levels were not measured, indirect parameters (anion gap, lactate, etc.) were used for diagnosis and prognosis assessments; this resulted in limited biochemical validation.
- Neuroimaging findings were obtained only in cases clinically indicated, so the radiological results could not be generalized to the entire sample.
- The developed MPMS has been tested only in this study; since no external or prospective validation has been conducted, its validity in different populations is limited.

6. Conclusion

In this study, data from patients exposed to methanol poisoning were analyzed, and the primary factors associated with mortality were identified. Advanced age, male gender, altered consciousness at presentation, and visual disturbances were found to be significant predictors of mortality. Low GCS, high APACHE II and SOFA scores, severe acidosis (pH < 7.0), low bicarbonate, and high lactate levels were strongly associated with mortality. Elevations in creatinine, troponin, amylase, and liver function tests indicated methanol's multiorgan toxicity. Elevated NLR and low PLR values among inflammatory indices, prolonged coagulation parameters (PT, INR, APTT), and radiological putamen involvement were identified as additional predictors of mortality. The MPMS, the most original outcome of our study, has been established as a practical and easily applicable scoring system that integrates clinical, biochemical, and radiological parameters to predict mortality with high sensitivity and specificity, and could contribute to reducing mortality at critical decision points.

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1938

Specific Neuroradiological Sign in a Trauma Patient: Mount Fuji.

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Introduction

Pneumocephalus refers to the presence of air within the cranial cavity and most commonly occurs following craniofacial trauma, neurosurgical procedures, or skull base defects [1]. Although typically benign and self-limiting, tension pneumocephalus is a rare but life-threatening condition characterized by progressive intracranial air accumulation causing mass effect and neurological deterioration [2]. Collapse of the frontal lobes and widening of the interhemispheric space between the tips of the frontal lobes on computed tomography resemble Mount Fuji, and this sign is considered a classic radiological indicator of this condition [3]. Here, we present a case of tension pneumocephalus following head trauma in an elderly patient, emphasizing the importance of early recognition and imaging.

Case:

A 92-year-old male presented to the emergency department after a ground-level fall at home while walking to the bathroom. According to history, he had experienced a similar fall four days prior but did not seek medical attention at that time. His past medical history was significant for a recent hospitalization due to gastrointestinal bleeding two months earlier. The patient had no known medical history other than benign prostatic hyperplasia (BPH).

Initial vital signs were as follows: temperature: 36.6°C, pulse: 72 bpm, oxygen saturation 90% on room air, blood pressure: 150/63 mmHg, and blood glucose level: 199 mg/dl. Neurological examination revealed a Glasgow Coma Scale (GCS) score of 12/15 (E4 M6 V3). The patient was followed with nasal oxygen support, and breath sounds were normal on auscultation. Cardiovascular examination revealed a regular heart rhythm, peripheral perfusion time was less than 3 seconds, and no pretibial edema was observed. Urinary output was 500 cc. Abdominal examination demonstrated a soft abdomen with normoactive bowel sounds, and oral intake was discontinued. The patient's initial laboratory results were as follows: eGFR: 37, AST:13 U/L, ALT:8 U/L, GGT: 17 U/L, LDH:165 U/L, total bilirubin 0.56mg/dl, direct bilirubin: 0.31 mg/dl, amylase: 97U/L, Lipase: 11 U/L, CRP 204.5 mg/L, hemoglobin (HGB): 12.4 g/dl, red blood cells (RBC):4.47 million/ul, white blood cells (WBC): 11,89 10*3/Ul, platelets (PLT): 297 10*3/Ul, INR: 1.11, troponin: 54.8 ng/L.

Non-contrast cranial computed tomography demonstrated extensive bilateral extra-axial air collections in the frontal regions, consistent with pneumocephalus. The frontal lobes were displaced posteriorly, findings suggestive of tension pneumocephalus (Mount Fuji sign). Additional extra-axial pneumocephalus was also observed in the anterior and temporal regions. Further evaluation with thin-slice CT was recommended to assess for a possible skull fracture. Bilateral extra-axial subdural effusions were noted in the parietal regions, measuring up to 6 mm at their maximum thickness. The cortical sulci appeared compressed secondary to the pneumocephalus. No pathological findings were identified in the mesencephalon, pons, medulla oblongata, or cerebellar hemispheres.

The ventricular system was normal in configuration and size. Sellar and parasellar structures were unremarkable. A non-contrast cranial CT scan demonstrated bilateral chronic subdural hematomas with mass effect. Loss of aeration was observed in the left maxillary sinus, with soft tissue densities filling the sinus and obliterating the left osteomeatal complex. These findings were consistent with chronic sinusitis of the left maxillary sinus.

Neurosurgery was consulted due to pneumocephalus and subdural effusion, and no urgent surgical intervention was recommended; close follow-up with levetiracetam 500 mg twice daily and further evaluation with contrast-enhanced brain MRI and heavily T2-weighted sequences were advised. Infectious diseases was consulted due to elevated C-reactive protein levels, and intravenous ceftriaxone 2 g twice daily and metronidazole 500 mg four times daily

were initiated, with recommendations for monitoring of acute phase reactants and chest imaging. Otorhinolaryngology was consulted to evaluate for possible otorrhea and rhinorrhea, and no evidence of either was detected on examination.

During intensive care unit follow-up, contrast-enhanced cranial MRI was performed. Imaging demonstrated persistent bilateral frontal extra-axial air collections consistent with pneumocephalus, more prominent on the right side, along with a bilateral frontal subdural effusion, also more pronounced on the right. Diffusion-weighted images revealed areas of diffusion restriction at the cortical-subcortical junction in the bilateral parietal regions and at the level of the left precentral gyrus, suggestive of acute ischemic changes. In addition, a lesion in the left corona radiata was noted, consistent with gliosis. Non-contrast cranial CT performed during follow-up demonstrated right-sided pneumocephalus and a right frontal subdural effusion, with compression of the lateral ventricles. Periventricular white matter hypodensities were observed, consistent with chronic ischemic changes. Additionally, a crescent-shaped subacute subdural hematoma was noted in the left parieto-occipital region.

After three days in the intensive care unit, the patient was transferred to the neurosurgery ward for further management.

Figure 1: Non-contrast cranial computed tomography demonstrated extensive bilateral extra-axial air collections in the frontal regions, consistent with pneumocephalus. The frontal lobes were displaced posteriorly, findings suggestive of tension pneumocephalus (Mount Fuji sign).

Discussion:

This case illustrates a rare but clinically significant presentation of tension pneumocephalus in an elderly patient following minor head trauma. Advanced ageand the presence of head trauma likely contributed to the development of intracranial air accumulation in this patient.

Radiologically, the presence of the Mount Fuji sign on CT is highly suggestive of tension pneumocephalus and indicates significant mass effect requiring close monitoring or intervention. In this case, despite imaging findings consistent with tension pneumocephalus, the patient was managed conservatively due to stable clinical status, emphasizing that management should be individualized based on both radiological and clinical findings. Management of pneumocephalus ranges from conservative measures to surgical decompression in cases of neurological deterioration or progressive mass effect. In this patient, conservative management with close observation, antiepileptic prophylaxis, and antibiotic therapy was deemed appropriate, and no immediate neurosurgical intervention was required.

In conclusion, this case highlights that tension pneumocephalus can develop even after minor trauma in elderly patients and may present with subtle clinical findings. Early imaging, careful multidisciplinary evaluation, and individualized management are essential to optimize outcomes.

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2010

The Double-Edged Sword: Intracerebral Hemorrhage and Aneurysm Risk in a Patient with Cardiac Valve Replacement

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INTRODUCTION

Intracerebral hematoma is associated with risk factors such as hypertension, anticoagulant use, and vascular malformations (1). The risk of bleeding is increased in patients receiving anticoagulant therapy following cardiac valve replacement (2). Intracranial hemorrhages developing in such patients may be related to both spontaneous causes and vascular pathologies. In particular, the presence of an aneurysm is one of the most important factors

determining the etiology and prognosis of the hematoma (3). In this report, we present a case of intracranial hematoma and a possible aneurysm in a patient with a history of cardiac valve replacement and cerebrovascular disease.

CASE PRESENTATION

A 70-year-old male patient presented to the emergency department with complaints of nausea, vomiting, and syncope. His Glasgow Coma Scale score was 15, blood pressure was 90/60 mmHg, heart rate was 39 beats/min, oxygen saturation was 94%, and body temperature was 36.4°C. Electrocardiography revealed sinus rhythm.

Neuroimaging demonstrated an approximately 5-cm hematoma adjacent to the ventricular system in the right occipital region, accompanied by perifocal edema and hemorrhagic extension into the ventricular system (Figure 1). These findings suggested an increased risk of intracranial pressure elevation and secondary neurological deterioration.

Figure 1. Brain CT Imaging of the Patient

The patient was consulted to the departments of neurosurgery and neurology. Neurosurgery did not consider emergency surgical intervention, whereas neurology requested CT angiography for further evaluation of a possible vascular pathology. CT angiography demonstrated a 4-mm hyperdense lesion within the center of the hematoma, suggestive of a possible aneurysm. Edema was present in the surrounding brain parenchyma adjacent to the hemorrhagic area. Interventional radiology did not recommend any endovascular procedure, and the patient was admitted to the intensive care unit for neurological monitoring and follow-up.

DISCUSSION

Although hypertension is the most common cause of intracerebral hematoma, anticoagulant therapy and vascular malformations are also important risk factors (4,5). In our case, anticoagulant use following cardiac valve replacement and a history of cerebrovascular disease increased the risk of hemorrhage.

CT angiography plays a critical role in the differential diagnosis of vascular pathologies (e.g., aneurysms) in patients with intracranial hematomas (6). Previous studies have reported that a hyperdense lesion within the center of a hematoma may be associated with an aneurysm (3). A similar finding was observed in our patient; however, no interventional procedure was considered.

A multidisciplinary approach is essential in the management of such cases. Close collaboration among neurology, neurosurgery, and interventional radiology teams is crucial in determining both surgical and conservative treatment strategies (2). In addition, intensive care unit monitoring is of critical importance for detecting hematoma progression and preventing secondary complications (1).

CONCLUSION

Intracerebral hematoma may follow a complicated clinical course, particularly in patients with a history of cardiac surgery and cerebrovascular disease. CT angiography is crucial for early diagnosis and interventional planning. A multidisciplinary approach and intensive care unit monitoring are essential for improving survival in these patients.

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Keywords: Intracerebral hematoma, Aneurysm, Emergency department

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Treatment management of high-grade renal laceration

A case report

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Abstract: The kidneys are the organ most frequently injured due to blunt trauma. Renal injuries account for between 1% and 5% of all trauma cases. Young men are affected more frequently. One quarter of all solid organ injuries are due to kidney trauma. Renal trauma can result in damage to the parenchyma and vascular structures, as well as the urinary collecting system and renal pelvis. Causes include direct blows, motor vehicle accidents and falls, inferior rib fractures, sports injuries and pedestrian injuries. The most common injury mechanism is sudden deceleration injury or a direct blow to the back or side. Penetrating trauma near the kidney can cause renal pedicle rupture and renal artery dissection. Renal injury should be suspected in patients presenting with costovertebral pain, abdominal or back pain, a palpable mass, ecchymosis, hypotension and shock. Bicycle accidents, especially in children, are a significant cause of renal trauma. Haemodynamic instability and haematuria increase suspicion of renal injury. However, the degree of haematuria does not predict the severity of renal injury and it may not be present at all. We present the case of a 16-year-old male patient with a grade 4–5 renal laceration caused by impact with a bicycle handlebar during a fall. Serious consequences can arise from seemingly minor injuries. Here, we present the emergency department management and non-operative treatment of a patient who developed a high-grade renal laceration due to blunt trauma.

Key words: Blunt trauma, renal laceration, hematuria

Introduction:

Genitourinary system injuries account for 10% of all trauma cases, and the kidney is the most commonly affected organ. Most of these injuries are caused by blunt abdominal trauma (80%–90%). Penetrating kidney trauma, on the other hand, occurs in 10–20% of cases (1). It is more common in men around the age of 30. The most common causes of blunt trauma are motor vehicle collisions (63%), followed by falls (14%), sports-related injuries (11%), pedestrian injuries (4%), and others (6%). The pathophysiology of blunt kidney trauma involves deceleration and acceleration forces caused by the traumatic event. The kidney is covered by retroperitoneal fat and Gerota's fascia. The renal pedicle and the ureteropelvic junction are the primary attachment structures. Deceleration forces can cause renal damage such as rupture or thrombosis, while acceleration forces can result in parenchymal and vascular injuries when the kidney strikes surrounding structures such as the ribs and spine.

Penetrating injuries consist of firearm injuries (65%) and stab wounds (2). Renal trauma may result in lacerations, contusions, vascular injuries, and urinary extravasation, along with injury to the collecting system. In cases of renal laceration, the medulla and collecting system remain unaffected. In renal contusion, while the renal capsule is preserved, the injury involves parenchymal ecchymosis, minor lacerations, and subcapsular hematomas.

If hematuria is present alongside localized pain in the upper and lateral regions of the abdomen, abdominal tenderness and swelling, a palpable mass, skin ecchymosis, rib fractures, hypotension, and shock, the suspicion of renal trauma is very high. The likelihood of renal injury is higher in patients with microscopic hematuria and a systolic blood pressure <90 mmHg. In renal trauma, there is no direct correlation between the presence of microscopic hematuria in urine analysis and the severity of the injury (3). Hematuria may not always be present in patients with polytrauma. Hematuria may not be present in cases of renal arterial thrombosis, renal pedicle injury, ureteral tears, and ureteropelvic junction injuries (4). Early diagnosis and treatment approach are of vital importance in renal trauma. Our aim is to review the diagnostic and treatment process of a patient who developed a renal laceration following a bicycle fall, in light of the literature, and to provide practical recommendations for the management of such cases.

Case presentation:

A 16-year-old male patient was brought to our hospital's emergency department following a bicycle fall. The patient's general condition is good; he is alert, oriented, and cooperative, with a GCS of 15. The neurological examination is unremarkable; blood pressure is 120/80 mmHg and heart rate is 90 bpm. ECG shows normal sinus rhythm; respiratory rate 16/min; bilateral breath sounds are normal; abdominal examination reveals pain and tenderness in the left upper quadrant; no guarding or rebound tenderness; no signs of ecchymosis, abrasions, or other lesions on the abdominal skin. WBC: 7.54 × 10^9/L, Hb: 14.2 g/dL; biochemical tests and other blood parameters were normal. Gross hematuria was detected in the patient's complete urinalysis. A urinary catheter was inserted, and IV hydration and analgesic therapy were initiated. Contrast-enhanced abdominal CT revealed no pathology in the liver, spleen, or gallbladder fossa. The left renal pelvic-calyceal system was found to be dilated to grade 3–4. A hematoma-like appearance was observed in the left renal pelvic-calyceal system and the proximal left ureter. An appearance consistent with a large hematoma extending from the left kidney toward the left pelvis and reaching as far as the splenic hilum in the superior region was observed (Figure 1). This finding was interpreted as indicative of a Grade 4–5 renal laceration (Figure 2).

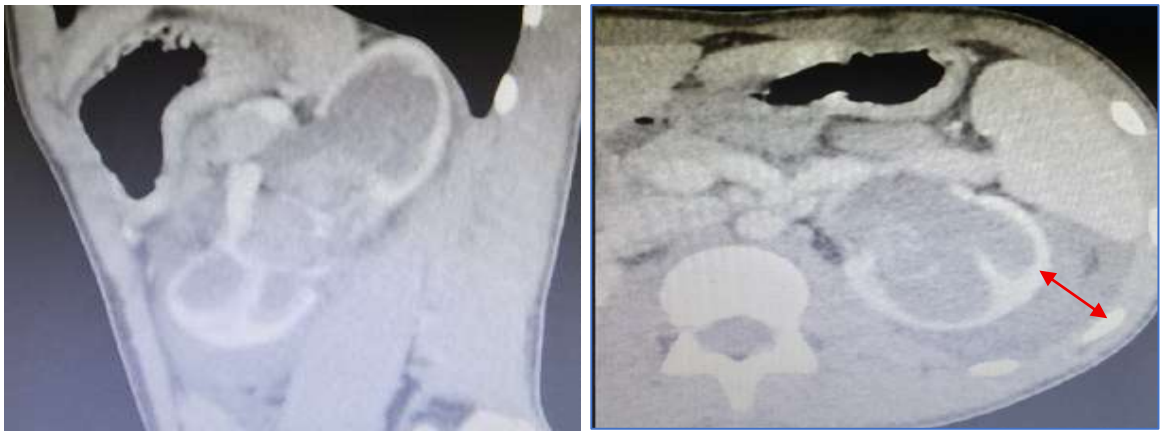

Figure 1: A large hematoma is observed around the left kidney (red arrow).

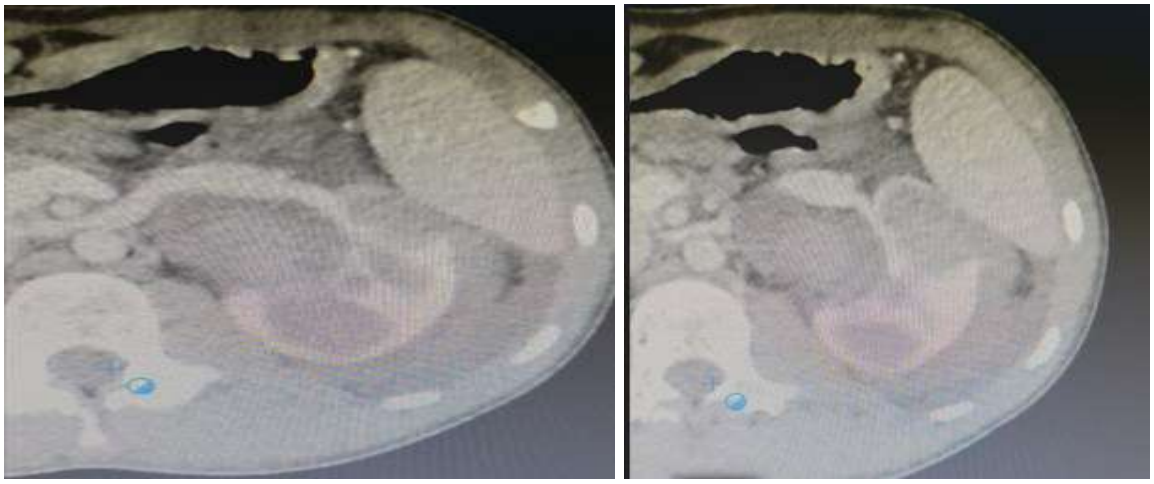

Figure 2: Renal trauma (Grade 4–5) with a renal laceration was observed.

Since the patient's vital signs and hemoglobin (HGB) level were stable, emergency surgery was not scheduled, and a decision was made to monitor the patient. After consultation with pediatric surgery, the patient was admitted to the pediatric intensive care unit. The patient was placed under continuous monitoring, and treatment was initiated with IV hydration, analgesics, ceftriaxone, and gastric protectants. The patient was monitored in a non-ambulatory state. Hemoglobin levels were monitored, and no significant drop in HGB was observed. The patient, who was being monitored with Regimen-1 oral feeding and a urinary catheter, continued to have hematuric urine. On the third day of hospitalization, amikacin was added due to a fever of 38 °C, and a contrast-enhanced abdominal CT scan was repeated. The abdominal CT revealed dilation of the left kidney causing parenchymal thinning; the transverse AP diameter of the pelvis was 38 mm, with parenchymal thickness measured at 4 mm at its thinnest point and 15 mm at its thickest point. Hemorrhage of varying densities was present filling the left renal collecting system, pelvis, and proximal ureter. The left ureter was filled with hemorrhage and measured 48 mm at its widest point. The continuity of the left ureter is not specified. Parenchymal integrity is lost in the middle segment of the left kidney,

and there is hemorrhage extending from the pelvis into the perirenal space. An interventional radiologist placed a nephrostomy catheter and removed the urinary catheter. A total of 2100 cc of hematuric urine was drained from the nephrostomy catheter, and the patient's hemoglobin level did not decrease. The patient, who was in good general condition, was transferred to the pediatric surgery ward on the 6th day. On the 10th day of follow-up, 900 cc of hematuric urine was drained from the nephrostomy catheter. An antegrade JJ stent was placed by the interventional radiology team, and the nephrostomy catheter was removed. The patient was discharged on the 11th day with recommendations, and an outpatient follow-up was scheduled for one week later.

Discussion:

Renal injuries account for 1–5% of genitourinary system injuries. The most common mechanism of kidney injury is blunt trauma, and the majority of these injuries are minor. In cases of minor injuries, a conservative treatment approach should be prioritized. Penetrating injuries are less common, and the likelihood of renal injury is higher in cases involving sharp or piercing instruments (5).

64% of renal injuries are contusions and subcapsular hematomas, 26% are lacerations, 6% are parenchymal injuries, and 4% are vascular injuries (6). For stable patients with suspected renal injury, IV-contrast CT imaging of the abdomen and pelvis is the "gold standard." Spiral CT provides rapid imaging; however, to allow for a clear assessment of the collecting system, a follow-up CT scan is recommended during the post-treatment follow-up period (4). The primary goal in renal injury is to protect the kidney. Since the severity of renal trauma can vary significantly, management options may also differ. While conservative management is the standard of care for low-grade blunt renal injuries, the decision to perform surgical treatment for high-grade blunt injuries and penetrating renal injuries is based on hemodynamic stability (7). In a multicenter study, conservative treatment was applied in approximately 75% of patients with grade 4 and 5 renal trauma, and failure was observed in only 6.5% of patients. It has been reported that conservative treatment does not prolong the length of hospital stay in cases of high-grade renal injury (8). In patients with suspected renal trauma, a thorough medical history should be obtained first. The nature of the injury, history of lumbar trauma, and presence of hematuria should be assessed. If the patient is hemodynamically stable, a medical history, physical examination, laboratory tests, and imaging should be performed. Treatment options include conservative management, minimally invasive procedures, and open surgery. Conservative management typically involves bed rest, analgesia, hemodynamic monitoring, serial laboratory evaluations, and repeat imaging if there is a deterioration in the patient's hemodynamics. Minimally invasive procedures include angiographic embolization for active bleeding and endourological stenting or placement of a nephrostomy tube for urinary leakage. In hemodynamically unstable patients, urgent hemodynamic support must be provided, and the decision for surgical intervention must be made and implemented promptly. Open surgeries typically include nephrectomy, partial nephrectomy, renorrhaphy, renal tamponade, or autotransplantation (9). Surgical treatment is recommended in cases of hemodynamic instability, a decrease in hemoglobin levels, the presence of an enlarging and pulsatile hematoma on radiological

imaging, persistent hypotension despite transfusion, or suspected injury to the renal pelvis and ureter (10). Lanchon et al. reported success rates of 88% and 50% when applying conservative treatment in patients with grade 4 and 5 renal trauma, respectively (11). In Altman et al.'s study, they reported that a conservative treatment approach is appropriate in cases of grade 5 renal injury if hemodynamics are stable (12). In the study by Hardee et al., a relationship was identified between the size of the perirenal hematoma and the need for surgical treatment, as well as intravenous contrast medium extravasation (13). According to the European Association of Urology guidelines, repeat radiological imaging is recommended in cases of complications such as fever, decreased hematocrit, or persistent flank pain (14). Complications may occur following renal trauma. Early complications include bleeding, infection, perinephric abscess, sepsis, hypertension, urinary fistula, urinary extravasation, and pyelonephritis, all of which may develop within 30 days. Late complications may include bleeding, hydronephrosis, chronic pyelonephritis, hypertension, arteriovenous fistula, hydronephrosis, stone formation, and pseudoaneurysms (15). In our case, despite the presence of blunt severe kidney injury, a conservative approach was adopted because the patient's vital signs and laboratory values were stable. A high fever was detected in the early stages, and no other complications were observed. In conclusion, rapid diagnosis in the emergency department setting prevents unwanted complications. Based on the patient's vital signs and laboratory findings, a conservative approach can be safely applied even in cases of high-risk kidney injuries in patients with stable hemodynamics. This treatment will reduce the need for unnecessary surgery.

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Acute Lymphoblastic Leukemia Presenting with Pulmonary Thromboembolism in the Postoperative Period: Case Report

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ABSTRACT

Introduction and Purpose

Pulmonary thromboembolism (PTE) is a common clinical entity, especially in the postoperative period, and carries a high mortality risk. However, in some cases, PTE may be the first clinical manifestation of underlying malignancies. Acute lymphoblastic leukemia (ALL) is a rare but aggressive hematologic malignancy in adults and usually presents with cytopenia or signs of infection. The aim of this case report is to emphasize the importance of establishing the underlying diagnosis of ALL in a patient presenting with PTE.

Case Presentation

A 58-year-old male patient was admitted to the emergency department with dyspnea and lower extremity complaints after varicose vein surgery. Thoracic computed tomography angiography revealed thromboembolism at the level of the subsegmentary branch of the right pulmonary artery. Laboratory tests revealed marked leukocytosis and thrombocytopenia. Upon observation of blastic cell infiltration in the peripheral smear, further hematologic evaluation was performed and the patient was diagnosed as acute lymphoblastic leukemia.

Results and Conclusion

In some cases, pulmonary thromboembolism may be the first sign of underlying malignancies. Especially in the presence of concomitant significant hematologic abnormalities, these findings should not be considered as merely reactive changes and further evaluation for hematologic malignancies should be performed. This case demonstrates the importance of comprehensive evaluation and investigation of underlying causes in patients diagnosed with PTE in the emergency department.

Keywords: Pulmonary thromboembolism, acute lymphoblastic leukemia, emergency department

Introduction

Pulmonary thromboembolism (PTE) is the most serious clinical form of venous thromboembolism and is more common in the presence of surgical intervention, immobilization, advanced age and malignancy (1). Thrombosis associated with malignancy is a well-known clinical entity and in some patients, a thromboembolic event may be the first clinical manifestation of an undiagnosed cancer (2). Although thrombosis in hematologic malignancies is less emphasized compared to solid tumors, it is an important cause of morbidity due to disease biology, inflammatory processes, hypercoagulability and treatment-related factors (3).

Acute lymphoblastic leukemia (ALL) is an aggressive malignancy characterized by uncontrolled proliferation of lymphoid precursor cells in the bone marrow. While adult ALL patients are mostly diagnosed with symptoms related to malaise, infection, bleeding tendency or cytopenia, initial presentation with a thromboembolic event is much rarer (4). Therefore, further evaluation in terms of underlying hematologic malignancies is of great importance in the presence of unexplained marked leukocytosis, thrombocytopenia or peripheral smear abnormalities on hemogram in patients with PTE.

In this case report, we aimed to reveal the underlying diagnosis of acute lymphoblastic leukemia in a patient who presented with postoperative pulmonary thromboembolism and

emphasized the importance of careful evaluation of accompanying hematologic abnormalities in patients presenting with PTE.

Case Report

A 58-year-old male patient was admitted to the emergency department with complaints of pain and swelling in the right lower extremity and shortness of breath. It was learned that the patient had a history of diabetes mellitus, a right leg varicose vein operation about 5-6 days before admission, and a history of lumbar hernia surgery about 3 months ago.

In the evaluation performed in the emergency department, the general condition of the patient was moderate, conscious, coherent and oriented. Although significant tachypnea and dyspnea at rest were not described at presentation, dyspnea was noted with exertion. Physical examination revealed a diameter difference between the lower extremities and increased temperature; the right lower extremity was more edematous than the left. Vital signs were as follows: arterial blood pressure 160/80 mmHg, pulse 130/min, temperature 36.2°C and oxygen saturation 95%.

Laboratory tests revealed marked leukocytosis. Leukocyte (WBC) was 334.110/mm³, hemoglobin (HGB) 11.4 g/dL, C-reactive protein (CRP) 161.3 mg/L, D-dimer 16.6 µg/mL, platelet (PLT) 61.000/mm³ and lactate dehydrogenase (LDH) 1093 U/L. Arterial blood gases were pH 7.31, pCO_2 49 mmHg, pO_2 27 mmHg, HCO_3 22 mmol/L and lactate 3.6 mmol/L.

Contrast-enhanced thoracic computed tomography angiography performed at an external center showed filling defects in the segmentary branches of the lower lobe of the right lung compatible with acute pulmonary thromboembolism (Figure 1-3). With these findings, the patient was evaluated as pulmonary thromboembolism. Ejection fraction was found to be 60% on echocardiography performed during cardiology consultation; no significant finding in favor of right heart overload was observed.

Figure 1. Contrast-enhanced thorax CT angiography sections show filling defects in the right lung lower lobe segmentary pulmonary artery branches consistent with thromboembolism.

Hematology consultation was requested due to the presence of marked leukocytosis and thrombocytopenia in addition to the diagnosis of pulmonary thromboembolism. Peripheral smear revealed anisopoikilocytosis in erythrocytes and diffuse blastic cell infiltration in the leukocyte formula. Based on these findings, the patient was evaluated as newly diagnosed acute leukemia, primarily acute lymphoblastic leukemia and referred to a higher center for further investigation and treatment. Further investigations revealed Philadelphia chromosome positivity. Hyper-CVAD A regimen (vincristine, cytarabine, doxorubicin, cyclophosphamide, methotrexate and dexamethasone) with imatinib treatment was planned.

Discussion

This case highlights the important points to be considered in the evaluation of patients presenting with postoperative pulmonary thromboembolism (PTE). Although factors such as surgical intervention, immobilization and venous stasis constitute a strong risk for PTE, accompanying clinical and laboratory findings should be carefully evaluated. In this patient, lower extremity edema, elevated D-dimer and filling defect on CT angiography supported the diagnosis of PTE . However, the most important feature that distinguishes this case from typical postoperative PTE is the presence of significant hematologic abnormalities.

Although mild to moderate leukocytosis may accompany PTE, the excessive level of leukocytosis found in this case cannot be explained by a simple inflammatory response. Accompanying thrombocytopenia and high LDH levels suggest the possibility of hematologic malignancy (5,6). Detection of blastic cell infiltration in the peripheral smear was decisive in terms of diagnosis and changed the clinical approach. This shows that hemogram is not only supportive but also a critical tool guiding the diagnosis in patients with a diagnosis of PTE in the emergency department.

The development of thrombosis in acute leukemia is multifactorial. Interaction of blast cells with the endothelium, increased procoagulant activity, inflammatory response and leukostasis tendency play a role in the pathogenesis of thrombosis (7,8). In this case, in addition to the existing malignancy, recent surgical intervention is thought to have contributed to the development of thrombosis. In this respect, the case reflects a picture in which the basic components of the Virchow triad (venous stasis, endothelial damage and hypercoagulability) come together in the same patient.

Conclusion

Pulmonary embolism as the first clinical manifestation of acute leukemia is rare. Therefore, underlying hematologic malignancies should be kept in mind especially in patients with PTE in the postoperative period in the presence of accompanying marked leukocytosis,

thrombocytopenia and peripheral smear abnormalities. Early hematology consultation is critical in terms of making the diagnosis without delay.

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A hidden airway threat: Isolated thyroid laceration without goiter after blunt neck trauma

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Abstract

Although soft tissue injuries are common after neck trauma, thyroid gland injury secondary to blunt trauma is rare. Isolated thyroid gland injury is extremely uncommon and accounts for approximately 1–2% of all blunt neck trauma cases (1,2). Symptoms of thyroid injury following blunt cervical trauma may include neck swelling, respiratory distress, dysphagia, hoarseness, and neck pain. We present the case of a 27-year-old female patient admitted after an in-vehicle traffic accident who was diagnosed with thyroid gland laceration following radiological evaluation.

Keywords

thyroid, thyroid gland, trauma, blunt trauma

Introduction

Soft tissue injuries following blunt trauma to the neck are relatively common. However, because the neck contains numerous vital anatomical structures, such injuries may result in serious complications. Thyroid gland injuries secondary to blunt cervical trauma are rare and are mostly observed in patients with pre-existing thyroid diseases such as goiter (3). Structures including the larynx, trachea, pharynx, esophagus, major vessels, and cervical spine may also be affected in these injuries. In this context, isolated thyroid gland trauma is particularly uncommon (3). Most reported cases are associated with motor vehicle accidents or direct blunt impact to the neck (1,2).

This case report highlights thyroid gland injury following a motor vehicle accident in a patient without a previously known thyroid disorder. The case emphasizes the importance of early diagnosis, conservative treatment, and close clinical observation in preventing life-threatening complications associated with these rare injuries.

Case Presentation

A 27-year-old female patient was brought to the emergency department after an in-vehicle traffic accident. She complained of pain and swelling in the right lower anterior neck. The patient had not been wearing a seatbelt, and the airbags had deployed during the collision. She had no respiratory distress at presentation.

Vital signs were as follows: blood pressure 120/80 mmHg, pulse rate 84 beats/min, oxygen saturation 96%, and body temperature 36.1°C. Physical examination revealed ecchymosis over the neck, presumed to be secondary to airbag-related trauma (Figure 1). Palpation demonstrated tenderness and swelling in the right lower cervical region (Figure 1). No pulsation was detected over the swelling, and carotid pulses were symmetrical and normal. Multiple facial lacerations related to trauma were also noted.

Bedside superficial cervical ultrasonography performed in the emergency department demonstrated a hypoechoic avascular area within the right thyroid lobe, consistent with thyroid injury (Figures 2 and 3). Hemorrhage within the sternocleidomastoid muscle was also detected. The remaining portions of the thyroid gland appeared normal on ultrasonographic examination (Figure 4).

Contrast-enhanced neck computed tomography confirmed laceration of the right thyroid lobe (Figures 5 and 6). No tracheal deviation or vascular injury was identified.

Laboratory investigations demonstrated hemoglobin (Hb) level of 14.10 g/dL, thyroid-stimulating hormone (TSH) level of 0.278 mIU/L, free T3 level of 4.87 pg/mL, and free T4 level of 2.15 ng/dL. Serial hemoglobin measurements obtained at 2-hour intervals were 13.60 g/dL and 13.20 g/dL, respectively. Other hematological, biochemical, renal, and liver function tests were within normal limits.

As the patient had neither respiratory distress nor stridor, conservative management was preferred. She was observed for 24 hours under inpatient monitoring. No complications developed during follow-up, and the patient was discharged with outpatient follow-up recommendations. She was advised to monitor for stridor, dyspnea, or increasing neck swelling at home.

At outpatient follow-up, the patient was clinically asymptomatic, and thyroid function tests had returned to normal ranges.

Discussion

Thyroid injury secondary to blunt cervical trauma is rare and is usually observed in patients with pre-existing thyroid disorders such as goiter (3). Isolated thyroid gland injury is extremely uncommon and accounts for approximately 1–2% of cases (4). Although most reported cases in the literature involve underlying thyroid pathology, the absence of pre-existing thyroid disease in our patient makes this case noteworthy.

Symptoms of thyroid injury following blunt neck trauma may include neck swelling, respiratory distress, dysphagia, hoarseness, and neck pain. Patients may initially present in stable condition; however, delayed clinical deterioration may result in life-threatening complications. Previous reports have demonstrated that severe respiratory distress may develop up to 24 hours after the initial trauma (5,6). Although emergency intubation may not be necessary in every patient, close observation remains crucial because progressive hematoma formation or soft tissue edema may subsequently compromise the airway (5,6).

In some patients, rupture of thyroid follicles and subsequent release of thyroid hormones into the systemic circulation may lead to thyroid storm (4). Although the exact pathophysiology of thyroid storm remains unclear, several triggering factors have been implicated, including infections, major trauma, surgical procedures, and childbirth (7).

Radhi et al. reported a very low prevalence of thyroid storm among head and neck emergency patients and identified trauma as a rare triggering factor (8). Similarly, a large nationwide study conducted in Japan demonstrated that trauma accounted for approximately 3.9% of

thyroid storm triggers (9).
In our case, although mildly suppressed TSH and mildly elevated T3 and T4 levels were detected, the patient demonstrated no clinical findings suggestive of thyroid storm and remained hemodynamically stable throughout follow-up.

A review by von Ahnen et al. analyzed 36 cases of thyroid rupture, hemorrhage, or hematoma secondary to blunt cervical trauma and reported that 13 patients (42%) underwent surgical treatment, while histopathological abnormalities were identified in 11 of these surgically treated cases (10). In contrast, our patient was successfully managed conservatively without the need for surgical intervention.

Given the rarity of thyroid injuries following blunt trauma, no standardized management guidelines currently exist. Patients should be initially evaluated and resuscitated according to Advanced Trauma Life Support (ATLS®) principles, with particular attention to airway, breathing, and circulation management (11).

Conclusion

Although thyroid gland injuries following blunt cervical trauma are rare, they may lead to potentially life-threatening complications. Therefore, thyroid injury should always be considered in the differential diagnosis of patients presenting with neck swelling, pain, or respiratory symptoms after blunt neck trauma. Early diagnosis and appropriate monitoring are critical for preventing complications and avoiding unnecessary surgical intervention. Conservative management may represent a safe and effective treatment option in appropriately selected patients; however, close observation remains essential because of the risk of delayed airway obstruction.

Figures

Figure 1. Cervical ecchymosis and swelling secondary to blunt neck trauma.

Figure 2. Hypoechoic appearance suggestive of thyroid laceration on ultrasonography.

Figure 3. Doppler ultrasonography demonstrating hypervascularity of the thyroid gland with no vascular flow within the lesion.

Figure 4. Normal appearance of the remaining thyroid tissue.

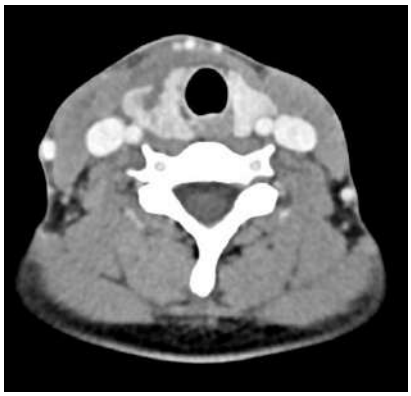

Figure 5. Contrast-enhanced neck CT demonstrating hypodense lesion compatible with right thyroid hematoma.

Figure 6. Contrast-enhanced neck CT confirming right thyroid laceration without airway or vascular injury.

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Gallstone Ileus with Spontaneous Rectal Evacuation via Enema: A Rare Case of Cholecystoduodenal Fistula

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INTRODUCTION

Gallstone ileus is a rare but potentially life-threatening complication of cholelithiasis, accounting for 1–4% of all mechanical intestinal obstructions. It is typically caused by the passage of a large gallstone into the intestinal lumen through a bilioenteric fistula, most commonly resulting from chronic cholecystitis (1,2). Although the ileocecal valve is the most frequent site of obstruction, gastric outlet obstruction due to a gallstone is referred to as Bouveret Syndrome. Complete passage of a gallstone through the gastrointestinal tract followed by rectal evacuation is exceedingly rare (3).
We present an unusual case in which a giant gallstone was evacuated rectally following an enema, resulting in resolution of the ileus, while an underlying 8 mm cholecystoduodenal fistula remained.

CASE

A 72-year-old male presented with a three-day history of generalized abdominal pain, bloating, and dark-colored vomiting. Initial evaluation revealed widespread air-fluid levels

and dilated colonic loops consistent with mechanical obstruction. Clinical relief was achieved following the administration of an enema.

Upon admission to our center, the patient's vital signs were stable. Laboratory results indicated significant leukocytosis (WBC: 18.73x10³/µL), neutrophilia (81.1%), and elevated CRP (143 mg/L). Contrast-enhanced CT scans revealed: An 8 mm cholecystoduodenal fistula between the gallbladder fundus and the duodenum. Pneumobilia within the intrahepatic bile ducts. increased wall thickness at the rectosigmoid junction and stranding in the mesorectal fatty planes, likely occurring during the rectal evacuation of the stone.

Although the obstruction was resolved via enema, intravenous broad-spectrum antibiotic therapy was initiated due to the risk of ascending cholangitis associated with the fistula and high inflammatory markers. An elective colonoscopy was subsequently planned.

DISCUSSION

Gallstone ileus is typically managed through surgical enterolithotomy (4). This case represents a rare example of "spontaneous resolution" involving the rectal evacuation of a giant stone originating from a cholecystoduodenal fistula following an enema. While enemas carry a risk of perforation in fragile bowel segments, in this instance, it successfully resolved the ileus (5).
However, even if the passage of the stone resolves the mechanical obstruction, the fistula and the inflammatory response persist. Clinicians must thoroughly evaluate the biliary system after the obstruction is cleared and closely monitor systemic inflammatory parameters.

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Keywords: Gallstone ileus; Cholecystoduodenal fistula; Pneumobilia; Therapeutic enema; Rigler's tria

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Inflammation-Based indices and functional outcomes after mechanical thrombectomy in acute ischemic stroke

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Introduction and Purpose

Mechanical thrombectomy is an established treatment for acute ischemic stroke caused by large vessel occlusion; however, functional outcomes remain heterogeneous despite successful recanalization [1]. Inflammatory processes have been increasingly implicated in post-stroke prognosis [2]. This study aimed to evaluate the association between inflammation-based indices and short-term functional outcomes in patients undergoing mechanical thrombectomy.

Materials and Methods

This retrospective observational study included patients who underwent mechanical thrombectomy for anterior circulation acute ischemic stroke. Patients receiving intravenous thrombolysis, those with posterior circulation involvement, and those presenting more than 8 hours after symptom onset were excluded. Functional outcome was assessed using the modified Rankin Scale (mRS) at discharge and dichotomized as favorable (mRS ≤3) and poor (mRS >3). Demographic characteristics, comorbidities, electrocardiographic rhythm, and admission laboratory parameters were recorded. Systemic inflammatory response index (SIRI), systemic immune-inflammation index (SII), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) were calculated. Comparisons were performed using Mann–Whitney U and Fisher's exact tests, as appropriate.

Results

A total of 18 patients with acute anterior circulation ischemic stroke who underwent mechanical thrombectomy were included. Patients receiving intravenous thrombolysis, posterior circulation strokes, and those presenting more than 8 hours after symptom onset were excluded. Favorable functional outcome (mRS ≤3) was observed in 8 patients (44.4%), while 10 patients (55.6%) had poor outcomes.

Patients with poor outcomes were significantly older compared with those with favorable outcomes (median 68 vs. 47 years, p=0.026). Atrial fibrillation was exclusively observed in

the poor outcome group (p<0.001). Other comorbidities, including diabetes mellitus, hypertension, and coronary artery disease, were similar between groups.

Inflammation-based indices were markedly higher in patients with poor functional outcomes. Median SIRI, SII, NLR, and PLR values were all significantly elevated in the poor outcome group compared with the favorable outcome group (all p<0.01) (**Table 1**).

A representative case demonstrating successful mechanical aspiration with complete reperfusion (TICI 3) is shown in **Figure 1.**

In-hospital mortality occurred in four patients, all within the poor outcome group. One patient developed intracranial hemorrhage and one patient experienced deep vein thrombosis during hospitalization.

Table 1. Baseline characteristics and inflammatory indices according to functional outcome

Variable	Favorable outcome (mRS ≤3, n=8)	Poor outcome (mRS >3, n=10)	*p* value
Age, years	47	68	**0.026**
Male sex, n (%)	5 (62.5)	3 (30.0)	0.168
Atrial fibrillation, n (%)	0 (0)	10 (100)	**<0.001**
Diabetes mellitus, n (%)	3 (37.5)	6 (60.0)	0.343
Hypertension, n (%)	3 (37.5)	7 (70.0)	0.168
Coronary artery disease, n (%)	3 (37.5)	5 (50.0)	0.596
SIRI	1.78	5.98	**<0.001**
SII	1034.8	2400.0	**0.001**
NLR	4.17	11.37	**0.001**
PLR	122.7	221.9	**0.006**

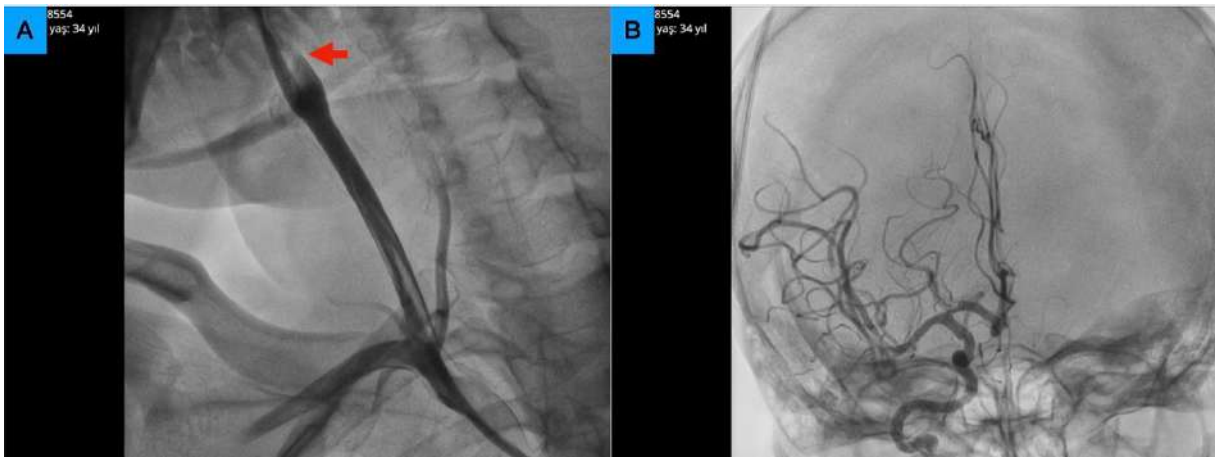

Figure 1. Representative digital subtraction angiography images of a patient with acute anterior circulation ischemic stroke. **(A)** Selective internal carotid artery angiography demonstrating total occlusion of the internal carotid artery (arrow). **(B)** Post-procedural angiography following mechanical aspiration showing successful reperfusion with TICI 3 flow in the distal internal carotid artery and its branches.

Conclusion

Inflammation-based indices measured at admission were strongly associated with poor short-term functional outcomes following mechanical thrombectomy. These findings are consistent with previous studies demonstrating the prognostic value of systemic inflammatory markers in acute ischemic stroke [3]. In particular, neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have been shown to reflect the balance between pro-inflammatory and anti-inflammatory responses and are associated with worse neurological outcomes [4].

Furthermore, composite indices such as the systemic immune-inflammation index (SII) and systemic inflammatory response index (SIRI) may provide a more comprehensive assessment of the inflammatory burden, integrating multiple hematological parameters [5]. These markers have been increasingly recognized as reliable predictors of prognosis in patients with ischemic stroke and may be useful in identifying high-risk patients in the early phase.

Keywords

Acute ischemic stroke; mechanical thrombectomy; inflammation; SIRI; SII; modified Rankin Scale

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Late-Onset Clozapine-Induced Agranulocytosis Presenting as Severe Febrile Neutropenia

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Introduction

Clozapine is an atypical antipsychotic indicated for treatment-resistant schizophrenia (1). Despite its efficacy, it carries a well-established risk of severe hematologic adverse effects, particularly agranulocytosis (1,4). Agranulocytosis is defined by a marked reduction in the absolute neutrophil count (ANC), predisposing patients to life-threatening infections and sepsis (3). For this reason, regular hematological monitoring is mandatory during clozapine therapy (4). We present a case of late-onset clozapine-induced agranulocytosis manifesting as severe febrile neutropenia.

Case

A 50-year-old woman with a 20-year history of schizophrenia was admitted to the emergency department (ED) with a one-week history of high fever and altered mental status. She had been receiving clozapine 300 mg/day for four years. On admission, her temperature was 39.4°C, heart rate 115 bpm, and blood pressure 114/73 mmHg.

Physical examination revealed bilateral rhonchi, an erythematous infected lesion on the right middle finger, and impaired orientation compared to baseline.

Laboratory investigations demonstrated profound pancytopenia: WBC 0.45 $\times 10^3/\mu L$, ANC 0.02 $\times 10^3/\mu L$, hemoglobin 8.8 g/dL, and CRP 32.7 mg/dL. Urinalysis was positive for nitrites and bacteria. Peripheral smear confirmed severe leukopenia and absence of neutrophils.

The patient was diagnosed with febrile neutropenia secondary to clozapine-induced agranulocytosis. Clozapine was immediately discontinued (4). Empirical broad-spectrum antibiotics (piperacillin/tazobactam) and granulocyte colony-stimulating factor (G-CSF) were initiated in the ED (2).

Discussion

Febrile neutropenia is a medical emergency requiring rapid etiological assessment (2). In psychiatric patients presenting with fever, drug-induced agranulocytosis—particularly due to clozapine—must be considered (4). Although the highest risk occurs within the first 18 weeks of treatment, late-onset hematologic toxicity can develop even after years of stable therapy

(1). Early discontinuation of clozapine and prompt initiation of antibiotics and G-CSF are critical to reducing morbidity and mortality (5).

Keywords: Agranulocytosis, Clozapine, Emergency Department, Febrile Neutropenia, Schizophrenia

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HIDDEN DANGER BEHIND SYNCOPE IN A PATIENT WITH DOWN SYNDROME: MASSIVE PULMONARY EMBOLISM

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Introduction

Syncope is a frequent emergency department presentation with a broad differential diagnosis, ranging from benign vasovagal episodes to life-threatening cardiovascular conditions. Pulmonary embolism (PE) is a rare but highly fatal cause of syncope. Diagnosis may be challenging, especially in patients with communication barriers, where classic symptoms such as chest pain or dyspnea cannot be reliably obtained.

Case

A 34-year-old male with Down syndrome presented with recurrent syncope episodes over the past 48 hours, each lasting 1–2 minutes with spontaneous recovery. He had previously been evaluated in neurology, and brain CT and MRI were normal.

On arrival to the emergency department, history was limited due to cognitive and communication difficulties. Vital signs showed hypotension (95/60 mmHg), tachycardia (124 bpm), tachypnea (24/min), and oxygen saturation of 91% on room air.

Physical examination played a key role and revealed loud P2, bilateral basal crackles, and left lower extremity swelling with a 3 cm increase in diameter and tenderness. Homans sign was positive, strongly suggesting deep vein thrombosis (DVT).

Arterial blood gas analysis demonstrated respiratory alkalosis with hypoxemia. D-dimer was markedly elevated (4850 ng/mL). ECG showed sinus tachycardia with right heart strain pattern. Troponin I was mildly elevated.

Based on clinical suspicion, the patient was classified as high probability for pulmonary embolism. CT pulmonary angiography confirmed a massive PE with near-complete occlusion of the right main pulmonary artery and RV/LV ratio >1, consistent with right ventricular strain.

The patient was started on therapeutic low-molecular-weight heparin and admitted to intensive care for close monitoring and possible thrombolysis if hemodynamic deterioration occurred.

Conclusion

Syncope may be the only presenting symptom of massive pulmonary embolism. In patients with limited communication ability, bedside physical examination is critical for diagnosis. Early recognition of DVT findings can rapidly guide life-saving management decisions.

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Diagnostic Profile of Tumor Lysis Syndrome in Oncologic Emergency Admissions: A Retrospective Comparative Study

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Introduction

Tumour lysis syndrome (TLS) is a term used to describe a range of metabolic complications that arise in patients with malignancies, either before or after treatment.[1] First described in

1929, this term refers to an oncological emergency characterised by the rapid and massive breakdown of malignant cells, which can sometimes be life-threatening.[1] The breakdown of these cells releases large amounts of potassium, phosphate and nucleic acids into the systemic circulation. The conversion of nucleic acids to uric acid leads to hyperuricaemia.[1,2] The key features of TLS are hyperuricaemia, hyperphosphataemia, secondary hypocalcaemia, hyperkalaemia and acute renal failure. Clinically, it can result in cardiac arrhythmia, seizures, multi-organ failure and even death.[2] The fact that the clinical and laboratory findings of TLS may overlap with those of other oncological emergencies complicates the diagnostic process and can lead to delays.[3] For this reason, TLS is defined in two ways, both clinically and in the laboratory, according to the Cairo-Bishop classification.[3] Although TLS is generally associated with haematological malignancies, it has been reported with increasing frequency in solid tumours in recent years.[4-6] This indicates that TLS is not limited to classic risk groups and may affect a broader patient population. A significant proportion of oncology patients initially present to the emergency department, and the early differentiation of TLS from other oncological emergencies is crucial for appropriate clinical management.[7] Although TLS has been extensively studied in the context of haematological malignancies, there are few studies that have comparatively assessed the diagnostic profile of TLS in oncology patients presenting to the emergency department against other oncological emergencies. This study aims to compare the demographic, clinical and laboratory characteristics of patients diagnosed with TLS in the emergency department setting with those of patients presenting with other oncological emergencies.

Keywords: Tumour lysis syndrome, oncological emergencies, emergency department

Materials and Methods:

This retrospective study was conducted in the emergency department of Dr. Abdurrahman Yurtaslan Ankara Oncology Training and Research Hospital between 2017 and 2020, following approval from the institutional ethics committee (2023-09/78). Patients younger than 18 years of age, those not receiving active chemotherapy and/or radiotherapy, and those not under follow-up at our institution were excluded from the study. A total of 1,593 patient records with a diagnosis of solid or hematological malignancy and receiving active oncological treatment were retrospectively reviewed. Among these, 761 patients who presented to the emergency department during the study period and were diagnosed with an oncologic emergency were included in the analysis. Tumor lysis syndrome (TLS) was diagnosed based on biochemical and clinical criteria defined in the literature, according to the Cairo–Bishop classification.[3]Patients were divided into two groups: those diagnosed with TLS (n=43) and those diagnosed with other oncologic emergencies (OE) (n=718). Demographic characteristics, cancer types, comorbidities, laboratory parameters at admission, and clinical outcomes were retrospectively obtained from the hospital information management system.

Statistical Analysis

All statistical analyses were performed using SPSS Statistics software (version 27.0; IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR; 25th–75th percentiles), depending on data distribution, while categorical variables were presented as frequencies (n) and percentages (%). Comparisons between groups were performed using Pearson's chi-square (χ^2) test or Fisher's exact test for categorical variables, as appropriate. Continuous variables were compared using the Mann–Whitney U test. A two-sided p-value of <0.05 was considered statistically significant**.**

Results:

A total of 761 patients diagnosed with oncologic emergencies were included in the study. A total of 761 patients diagnosed with oncologic emergencies were included in the study. The mean age was 58.63±12.97 years, and 51.5% of the patients were male while 44.5% were female. Solid malignancies were present in 90.8% (n=691) of the cases, whereas 9.2% (n=70) had hematological malignancies. Metastatic disease was identified in 89.6% of the patients. When the distribution of oncological emergency diagnoses was examined, the most common causes were found to be mechanical and blood-related emergencies (Table 1).

Table 1. Emergency diagnosis for patients with cancer admitted to the emergency service

Oncologic Emergencies (N=761)	(n/%)
Metabolic causes	**83 (11.0)**
TLS	43 (5.7)
Syndrome involving inappropriate ADH	22 (2.9)
Maling hypercalcemia	18 (2.4)
Mechanic causes	**361 (47.4)**
Malignant pleural effusion	128 (16.8)
Intestinal and biliary obstruction	91 (12.0)
Increased ICP	83 (10.9)
Cardiac tamponade	18 (2.4)
Pathological fracture	14 (1.8)
VCSS	12 (1.6)
Spinal cord compression	11 (1.4)
Hyper viscosity	4 (0.5)
Treatment-related causes	**20 (2.6)**
Hemorrhagic cystitis	9 (1.2)
Anaphylaxis/capillary leakage syndrome	9 (1.2)
Chemotherapeutic extravasation	2 (0.2)
Blood disorders	**347 (45.6)**
Febrile neutropenia and sepsis/septic shock	174 (22.9)
Anemia/leucopenia/thrombocytopenia	85 (11.2)

Acute bleeding/coagulopathy	45 (5.9)
Arterial or venous embolism	41 (5.4)
GVHD	2 (0.3)
Multiple oncologic emergency diagnoses	**50 (6.6)**

Total number of diagnoses: ADH: Anti-diuretic hormone, ICP: Intracranial pressure, VCSS: Vena cava superior syndrome, GVHD: Graft-versus-host disease, TLS: Tumor lysis syndrome

Of the study population, 5.7% (n=43) were diagnosed with tumor lysis syndrome (TLS), while 94.3% (n=718) were classified as other oncologic emergencies (OE). No significant differences were observed between the groups in terms of age and gender (p>0.05) Notably, all patients in the TLS group had solid malignancies, which was statistically significant compared to the OE group (p=0.026)(Table 2). The most common malignancies in the TLS group were lung, breast and gastric cancers. Comorbidity analysis showed that the overall rate of comorbid conditions was significantly higher in the TLS group (p=0.003). In particular, the prevalence of diabetes mellitus was markedly higher in the TLS group (p=0.006) (Table 2). All patients in the TLS group were in the metastatic stage, and 88.4% (n=38) had received chemotherapy within the past month. The most common presenting complaints were fatigue, lower extremity edema, nausea–vomiting, and dyspnea.

Table 2: Age, sex, cancer type, and comorbidities of the groups

	TLS (n=43)	Other OEs (n=718)	P
Age	62.0 (52.0-71.0)	59.0 (51.0-68.0)	.442*
Sex			.096†
Female	16 (37.2)	361 (50.3)	
Male	27 (62.8)	357 (49.7)	
Cancer type (n/%)			.026‡
Solid	43 (100.0)	648 (90.3)	
Hematologic	-	70 (9.7)	
Comorbidities (n/%)	34 (79.1)	402 (56.0)	.003†
Hypertension	17 (39.5)	284 (39.6)	.998†
DM	16 (37.2)	141 (19.6)	.006†
CAD	7 (16.3)	83 (11.6)	.352†
COPD	6 (14.0)	68 (9.5)	.335†
Asthma	2 (4.7)	16 (2.2)	.270‡

Mann-Whitney U test, †Pearson Chi-square test, ‡ Fisher's exact test, TLS: Tumor lysis syndrome, CAD: Coronary arterial disease, COPD: Chronic obstructive pulmonary disease

Laboratory findings showed that leukocyte count, neutrophil count, blood urea nitrogen (BUN), creatinine, uric acid, potassium, phosphorus, liver enzymes, C-reactive protein (CRP), and D-dimer levels were significantly higher in the TLS group. In contrast, sodium, calcium, total protein, and albumin levels were significantly lower (all p<0.05) (Table 3).

Table 3: Laboratory parameters of tumor lysis and other oncological emergency groups

	TLS (n=43)	Other OE (n=718)	P*
	Median (IQR; 25-75 percentiles)		
WBC (x10^3/mm^3)	15. 2 (9.7-20.0)	6.6 (2.1-11.3)	<0.001
Neutrophile (/mm^3)	12.2.0 (7.7-16.9)	4.4 (0.9- 8.5)	<0.001
Hb (g/dL)	10.8 (8.0-12.2)	10.4 (8.9-12.0)	.983
Platelet (x10^3/mm^3)	205.0 (81.0-287.0)	183.0 (96.0-300.0)	.786
Glucose (mg/dL)	115.0 (94.0-153.0)	114.0 (98.5-145.5)	.709
BUN (mg/dL)	65.0 (42.0-91.0)	17.0 (11.6-26.0)	<0.001
Creatinine (mg/dL)	2.5 (1.9-3.7)	0.7 (0.5-1.0)	<0.001
Uric acid (mg/dL)	10.1 (9.8-12.0)	5.1 (3.5-6.3)	<0.001
Na (mg/dL)	133.0 (128.8-137.3)	136.0 (133.0-138.0)	.015
K (mg/dL)	6.5 (6.2-6.8)	4.1 (3.7-4.6)	<0.001
Ca (mg/dL)	6.2 (6.1-7.0)	8.2 (7.7-8.9)	<0.001
P (mg/dl)	6.1 (5.7-7.1)	3.4 (2.5-4.2)	<0.001
Total protein (mg/dL)	5.0 (4.4-5.4)	5.5 (5.0-6.2)	<0.001
Albumin	2.3 (2.0-2.8)	3.1 (2.5-3.6)	<0.001
ALT (U/L)	39.0 (20.0-146.0)	19.0 (12.0-33.0)	<0.001
AST (U/L)	64.0 (29.0-183.0)	25.0 (17.0-44.0)	<0.001
GGT (U/L))	82.0 (50.0-255.0)	46.0 (23.5-111.0)	<0.001
Amylase (U/L)	89.0 (41.0-125.0)	46.0 (31.5-76.5)	<0.001
Total bilirubin (mg/dL)	1.1 (0.5-2.8)	0.7 (0.4-1.1)	.005
Direct bilirubin (mg/dL)	0.4 (0.1-1.8)	0.2 (0.1-0.4)	.002
CRP (mg/dL)	222.5 (139.8-254.5)	90.0 (25.0-174.0)	<0.001
D-Dimer (ng/mL)	7040.0 (2617.5-12000.0)	2597.0 (1298.0-4400.0)	.003

** Mann-Whitney U test, TLS: Tumor lysis syndrome*

Regarding emergency department outcomes, the rate of direct discharge was significantly lower in the TLS group (p=0.005). The overall mortality rate in the TLS group was 67.4%, which was significantly higher than that observed in the OE group (p<0.001) (Table 4).

Table 4: Outcomes of the groups in the emergency service and overall outcomes

	TLS (n=43)	Other OEs (n=718)	P
Outcome in ES			.005*
Discharge from ES	1 (2.3)	127 (17.7)	
Hospitalization	42 (97.7)	591 (82.3)	
Overall outcome			<0.001†
Discharge from the hospital	14 (32.6)	584 (81.3)	
Mortality	29 (67.4)	134 (18.7)	

Fisher's exact test, †Pearson Chi-square test, TLS: Tumor lysis syndrome

Discussion

The reported incidence of tumor lysis syndrome (TLS) varies depending on the patient population and clinical setting and is generally estimated to be between 3% and 7%.[2,7] In the present study, the incidence of TLS was 5.7%, which is consistent with the existing literature. Although TLS has traditionally been associated with hematological malignancies, an increasing number of studies have demonstrated that it can also occur in patients with solid tumors.[5] Gemici et al. emphasized that TLS in solid tumors, although rare, represents a clinically significant condition.[6] One of the most striking findings of our study is that all patients in the TLS group had solid malignancies. The most common tumor types were lung, breast, and gastric cancers. While this finding is partially consistent with previous reports describing TLS in metastatic solid tumors such as breast and lung cancer, the absence of hematological malignancies in our TLS cohort may be attributed to the relatively low number of such patients in our study population and the heterogeneity of included malignancies.[5,6] In terms of comorbidities, the overall burden of comorbid disease, particularly diabetes mellitus, was significantly higher in the TLS group. Although the association between TLS and comorbidities has not been clearly established in the literature, impaired renal functional reserve has been suggested to exacerbate TLS-related complications.[4,5] This may indicate that chronic conditions such as diabetes mellitus could negatively influence TLS prognosis, potentially contributing to worse clinical outcomes. Laboratory findings in our study strongly support the classical biochemical profile of TLS. Elevated levels of uric acid, potassium, and phosphorus, along with decreased calcium levels, were consistent with previous reports.[1,2] In addition, the marked deterioration in renal function parameters further highlights the close relationship between TLS and acute kidney injury. Notably, inflammatory markers such as CRP, leukocyte count, and D-dimer were also significantly elevated in the TLS group. This finding suggests that TLS may not solely represent a metabolic derangement but may also be associated with a pronounced systemic inflammatory response. Consequently, TLS may clinically mimic conditions such as sepsis, potentially leading to diagnostic delays in the emergency setting. The most remarkable finding of this study is the markedly elevated mortality rate of 67.4% in the TLS group, which is substantially higher than the 15–30% mortality reported in the literature.[2–4] This discrepancy may be explained by the fact that our study population consisted of patients presenting to the emergency department, the majority of whom had advanced-stage metastatic disease. In addition, the low rate of direct discharge and high hospitalization rate among TLS patients further support the severity and acute nature of this condition.

Conclusion

In conclusion, TLS in patients presenting with oncologic emergencies to the emergency department is associated with distinct clinical and laboratory characteristics and significantly higher mortality compared to other oncologic emergencies. Careful evaluation of routine laboratory parameters, particularly in patients with solid malignancies, may facilitate early recognition of TLS. Early identification and prompt management are crucial for improving clinical outcomes in this high-risk patient population.

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Oxygen-Resistant Cyanosis: A Case of Suspected Methemoglobinemia in a Secondary Care Emergency Department Confirmed by Co-oximetry at a Tertiary Center

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Introduction

Methemoglobinemia is a dyshemoglobinemia caused by oxidation of hemoglobin iron from the ferrous (Fe^{2+}) to the ferric (Fe^{3+}) state, resulting in functional hypoxia. MetHb levels are

normally below 1–2%, while higher levels may lead to cyanosis and systemic symptoms. Acquired forms are most commonly associated with local anesthetics, particularly benzocaine. In the emergency setting, cyanosis unresponsive to oxygen therapy, combined with discrepancy between pulse oximetry and arterial blood gas findings, suggests dyshemoglobinemia and raises suspicion for saturation gap.

Case Presentation

A 32-year-old male presented with dyspnea and lip cyanosis 30 minutes after a dental procedure involving topical benzocaine. Vital signs were: blood pressure 124/78 mmHg, heart rate 108/min, respiratory rate 22/min, temperature 36.8°C, and SpO_2 83% on room air. The patient was conscious, with grayish discoloration of the lips and nail beds. Lung examination was normal.

Oxygen therapy was initiated via a non-rebreather mask at 10 L/min. Arterial blood gas analysis revealed pH 7.40, $PaCO_2$ 37 mmHg, and PaO_2 172 mmHg. Despite high calculated oxygen saturation, pulse oximetry remained at 84–85%, suggesting dyshemoglobinemia and raising suspicion for saturation gap.

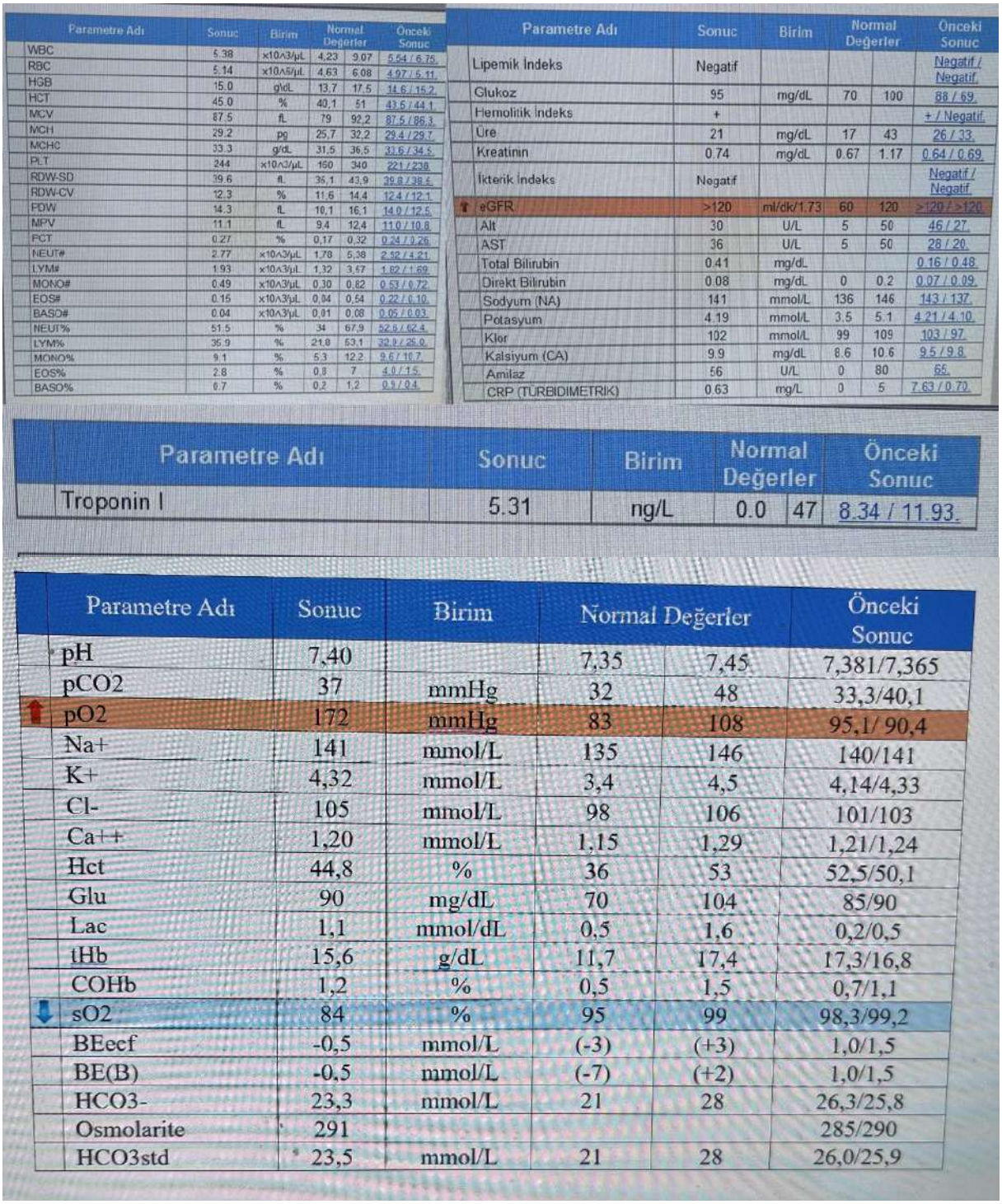
The patient was referred to a tertiary center. External records revealed a MetHb level of 18%. After methylene blue therapy in the intensive care unit, clinical improvement was observed, and the patient was discharged within 24 hours.

Discussion

Methemoglobinemia causes functional hypoxia and may mimic pulmonary disorders. Cyanosis typically appears when MetHb exceeds 10%, while symptoms such as dyspnea may occur at higher levels. Acquired cases are most commonly associated with benzocaine exposure. Pulse oximetry often shows values around 82–86% despite normal or elevated PaO_2, creating discrepancy suggestive of dyshemoglobinemia (saturation gap). Definitive diagnosis is established by co-oximetry, and methylene blue is recommended in symptomatic patients or when MetHb exceeds 20%.

Conclusion

Methemoglobinemia should be considered in patients with cyanosis unresponsive to oxygen therapy despite normal or elevated PaO_2. In settings where co-oximetry is unavailable, early referral based on clinical suspicion is essential. This case highlights that discrepancy between pulse oximetry and arterial blood gas findings is a key diagnostic clue in the emergency department.



Parametre Adı	Sonuc	Birim	Normal Değerler		Önceki Sonuc
WBC	5.38	x10^3/µL	4.23	9.07	5.54 / 6.75.
RBC	5.14	x10^6/µL	4.63	6.08	4.97 / 5.11.
HGB	15.0	g\dL	13.7	17.5	14.6 / 15.2.
HCT	45.0	%	40.1	51	43.6 / 44.1
MCV	87.5	fL	79	92.2	87.5 / 86.3.
MCH	29.2	pg	25.7	32.2	29.4 / 29.7.
MCHC	33.3	g/dL	31.5	36.5	33.6 / 34.5.
PLT	244	x10^3/µL	150	340	221 / 230.
RDW-SD	39.6	fL	36.1	43.9	39.8 / 38.6.
RDW-CV	12.3	%	11.6	14.4	12.4 / 12.1.
PDW	14.3	fL	10.1	16.1	14.0 / 12.5.
MPV	11.1	fL	9.4	12.4	11.0 / 10.8.
PCT	0.27	%	0.17	0.32	0.24 / 0.26.
NEUT#	2.77	x10^3/µL	1.78	5.38	2.52 / 4.21.
LYM#	1.93	x10^3/µL	1.32	3.57	1.62 / 1.69.
MONO#	0.49	x10^3/µL	0.30	0.82	0.53 / 0.72.
EOS#	0.15	x10^3/µL	0.04	0.54	0.22 / 0.10.
BASO#	0.04	x10^3/µL	0.01	0.08	0.05 / 0.03.
NEUT%	51.5	%	34	67.9	52.6 / 62.4.
LYM%	35.9	%	21.8	53.1	32.9 / 25.0.
MONO%	9.1	%	5.3	12.2	9.6 / 10.7.
EOS%	2.8	%	0.8	7	4.0 / 1.5.
BASO%	0.7	%	0.2	1.2	0.9 / 0.4.

Parametre Adı	Sonuc	Birim	Normal Değerler		Önceki Sonuc
Lipemik İndeks	Negatif				Negatif / Negatif.
Glukoz	95	mg/dL	70	100	88 / 69.
Hemolitik İndeks	+				+ / Negatif.
Üre	21	mg/dL	17	43	26 / 33.
Kreatinin	0.74	mg/dL	0.67	1.17	0.64 / 0.69.
İkterik İndeks	Negatif				Negatif / Negatif.
eGFR	>120	ml/dk/1.73	60	120	>120 / >120.
Alt	30	U/L	5	50	46 / 27.
AST	36	U/L	5	50	28 / 20.
Total Bilirubin	0.41	mg/dL			0.16 / 0.48.
Direkt Bilirubin	0.08	mg/dL	0	0.2	0.07 / 0.09.
Sodyum (NA)	141	mmol/L	136	146	143 / 137.
Potasyum	4.19	mmol/L	3.5	5.1	4.21 / 4.10.
Klor	102	mmol/L	99	109	103 / 97.
Kalsiyum (CA)	9.9	mg/dL	8.6	10.6	9.5 / 9.8.
Amilaz	56	U/L	0	80	65.
CRP (TURBIDIMETRIK)	0.63	mg/L	0	5	7.63 / 0.70.

Parametre Adı	Sonuc	Birim	Normal Değerler		Önceki Sonuc
Troponin I	5.31	ng/L	0.0	47	8.34 / 11.93.

Parametre Adı	Sonuc	Birim	Normal Değerler		Önceki Sonuc
pH	7,40		7,35	7,45	7,381/7,365
pCO2	37	mmHg	32	48	33,3/40,1
pO2	172	mmHg	83	108	95,1/ 90,4
Na+	141	mmol/L	135	146	140/141
K+	4,32	mmol/L	3,4	4,5	4,14/4,33
Cl-	105	mmol/L	98	106	101/103
Ca++	1,20	mmol/L	1,15	1,29	1,21/1,24
Hct	44,8	%	36	53	52,5/50,1
Glu	90	mg/dL	70	104	85/90
Lac	1,1	mmol/dL	0,5	1,6	0,2/0,5
tHb	15,6	g/dL	11,7	17,4	17,3/16,8
COHb	1,2	%	0,5	1,5	0,7/1,1
sO2	84	%	95	99	98,3/99,2
BEecf	-0,5	mmol/L	(-3)	(+3)	1,0/1,5
BE(B)	-0,5	mmol/L	(-7)	(+2)	1,0/1,5
HCO3-	23,3	mmol/L	21	28	26,3/25,8
Osmolarite	291				285/290
HCO3std	23,5	mmol/L	21	28	26,0/25,9

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Emergency Department Rabies Vaccinations: An Epidemiological Study at Kars University Hospital

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Abstract

Objective: Rabies is a viral zoonotic disease, mostly transmitted from rabid dogs, that causes encephalomyelitis in humans and is fatal. This study aimed to investigate the epidemiology of cases who presented to the emergency department due to suspected rabies animal contact and received rabies vaccination. Materials and Methods: The gender, age, mode of contact with suspected rabies animal, animal species, ownership and vaccination status of the animals, wound care, and whether vaccination or immunoglobulin was administered were retrospectively examined in patients who presented to the emergency department. Results: The study was completed with a total of 107 cases, 67 men and 40 women. The animals causing suspected contact were 39 dogs, 36 cats, 9 foxes, 8 horses, 6 bears, 6 sheep, and 5 cows. Of these animals, 36.4% were stray and unvaccinated, while 33.6% were owned and vaccinated. The rate of escaped animals was 23.4%, while those that were owned and vaccinated were very few (6.5%). Thirty-four cases received only vaccination (rabies, tetanus, or rabies+tetanus). Forty-two patients received wound care and vaccination prophylaxis, while 31 patients received immunoglobulin treatment. Conclusion: It was determined that contact with risky animals was most common in young and middle-aged men. These animals are usually stray, unvaccinated, or escaped. Taking stray street animals into shelters under veterinary control and raising awareness about rabies could significantly reduce potential cases.

Keywords: Rabies; suspected bite, animal, vaccine

Introduction

Rabies is an acute viral zoonotic disease that causes fatal encephalomyelitis in humans (1). This disease, which causes approximately 60,000 deaths annually, is caused by rabid dogs in 99% of cases. Wild animals such as wolves, foxes, jackals, wild cats, skunks, and weasels cause the disease, while domestic animals, primarily dogs, but also cats, cattle, sheep, goats, horses, and donkeys, are responsible (2). Transmission occurs through bites or scratches from an infected animal, resulting in a breach of skin integrity, or through contact with saliva from a wounded area (3). Proper wound care, the first step in treatment, significantly reduces the

risk of rabies. Early prophylaxis is necessary in all suspected rabies exposures. This includes tetanus vaccine, rabies vaccine, and/or rabies immunoglobulin (Ig).

Results

Of the 107 cases that presented to our center, 67 (62.6%) were male and 40 (37.4%) were female. The average age of the cases was 31.5 ± 13.2 years (min: 16 years, max: 70 years). Of the patients, 30 (28%) had minor abrasions, 29 (27.1%) had scratches, 26 (24.3%) had bites, 13 (12.1%) had both bites and scratches, and 9 (8.4%) had contact with saliva. It was determined that 39 (36.4%) of the animals contacted were stray and unvaccinated. 36 (33.6%) animals were owned and unvaccinated, while 7 (6.5%) animals were both owned and vaccinated. Information could not be obtained about 25 (23.4%) animals as they had escaped. Of the animals that caused suspected contact, 39 cases were identified as dogs (36.4%), 36 cases as cats (33.6%), 9 cases as foxes (8.4%), 8 cases as horses (7.5%), 6 cases as bears (5.6%), 6 cases as sheep (5.6%), and 5 cases as cows (4.7%) (Table 1).

Table 1. Animal species, types of injury, ownership status, and medical interventions

Variable	Category	n	%
Animal Groups	Dog	39	36.4
	Cat	36	33.6
	Fox	9	8.4
	Sheep	6	7.5
	Horse	8	7.5
	Bear	5	4.7
	Cow	4	3.7
Type of Injury	Minor scrape	30	28.0
	Scratch/bruise	29	27.1
	Bite	26	24.3
	Bite + scratch	13	12.1
	Saliva contact	9	8.4
Animal's Status	Unowned, unvaccinated	39	36.4
	Owned, unvaccinated	36	33.6
	Escaped/unidentified	25	23.4
	Owned, vaccinated	7	6.5

Table 2. Animal species and post-exposure treatment approaches

Animal Groups	Vaccine Treatment Only n (%)	Vaccination + Wound Care n (%)	Ig-Containing Treatment n (%)	Total
Dog	8 (21.6)	14 (37.8)	15 (40.5)	37
Cat	12 (33.3)	16 (44.4)	8 (22.2)	36

Other (Fox Sheep, Cow, Horse,Bear)	14 (41.2)	12 (35.3)	8 (23.5)	34
Total	34 (31.8)	42 (39.3)	31 (29.0)	107

Of the cases presenting to the emergency department, 34 (31.8%) received only vaccination (rabies, tetanus, or rabies+tetanus). 42 (39.3%) received wound care and vaccination prophylaxis, while 31 (29%) received immunoglobulin treatment. The rate of immunoglobulin-containing treatment in dog contact cases (40.5%) was descriptively high compared to other groups (Table 2).

Table 3. Animal ownership and vaccination status, and immunoglobulin administration rates

Animal's Status	Ig Absent n (%)	Ig Present n (%)	Total
Unowned, unvaccinated	21 (60.0)	14 (40.0)	35
Escaped/unidentified	14 (58.3)	10 (41.7)	24
Owned, unvaccinated	26 (81.3)	6 (18.8)	32
Owned, vaccinated	15 (93.8)	1 (6.3)	16
Total	76 (71.0)	31 (29.0)	107

There is a statistically significant relationship between the animal's condition and immunoglobulin use (p=0.020). The rate of immunoglobulin use was significantly higher in cases involving stray/unvaccinated (40.0%) and escaped/unidentified (41.7%) animals compared to owned/vaccinated (6.3%) cases (Table 3).

Table 4. Statistical tests and comparisons

Comparison	p value
Gender ↔ Animal Species	0.058
Gender ↔ Type of Injury	0.877
Gender ↔ Animal Condition	0.12
Animal Species ↔ Treatment	0.03
Type of Injury × Immunoglobulin	0.038
Animal Condition × Immunoglobulin	0.020

There was no statistically significant relationship between the type of animal that came into contact with male and female patients, the mode of contact, and animal ownership (p>0.05). There was a statistically significant relationship between the type of injury (contact) and immunoglobulin (Ig) use (p=0.038). Regarding the relationship between animal type and treatment, Ig use was higher in those who had contact with dogs (Table 4).

Discussion

In the study conducted by Canpolat et al., it was observed that the male gender ratio was higher in patients who received prophylactic treatment due to suspected rabies (4). Important reasons for the higher male patient ratio in our study include the fact that men use open spaces more for work and social activities; they are more involved in animal husbandry and therefore have more contact with animals.

In a 4-year study, the average age of the rabies-risk animal contact patient population was found to be 23.7 ± 15.8 years (5). In our study, since the younger age group was predominant, a similar rate of 31.5 years was observed. This shows that the majority of cases presenting to the emergency department are afraid of rabies after contact or bites and believe that medical measures should be taken quickly. In our study, contact with dogs constituted 36% of the cases. In the study conducted by Temiz et al., it was observed that in patients vaccinated due to suspected rabies, 75.4% of the animals they contacted were owned and 24.6% were unowned (6). In another study, it was found that 31.3% of the animals suspected of having rabies and contacted were stray, while 68.7% were owned (7). In contrast, in the study conducted by Şengöz et al., it was found that 70% of the animals suspected of having rabies and contacted were stray (8). Similarly, in our study, the majority of the animals suspected of having rabies and contacted (60%) were stray animals. This result suggests that the relevant institutions should follow up and pay more attention to the control of stray animals in our region.Furthermore, in our study, Ig use was preferred in cases involving contact with stray/unvaccinated or escaped animals, as well as in cases of serious injuries resulting from bites. A study conducted in Turkey showed that approximately 30% of all suspected rabies cases received Ig treatment (9). The most important factor that changed this ratio was whether all cases had been vaccinated against rabies. Patients who had not received the rabies vaccine were excluded from the study.

Conclusion

Rabies-carrying animals posing a threat to human health is a significant public health problem in our country. We observe that contact with high-risk animals is very common among young and middle-aged men, and these animals are generally stray and/or unvaccinated, as well as escaped animals. We believe that raising awareness about rabies and taking stray street animals into shelters under veterinary supervision will significantly reduce potential rabies cases.

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INVESTIGATION OF THE EFFECT OF INFLAMMATORY INDEXES AND SHOCK INDEXES ON CLINICAL COURSE AND PROGNOSIS IN PULMONARY EMBOLISM

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Abstract

Introduction: Pulmonary embolism (PE) is a life-threatening condition caused by thrombi arising from the deep veins of the lower extremities and causing obstruction in the pulmonary artery and/or its branches. This study investigated the effect of the systemic immune-inflammatory index (SII), systemic inflammatory response index (SIRI), shock index (SI), and modified shock index (MSI) on the clinical course and prognosis in PE patients.

Materials and Methods: The study was conducted using data from a single hospitalization episode of adult patients with a diagnosis of PE confirmed by CTPA. A total of 259 adult patients were included in the study. The data consisted of age, gender, vital signs

(temperature, pulse, blood pressure, SpO_2), comorbidities, presenting complaints, and hematological parameters (complete blood count, biochemistry, CRP, blood gas, lactate, D-dimer, troponin, etc.). In addition, SIRI, SII, SI, and MSI values were calculated and added to the data. The days of admission to the ward and intensive care unit were recorded for the patients. Patient mortality status was assessed, and the study population was stratified into two groups: survivors and non-survivors. Furthermore, the mortality status of the patients was divided into three groups based on time: survivors, early mortality (≤28 days), and late mortality (>28 days).

Results: Mortality occurred during hospitalization in 36 patients. Mortality occurred in the early period (≤28 days) in 30 patients and in the late period (>28 days) in 6 patients. When comparing the survivors and non survivors, the mortality group had a noticeably higher age. Regarding the length of stay, the intensive care unit stay was significantly longer in the deceased patients, while the ward stay was noticeably shorter in these patients. When comparing blood parameters between the two groups, white blood cells (WBC), monocytes, glucose, aspartate aminotransferase (AST), lactate dehydrogenase (LDH), D-dimer, lactate, and urea were significantly higher in the mortality group; hemoglobin (Hb), lymphocytes, albumin, calcium (Ca), and pH were significantly lower. In addition, SII, SIRI, SI, and MSI were noticeably higher in the mortality group.

Conclusion: SII, SIRI, SI, and MSI are important parameters for predicting the prognosis of patients with PE. These parameters are readily accessible and cost-effective, thereby rendering them favorable for routine clinical use.

Keywords: Inflammatory indices, mortality, pulmonary embolism, shock indices

Introduction

Pulmonary embolism (PE) is a life-threatening clinical condition that typically arises when thrombi from the deep veins of the lower extremities travel to the lungs, causing vascular obstruction (1). It is the third most common acute cardiovascular event worldwide after myocardial infarction (MI) and stroke (2). Increased coagulation, venous stasis, endothelial damage, and inflammation play a role in its pathogenesis (3). These three factors commonly coexist in the majority of patients with pulmonary embolism (4). The clinical findings varies depending on the thrombus load, demographic characteristics, and time of presentation, but the symptoms and findings are nonspecific (5). Diagnosis begins with clinical suspicion, particularly in patients with tachycardia and dyspnea (6).

Parameters indicating systemic inflammation in PE and biomarkers used for early risk classification are prominent (7,8). The systemic immune inflammatory index (SII) and systemic inflammatory response index (SIRI) are used to determine the state of inflammation (7,9). The shock index (SI) and modified shock index (MSI) are biomarkers used to predict

early risk status in patients (8). SIRI: Monocyte x Neutrophil/Lymphocyte, SII: Platelet x Neutrophil/Lymphocyte, SI: Heart Rate (HR)/Systolic Blood Pressure (SBP), and MSI: Heart Rate/Mean Arterial Pressure (MAP) are calculated using these formulas (7,9,10).

In recent years, studies on these indices have increased, and noninvasive markers are being investigated. In our study, we considered these factors and aimed to evaluate the effect of SII, SIRI, SI, and MSI on the clinical course and prognosis of PE and to contribute to the literature.

Materials and Methods

The study was initiated after obtaining ethics committee approval from the Non-Interventional Research Ethics Committee of Elazığ Fethi Sekin City Hospital on June 12, 2025, with session number 2025/11-19. Adult patients admitted to the Chest Diseases Service, Chest Diseases Intensive Care Unit, and Anesthesia Resuscitation Intensive Care Units between January 1, 2019, and December 31, 2023, were retrospectively screened from the hospital system. After exclusion criteria, 259 PE patients hospitalized from the Emergency Medicine Clinic were included in the study. The study was conducted using data from a single hospitalization episode of adult patients with a PE diagnosis confirmed by CTPA. A total of 259 adult patients were included in the study. Data included age, gender, vital signs (temperature, pulse, blood pressure, SpO2), comorbidities, presenting complaints, and hematological parameters (complete blood count, biochemistry, CRP, blood gas, lactate, D-dimer, troponin, etc.). In addition, SIRI, SII, SI, and MSI values were calculated and added to the data. The days of admission to the ward and intensive care unit were recorded for the patients. The mortality status of the patients was examined.

Statistical Analysis

All statistical analyses were performed using R software (v4.5.1; stats, pROC, rms, rmda) and revalidated using Python (v3.10). This dual-platform approach was chosen to enhance the reproducibility of results and computational reliability. The distribution of continuous variables was assessed using the Shapiro–Wilk test; normally distributed data were summarized as mean ± standard deviation, and non-normally distributed data as median (Q1–Q3). Categorical variables were presented as counts and percentages. Independent samples t-test or Mann–Whitney U test was used for group comparisons, and chi-square or Fisher's exact test was used for categorical variables. The relationship of variables with mortality was evaluated using univariate logistic regression analysis. Significant variables were included in the multivariate model. ROC curves and AUC values were calculated to analyze discriminative ability. Additionally, the obtained AUC values were compared using the DeLong test. Based on the obtained p-values, $p<0.05$ was considered statistically significant.

Results

The study included 259 (100%) adult patients with a PE diagnosis confirmed by CTPA based on clinical and laboratory findings. Of these patients, 95 (37%) were male and 164 (63%) were female. Of the patients included in the study, 223 (86%) survived, while 36 (14%) died. Age was remarkably higher in the mortality group. Gender distribution was similar between groups. In the mortality group, SpO2 was noticeably lower, respiratory rate was remarkably higher, length of hospital stay was noticeably shorter, and length of intensive care unit stay was significantly longer (Table 1).

Table 1. Comparison of age, vital signs, and length of stay between surviving and deceased patients

Variable	Survivors (mean±SD) n:223	Mortality (mean±SD) n:36	p
Age	69.27±15.55	79.31±11.65	**<0.001**
Systolic blood pressure	126.65±23.84	116.97±32.80	>0.05
Diastolic blood pressure	77.25±14.67	70.83±18.56	>0.05
MAP	93.72±16.19	86.21±21.86	>0.05
Fever	36.53±0.54	36.49±0.48	>0.05
Pulse	97.44±22.05	106.78±21.21	<0.05
SpO2	89.06±7.47	84.33±9.61	**<0.01**
Respiratory rate	18.70±3.02	20.28±4.39	**<0.05**
Service admission day	9.48±7.00	6.00±15.86	**<0.001**
ICU admission day	4.15±12.40	20.89±42.57	**<0.001**
Total admission days	13.56±13.80	26.86±44.28	>0.05

MAP: Mean arterial pressure, ICU: Intensive care unit

In the mortality group, white blood cells (WBC), monocytes, glucose, aspartate aminotransferase (AST), urea, lactate dehydrogenase (LDH), D-dimer, and lactate levels were noticeably elevated, while hemoglobin (Hb), lymphocytes, calcium (Ca), albumin, and pH levels were noticeably lower. There were no statistically significant differences between the groups in other hematological parameters (Table 2).

Table 2. Comparison of hematological parameters between survivors and patients who died.

Variable	Survivors (mean±SD) n:223	Mortality (mean±SD) n:36	p
WBC	10.66 ± 4.02	12.96 ± 5.55	**<0.05**
Hb	13.73 ± 9.82	12.47 ± 2.12	**<0.05**
Plt	247.76 ± 99.46	240.42 ± 101.08	>0.05
Neutrophil	7.55 ± 4.14	10.13 ± 4.47	>0.05
Lymphocyte	1.74 ± 1.16	0.93 ± 0.62	**<0.05**
Monocyte	0.96 ± 0.73	1.70 ± 1.39	**<0.05**
Glucose	148.11 ± 63.78	166.17 ± 53.00	**<0.05**
AST	32.96 ± 35.29	75.39 ± 137.11	**<0.01**
ALT	27.39 ± 30.76	54.06 ± 96.95	>0.05
Üre	45.06 ± 21.45	73.49 ± 42.06	**<0.001**
Na	137.28 ± 3.74	138.56 ± 8.52	>0.05
Cl	104.23 ± 3.76	103.81 ± 7.79	>0.05
K	4.53 ± 2.81	4.59 ± 0.84	>0.05
Ca	8.99 ± 0.58	8.51 ± 0.73	**<0.001**
LDH	330.58 ± 165.23	448.91 ± 298.74	**<0.001**
Albumin	37.37 ± 21.35	32.92 ± 5.23	**<0.01**
CRP	60.06 ± 71.65	81.89 ± 78.97	>0.05
D-dimer	5.33 ± 4.51	8.46 ± 14.51	**<0.05**
Hs-TnI	170.98 ± 356.03	225.41 ± 432.58	>0.05
pH	7.38 ± 0.07	7.33 ± 0.16	**<0.05**
pCO2	42.25 ± 7.75	45.82 ± 15.97	>0.05
pO2	48.26 ± 22.78	48.02 ± 29.26	>0.05
HCO3	25.30 ± 3.62	24.78 ± 8.06	>0.05
Lactate	2.15 ± 1.57	3.63 ± 2.84	**<0.001**

WBC: White blood cell, Hb: Hemoglobin, Plt: Platelet, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, Na: Sodium, Cl: Chloride, K: Potassium, Ca: Calcium, LDH: Lactate dehydrogenase, CRP: C-reactive protein, Hs-TnI: High Sensitivity Troponin I

When comparing SIRI, SII, SI, and MSI values between the surviving and mortality groups, all four scores were significantly higher in the mortality group (Table 3).

Table 3. Comparison of SIRI, SII, SI, and MSI in surviving and mortality patients

Index	Survivors (mean±SD) n:223	Mortality (mean±SD) n:36	p
SIRI	5.85±7.13	13.02±17.76	**<0.001**
SII	1732.66±1705.22	2713.73±2239.71	**<0.01**
SI	0.80±0.25	1.00±0.39	**<0.001**

MSI	1.07±0.31	1.34±0.53	**<0.001**

SIRI: Systemic Inflammatory Response Index, SII: Systemic Immune-Inflammatory Index, SI: Shock Index, MSI: Modified Shock Index

The performance of the indices (SIRI, SII, SI, MSI) in distinguishing mortality was evaluated using ROC analysis. AUC and 95% CI values were obtained using bootstrap (B=1000), and a two-tailed test was applied for the H0: AUC=0.5 hypothesis (SIRI, SII, SI, and MSI affect prognosis and clinical course in PE). The results are presented in Table 4, and the relevant curves are shown in Figure 1.

In the ROC analysis, SIRI had the highest AUC value, followed by SI and MSI, respectively. This suggests that SIRI reflects the inflammatory burden more accurately than the other indices. However, the AUC values of all indices were moderate. This indicates that these parameters alone have limited power in predicting mortality. The findings reveal that while systemic inflammation is important in the course of PE, it must be supported by multiparametric models; or we can say that these scores are not sufficient for decision-making alone but can strengthen clinical evaluation (Table 4, Figure 1).

Table 4. ROC analyses: Characteristic analysis of SIRI, SII, SI, and MSI for mortality prediction

Index	AUC (95% CI)	p	n
SIRI	0.707 (0.608-0.789)	**<0.001**	259
SII	0.651 (0.553-0.748)	**<0.01**	259
SI	0.676 (0.574-0.775)	**<0.01**	259
MSI	0.679 (0.571-0.781)	**<0.01**	259

SIRI: Systemic Inflammatory Response Index, SII: Systemic Immune-Inflammatory Index, SI: Shock Index, MSI: Modified Shock Index

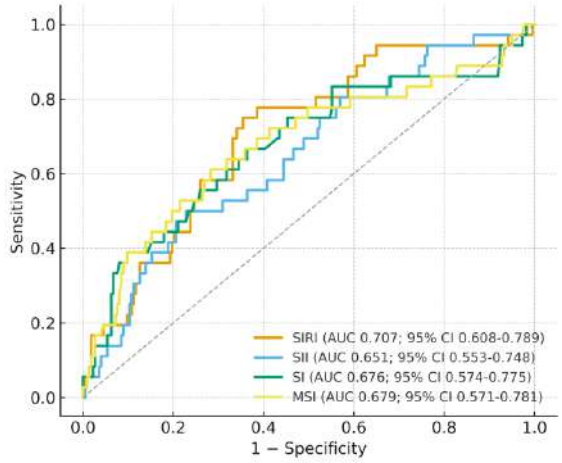

Figure 1. ROC curves: Discrimination of SIRI, SII, SI, and MSI indices for in-hospital mortality (AUC and 95% CI; bootstrap B=1000)

Age and gender were retained as mandatory covariates in the multivariate logistic model. Only age remained independently significant in this multivariate model.

The model's LR test was significant ($p<0.001$) and McFadden pseudo-R^2 = 0.208. This level indicates average adequacy in explaining mortality (Table 5, Figure 2). Thus, age is the independent predictor in the model. The indices and gender did not provide additional contribution when other variables were controlled.

Table 5. Multivariate logistic regression

Variable	OR (95% CI)	p
Yaş	2.27 (1.32-3.90)	**<0.01**
Cinsiyet	0.41 (0.15-1.08)	>0.05
SIRI	1.45 (0.91-2.33)	>0.05
SII	1.18 (0.77-1.82)	>0.05
SI	1.07 (0.30-3.76)	>0.05
MSI	1.76 (0.50-6.21)	>0.05

SIRI: Systemic Inflammatory Response Index, SII: Systemic Immune-Inflammatory Index, SI: Shock Index, MSI: Modified Shock Index

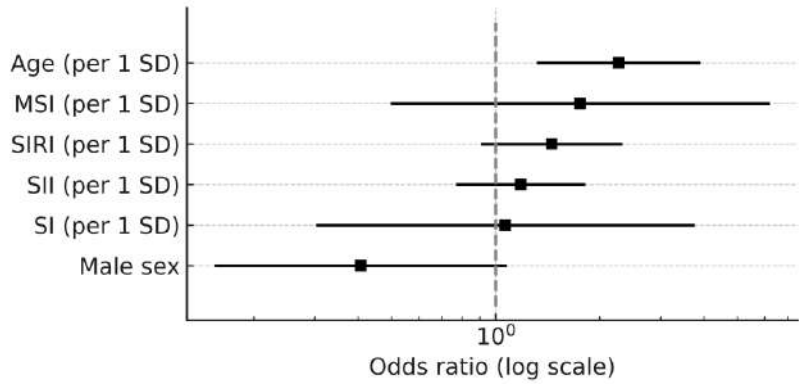

Figure 2. Adjusted odds ratios in the multivariate logistic model

The graph was created based on OR values and a 95% CI (confidence interval). The vertical dashed line indicates OR=1.

Patients were examined in three groups: survivors, early mortality (≤28 days), and late mortality (>28 days). To determine the relationship between mortality timing and clinical and laboratory profiles, inflammatory markers and shock indices (SII, SIRI, SI, MSI), D-dimer,

troponin, blood gas parameters (pH, pCO2, HCO3, lactate), biochemical indicators (albumin, CRP), vital signs including SpO2, were compared among these three groups created for the study (Tables 6-7).

SII and SIRI values were noticeably higher in the early and late mortality groups compared to survivors. A similar trend was observed in the MSI and SI indices (Table 6).

Table 6. Systemic inflammation and hemodynamic indices (SII, SIRI, SI, MSI) between groups

Variables	Grup	n	Median [IQR]	p (KW)
SIRI	Survivor	223	3.4 [2.0-6.8]	**<0.001**
	Early mortality	30	6.6 [4.5-12.8]	
	Late mortality	6	9.6 [5.6-11.6]	
SII	Survivor	223	1132.4 [658.5-2009.8]	**<0.05**
	Early mortality	30	1891.4 [1034.1-3899.9]	
	Late mortality	6	2196.4 [1293.7-5073.3]	
SI	Survivor	223	0.8 [0.6-0.9]	**<0.01**
	Early mortality	30	0.9 [0.8-1.2]	
	Late mortality	6	0.8 [0.6-1.1]	
MSI	Survivor	223	1.0 [0.9-1.2]	**<0.01**
	Early mortality	30	1.3 [1.1-1.6]	
	Late mortality	6	1.0 [0.9-1.4]	

SIRI: Systemic Inflammatory Response Index, SII: Systemic Immune-Inflammatory Index, SI: Shock Index, MSI: Modified Shock Index

The median D-dimer level was higher in early mortality; in late mortality (n=6), the median was lower. Although the median was lower in late mortality, the sample size was small (n=6, $p<0.01$). There was no significant difference in troponin levels between the early and late mortality groups compared to survivors. Acid-base balance and oxygenation parameters showed significant differences between the three groups. Lactate levels, in particular, were higher in the early mortality group. Although low albumin and high CRP values were frequently observed in the mortality groups, only albumin levels showed a remarkably difference between the two values. SpO2 levels were lower in the early mortality group (Table 7).

Table 7. Comparison of D-dimer, troponin, blood gas parameters (pH, pCO_2, HCO_3, lactate), biochemistry (albumin, CRP), and SpO_2 between groups (survivors, early mortality ≤28 days, late mortality >28 days)

Değişken	Grup	n	Median [IQR]	p (KW)
	Survivor	223	4.2 [2.1-8.0]	

D-dimer	Early mortality	30	7.4 [4.6-8.0]	**<0.01**
	Late mortality	6	2.7 [1.6-3.9]	
Troponin	Survivor	223	35.0 [9.0-158.1]	>0.05
	Early mortality	30	72.4 [22.4-174.8]	
	Late mortality	6	56.0 [22.3-128.3]	
pH	Survivor	223	7.4 [7.3-7.4]	**<0.05**
	Early mortality	30	7.4 [7.3-7.4]	
	Late mortality	6	7.3 [7.2-7.4]	
pCO2	Survivor	223	41.3 [37.0-46.8]	**<0.01**
	Early mortality	30	38.5 [35.3-48.2]	
	Late mortality	6	60.7 [46.8-71.5]	
HCO3	Survivor	223	25.1 [23.4-27.2]	**<0.01**
	Erken mortalite	30	21.7 [19.4-27.3]	
	Late mortality	6	28.9 [26.5-31.5]	
Lactate	Survivor	223	1.8 [1.2-2.5]	**<0.01**
	Early mortality	30	2.8 [1.7-4.3]	
	Late mortality	6	2.4 [1.2-5.8]	
Albumin	Survivor	223	36.0 [33.0-39.0]	**<0.01**
	Early mortality	30	34.0 [32.0-36.0]	
	Late mortality	6	27.5 [26.2-28.8]	
CRP	Survivor	223	31.0 [14.8-81.7]	>0.05
	Early mortality	30	47.2 [20.2-89.0]	
	Late mortality	6	102.0 [87.1-113.5]	
SpO2	Survivor	223	90.0 [85.0-95.0]	**<0.01**
	Early mortality	30	85.5 [81.2-88.8]	
	Late mortality	6	86.0 [81.5-89.8]	

CRP: C-reactive protein

Discussion

Studies in the literature regarding the relationship between PE and SII, SIRI, SI, and MSI are still very new and few in number. Future studies in this area may provide different perspectives on predicting mortality and prognosis in patients presenting to the emergency department. We also wanted to contribute to the literature by investigating the relationship between clinical course and prognosis and these scores in patients who presented to the emergency department with a diagnosis of PE.

Ösken A. et al. evaluated the SII index as an independent predictor of overall mortality in a study conducted in 2021 on 514 patients with a mean age of 61.5 years. Twenty-eight patients (5.4%) died within the first 30 days and 52 patients (10.1%) died within the following 29

months, resulting in an overall mortality rate of 15.6%. Age ($p<0.001$) and SII ($p=0.003$) were shown to be significantly associated with mortality (11). In our study, 223 (86%) of 259 (100%) patients survived, while 30 (12%) patients died within the first 28 days and 6 (2%) patients died after 28 days. When looking at our overall mortality rate, it is consistent with the literature, but the early mortality rate is higher than reported in the literature. This may be due to the higher mean age in our study (69.27±15.55 in surviving patients, 79.31±11.65 in deceased patients).

Duyan M. et al. investigated the relationship between multiple biomarkers and PE in 193 patients in 2022 and found the SII value to be remarkably higher in mortal patients ($p<0.05$). In the ROC curve analysis, SII was found to have "excellent" diagnostic power with a high AUC (0.84) value, and they evaluated it as a valuable and potentially useful biomarker for identifying high-risk PE patients (12). In our study, we also found that SII was significant in predicting mortality in the ROC analysis with an AUC value of 0.651, similar to the literature.

Another study in the literature examined 139 PE patients and found SII, lactate, and age to be independent risk factors for 30-day mortality ($p<0.05$) (13). In our study, SII and lactate were significant when comparing survivors in the early (≤28 days) and late mortality (>28 days) groups. These findings were also consistent with the literature ($p<0.05$ and $p<0.01$, respectively). In a study investigating venous thromboembolism (VTE), including DVT and PE, in patients after hip fracture, 346 elderly patients with hip fracture were studied, and SII was highlighted as a strong predictor of VTE development. Due to its easy accessibility and reasonable cost, it has been suggested that SII could play a leading role in the prevention, management, and treatment of VTE development after hip fracture in elderly patients (14). Based on the studies conducted, we see that SII and SIRI are important clinical and prognostic markers in PE.

Türkday Derebey S. et al. reported in their study on patients with PE that MSI showed a significant correlation with PESI, sPESI, and SI scores ($p<0.001$) and had the highest AUC value (0.739, $p=0.002$) in predicting patient mortality or hemodynamic deterioration. MSI was found to be superior to SI (15). In our study, ROC analysis showed that MSI (AUC=0.679) was slightly superior to SI (AUC=0.676). Another recent study showed that SII and SIRI were remarkably associated with high risk in acute PE in patients aged 18 years and older (16).

In a study investigating the relationship between SI and mortality based on 205 patients, 24.9% of patients died. In patients with fatal outcomes, the mean age and heart rate were higher, while the mean systolic and diastolic blood pressures were lower ($p<0.05$). The results of univariate and multivariate analyses showed that SI and age were independently and significantly associated with predicting in-hospital mortality ($p<0.05$) (17). In our study, age and gender were found to be significant in predicting mortality in univariate analysis ($p<0.001$

and p<0.001, respectively), while only age remained significant in multivariate analysis (p<0.01). When systolic and diastolic pressures were compared between the survivor and mortality groups, both were lower in the mortality group, but there was no statistically significant difference between the groups (p>0.05); however, SI and MSI were remarkably associated with mortality (p<0.001). We presume this situation stems from the use of heart rate along with blood pressure values in the calculation of SI and MSI. We believe that using shock indices instead of blood pressure alone is more effective when evaluating patients today.

Conclusion

In PE, clinical follow-up plays an important role in predicting prognosis and mortality using noninvasive markers. Studies on biomarkers such as SII, SIRI, SI, and MSI have increased in recent years, and the clinical use of these indices has also increased. Our study shows that the moderate AUC values of these four indices indicate that they are valuable in predicting mortality but have limited power as standalone predictors of mortality. In conclusion, we found that these indices, which we compared in terms of predicting mortality, are parameters that can enhance clinical evaluation rather than being decisive. Our findings reveal that systemic inflammation and hemodynamic status are important in the clinical course and prognosis of PE, but they need to be supported by multiparametric models.

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2510

Cardiac Tamponade Associated with Atypical Antipsychotics: A Case Series of Two Patients

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Introduction

Atypical antipsychotics are increasingly used in the treatment of various psychiatric disorders, particularly schizophrenia and bipolar disorder. Recent evidence has highlighted the clinical significance of the cardiovascular adverse effects of these drugs. Atypical antipsychotics have been associated with significant cardiac side effects, including arrhythmias, cardiomyopathy, myocarditis, and pericardial diseases (1,2).

In the literature, pericardial diseases associated with antipsychotics are most frequently associated with clozapine use (3–6). However, cases of pericardial effusion and cardiac tamponade associated with risperidone, paliperidone, and other atypical antipsychotics have also been reported, albeit rarely (7,9). In many of these cases, symptoms emerged during the early phase of treatment or following dose escalation and improved after discontinuation of the offending agent (5,8).

Clozapine-induced pericarditis may present with a clinical spectrum ranging from pleuritic chest pain to cardiac tamponade, with symptom resolution typically observed following drug discontinuation (3-5).

In this case report, we present two patients receiving atypical antipsychotics with different active compounds who developed cardiac tamponade on the same day of presentation to the emergency department. The aim is to highlight antipsychotic-associated pericardial complications and increase clinician awareness.

Case Report

Case 1

An 84-year-old woman presented to the emergency department with progressively worsening dyspnea over four days. Her medical history was significant for hypertension, diabetes mellitus, chronic kidney disease, and congestive heart failure. Home medications included nebivolol, apixaban, isosorbide mononitrate, furosemide, linagliptin, quetiapine, and sertraline. She was receiving long-term home oxygen therapy.

On presentation, the patient was conscious, oriented, and cooperative. Initial vital signs were as follows: blood pressure 130/70 mmHg, heart rate 51 beats/min, respiratory rate 18

breaths/min, body temperature 36.7°C, and oxygen saturation 99% while receiving 2–4 L/min supplemental oxygen via nasal cannula. Electrocardiography demonstrated sinus bradycardia with a heart rate of 51 beats/min, low-voltage QRS complexes, and nonspecific ST–T wave abnormalities. During emergency department follow-up, hypotension developed, with blood pressure decreasing to 80/50 mmHg.

Physical examination revealed muffled heart sounds, bilateral decreased basal breath sounds, jugular venous distension, and bilateral pretibial edema (3+). No additional abnormal findings were noted. Medication history revealed that the patient's quetiapine dose had been increased from 50 mg/day to 200 mg/day approximately two months earlier, while sertraline 50 mg/day had been continued.

Point-of-care ultrasonography revealed a large circumferential pericardial effusion measuring approximately 3 cm adjacent to the right ventricle, accompanied by late diastolic right atrial collapse, early diastolic right ventricular collapse, abnormal interventricular septal motion, and a plethoric inferior vena cava with reduced respiratory variation, consistent with cardiac tamponade physiology.

Based on clinical and ultrasonographic findings, cardiac tamponade was diagnosed. Emergent pericardiocentesis was performed following hemodynamic deterioration and development of pulmonary edema.

Case 2

A 76-year-old woman was admitted to the emergency department after a syncopal episode. Her medical history included hypertension, diabetes mellitus, congestive heart failure, and schizophrenia. Current medications included torasemide, quetiapine, risperidone, olanzapine, amlodipine, metformin, and atorvastatin.

On admission, the patient was confused, disoriented, and uncooperative. Vital signs were as follows: blood pressure 150/90 mmHg, heart rate 77 beats/min, respiratory rate 14 breaths/min, body temperature 36.4°C, and oxygen saturation 92% on room air. Electrocardiography demonstrated sinus rhythm with occasional ventricular premature complexes.

Neurological examination was limited because of the patient's mental status; however, no focal neurological deficits were identified. The remainder of the physical examination was unremarkable.

Medication review revealed that the patient had been receiving olanzapine 15 mg/day, quetiapine 50 mg/day, and risperidone 1 mg/day for schizophrenia, with the risperidone dose increased to 5 mg/day eight days prior to presentation.

Point-of-care ultrasonography demonstrated a large pericardial effusion measuring approximately 3.5 cm adjacent to the right atrium, with associated echocardiographic findings consistent with cardiac tamponade physiology.

During emergency department follow-up, the patient developed hypotension (blood pressure 90/60 mmHg) accompanied by tachycardia, raising suspicion for cardiac tamponade. The patient was subsequently admitted to the cardiology department for urgent pericardiocentesis.

Discussion

Although QT prolongation, myocarditis, and pericardial diseases are recognized cardiac adverse effects of atypical antipsychotics, pericardial effusion and cardiac tamponade remain rare. Most reported cases are associated with clozapine and are thought to involve immune-mediated mechanisms (4–6). However, similar complications have also been reported with other agents such as risperidone and paliperidone (7,9). The occurrence of cardiac tamponade in two patients receiving different antipsychotic agents suggests that this class of drugs may pose a shared risk for severe cardiac complications.

Proposed mechanisms include hypersensitivity reactions, immune-mediated inflammation, and direct cardiotoxicity (4–6). The temporal association—particularly early onset after treatment initiation or dose escalation—supports a drug-related etiology (5,7).

Cardiac tamponade may present with nonspecific symptoms, and in psychiatric patients, somatic complaints may be overlooked. Therefore, pericardial effusion and tamponade should be considered in patients receiving antipsychotics who present with unexplained dyspnea, chest pain, tachycardia, or hypotension (10,11). Bedside echocardiography plays a crucial role in rapid diagnosis and timely management (11).

The presence of tamponade in both cases highlights the potentially rapid progression and fatal nature of this condition. Consistent with previous reports, clinical and echocardiographic improvement following discontinuation of the antipsychotic agent supports a causal relationship (5,8,9).

Conclusion

These cases demonstrate that pericardial effusion associated with antipsychotic use may progress to cardiac tamponade, a potentially fatal complication.

This report contributes to the limited literature on antipsychotic-associated cardiac tamponade and underscores the need for increased clinical awareness. Early recognition, multidisciplinary management, and prompt intervention are essential to reduce morbidity and mortality.

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Psoas Abscess: A Case Report of a Patient Presenting with Fever and Hip Pain

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Abstract

A psoas abscess is a rare infection that may lead to significant morbidity and mortality if diagnosis is delayed. Clinical findings are often nonspecific and may present as fever, low back pain, flank pain, groin pain, limited hip movement, and difficulty in walking. Therefore, diagnosis may be delayed, particularly in the emergency department. Current management

recommends the combination of broad-spectrum antibiotic therapy with image-guided percutaneous drainage or, in selected cases, surgical drainage.

In this case report, we present a patient who was admitted with fever, right flank pain, and difficulty in walking, diagnosed with a psoas abscess by computed tomography, and successfully treated with percutaneous drainage.

Keywords: psoas abscess, iliopsoas abscess, emergency department, percutaneous drainage, case report

Introduction

A psoas abscess is defined as a purulent infection developing within the psoas or iliopsoas muscle compartment. The primary form usually occurs as a result of hematogenous spread, whereas the secondary form arises from infections in adjacent structures. Due to its nonspecific clinical presentation, patients are often initially evaluated under differential diagnoses such as musculoskeletal disorders, renal colic, vertebral infections, or intra-abdominal infections. Although the most commonly reported symptoms are fever, low back/flank pain, and hip pain, the classic triad is not present in all patients. Computed tomography is one of the main imaging modalities for diagnosis. The mainstay of treatment is drainage in combination with antibiotic therapy (1).

Case Report

A 44-year-old male patient presented to the emergency department with a three-day history of fever, malaise, right flank pain, and pain radiating to the right hip. His medical history included type 2 diabetes mellitus and hypertension. He reported difficulty walking due to increasing pain over the past week, with a marked increase in pain especially when the right hip was extended. There was no history of trauma. He reported mild dysuria; however, there were no significant gastrointestinal complaints.

At admission, vital signs were as follows: temperature 37.6°C, blood pressure 95/60 mmHg, heart rate 116 beats/min, respiratory rate 20 breaths/min, and oxygen saturation 97%. Physical examination revealed tenderness in the right flank and right lower abdominal quadrant. Pain increased with passive extension of the right hip, and examination findings suggestive of psoas irritation were noted. No neurological deficit was detected.

Laboratory investigations revealed a white blood cell count of 27,000/μL, neutrophil percentage of 86%, C-reactive protein of 196 mg/L, erythrocyte sedimentation rate of 90 mm/hour, and procalcitonin of 2.9 ng/mL. Serum creatinine was 1.1 mg/dL. Urinalysis showed leukocyturia. Blood and urine culture samples were obtained.

Initially, differential diagnoses included urinary tract infection, pyelonephritis, and retroperitoneal infection. Since abdominal ultrasonography provided limited evaluation, contrast-enhanced abdominal computed tomography was performed. Imaging revealed a hypodense collection measuring approximately 4 × 4 cm within the right psoas muscle, showing peripheral contrast enhancement, consistent with an abscess. Inflammatory changes were also observed in the right perinephric fat tissue. These findings were consistent with a psoas abscess.

Following consultations with infectious diseases and interventional radiology, the patient was hospitalized from the emergency department. Empiric intravenous piperacillin–tazobactam and vancomycin therapy was initiated. On the same day, CT-guided percutaneous catheter drainage was performed, and approximately 40 mL of purulent material was evacuated. Culture of the drainage material grew *Staphylococcus aureus*, while blood cultures remained sterile. Antibiotic therapy was adjusted according to culture and sensitivity results. During follow-up, fever subsided, inflammatory markers decreased, and hip pain significantly improved. Control imaging demonstrated a marked reduction in the collection. The patient was discharged after 14 days of intravenous therapy with a recommendation for oral antibiotics. At the one-month outpatient follow-up, he was asymptomatic and laboratory values had returned to normal.

Discussion

Although a psoas abscess is a rare condition, it represents an important diagnostic pitfall in patients presenting with nonspecific symptoms. While patients may present with fever, low back pain, and pain on hip movement, the classic clinical triad is not always present; therefore, diagnosis may be delayed. In particular, comorbidities such as diabetes mellitus that may cause immunosuppression can facilitate the development of infection (2).

Psoas abscesses are classified as primary and secondary. Primary cases occur via hematogenous spread, whereas secondary cases may develop due to spread from vertebral osteomyelitis, Crohn's disease, urinary tract infections, appendicitis, or other adjacent infectious foci (3). In our case, concomitant leukocyturia and perinephric inflammatory changes suggested possible secondary spread; however, a definitive primary source could not be identified.

Imaging plays a critical role in diagnosis. Although ultrasonography may be helpful in some cases, its sensitivity may be limited due to the retroperitoneal location of the lesion (4). Computed tomography is highly valuable in demonstrating the localization, size, and relationship of the abscess with adjacent structures, and it also facilitates planning for percutaneous drainage (3,4).

The treatment approach in most patients includes antibiotic therapy combined with drainage. Current literature reports that percutaneous drainage is a minimally invasive and effective option; however, surgical drainage may be required in cases of multiloculated collections, failed percutaneous intervention, or underlying surgical pathology (5). In our case, successful outcomes were achieved with early CT-guided percutaneous drainage and appropriate antibiotic therapy.

This case highlights that psoas abscess should always be considered in the differential diagnosis of patients presenting to the emergency department with fever accompanied by low back, flank, or hip pain—especially when pain increases with hip extension and inflammatory markers are elevated.

Conclusion

Psoas abscess is a rare but clinically significant infection. Because it presents with nonspecific symptoms, diagnosis may be delayed. It should be considered in patients with fever, low back/flank pain, hip pain aggravated by movement, and elevated inflammatory markers. Early imaging, appropriate antibiotic therapy, and timely drainage can lead to successful clinical outcomes.

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Ruptured Ectopic Pregnancy Presenting with Pelvic Free Fluid in a Hemodynamically Stable Patient: A Case Mimicking Appendicitis

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Introduction

Ectopic pregnancy is defined as implantation of the fertilized ovum outside the uterine cavity and accounts for approximately 1–2% of all pregnancies. It is one of the major causes of maternal mortality in early pregnancy (1). Approximately 95% of cases are located in the fallopian tubes (2). When rupture occurs, intra-abdominal bleeding and hemoperitoneum may develop.

The classic clinical triad includes lower abdominal pain, vaginal bleeding, and amenorrhea; however, hemodynamic parameters may remain normal in the early stages (3). This may lead to clinical misinterpretation and diagnostic delay.

For instance, the absence of an intrauterine gestational sac on ultrasonography despite β-hCG levels above the discriminatory threshold (1500–2000 mIU/mL) raises strong suspicion for ectopic pregnancy (4). The presence of pelvic free fluid, especially when associated with hemoperitoneum, is a strong indicator of rupture (5).

In this study, we present a case of ectopic pregnancy that appeared hemodynamically stable but demonstrated laboratory and imaging findings suggestive of rupture.

Case

A 28 year old female presented to the emergency department with right lower quadrant-dominant lower abdominal pain, nausea, and vomiting that started 12 hours prior. Her vital signs were stable (blood pressure and heart rate within normal limits).

On physical examination, tenderness and guarding were noted in the right lower quadrant and suprapubic region. Her medical history included Factor V Leiden mutation and a previous ectopic pregnancy. She reported having menstrual bleeding eight days prior.

Laboratory investigations revealed β-hCG of 1885 mIU/mL and CRP of 25 mg/L. Arterial blood gas analysis showed pH 7.30, HCO_3 19.9 mmol/L, and lactate 3.2 mmol/L. Elevated lactate may indicate early tissue hypoperfusion (6).

Transvaginal ultrasonography showed no intrauterine gestational sac and revealed free fluid in the pelvic region. Considering appendicitis in the differential diagnosis, contrast-enhanced abdominal computed tomography was performed.

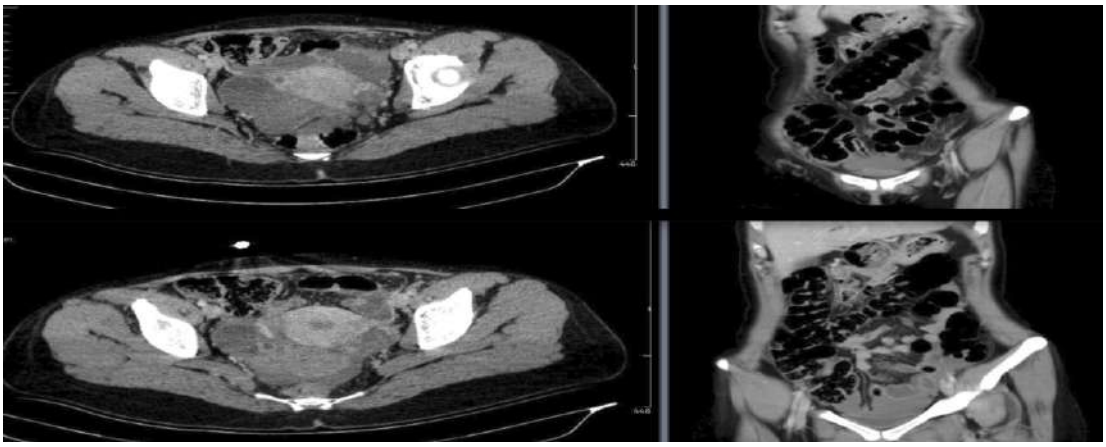

Figure 1. CT imaging demonstrated pelvic free fluid and findings consistent with tubo-ovarian pathology around the right ovary, while no inflammatory changes were observed around the appendix.

Based on the combined clinical, laboratory, and imaging findings, a preliminary diagnosis of ruptured ectopic pregnancy was made. The patient was informed about the risk of intra-abdominal bleeding and the need for emergency surgery. However, she refused treatment and left the hospital after signing an informed refusal form, intending to seek fertility-preserving management at another center.

Discussion

Early diagnosis of ectopic pregnancy is critical to minimize mortality (1). Hemodynamic stability does not exclude rupture, as bleeding may initially be masked by compensatory mechanisms (3).

The presence of pelvic free fluid, particularly in a β-hCG-positive patient without evidence of intrauterine pregnancy, is a strong indicator of rupture (5). Elevated lactate levels may serve as an early marker of systemic hypoperfusion and should raise suspicion for occult bleeding despite apparent clinical stability (6).

Appendicitis is another condition that may cause lower quadrant pain; however, gynecological pathologies must always be considered in women of reproductive age (7). In this case, CT imaging played a role in excluding appendicitis.

Women with a history of ectopic pregnancy have a higher risk of recurrence (2). Therefore, a high index of clinical suspicion should be maintained in patients with risk factors.

Conclusion

Ectopic pregnancy must be excluded in women of reproductive age presenting with lower abdominal pain. Stable vital signs do not eliminate the risk of rupture. β-hCG levels, absence of an intrauterine gestational sac, and presence of pelvic free fluid should be evaluated together. Elevated lactate may indicate early hypoperfusion. Clinical and radiological correlation is essential for timely intervention.

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Keywords
Ectopic pregnancy, Rupture, Pelvic free fluid, Lactate, Differential diagnosis of appendicitis

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Evaluation of the demographic characteristics and laboratory parameters of dialysis patients

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ABSTRACT

Aim:

Patients with chronic kidney disease (CKD) receiving dialysis treatment frequently present to emergency departments due to various clinical conditions. This study aimed to evaluate the demographic characteristics and laboratory parameters of dialysis patients presenting to the emergency department and to investigate potential differences according to sex.

Materials and Methods:
This retrospective study included patients with chronic kidney disease who presented to the emergency department between February 15, 2026, and March 15, 2026, and had received dialysis treatment at least once. During the study period, 31 patients were identified, and 23 patients who met the inclusion criteria were analyzed. Demographic data and laboratory parameters were obtained from the hospital's electronic medical record system. The normality of data distribution was assessed using the Shapiro–Wilk test. Variables with normal distribution were expressed as mean ± standard deviation, whereas non-normally distributed variables were presented as median (interquartile range, IQR). Comparisons according to sex were performed using the Independent Samples t-test or the Mann–Whitney U test.

Results:
Of the 23 patients included in the study, 11 (47.8%) were male and 12 (52.2%) were female. Albumin levels were found to be significantly higher in male patients compared to female patients (3.59 ± 0.43 g/dL vs. 3.04 ± 0.64 g/dL, $p = 0.026$). Hemoglobin levels were also higher in male patients ($p = 0.023$). In contrast, no statistically significant differences were observed between sexes in terms of age, CRP, neutrophil, amylase, lymphocyte, platelet, monocyte, creatinine, urea, potassium, and sodium levels ($p > 0.05$).

Conclusion:
It was demonstrated that albumin and hemoglobin levels were higher in male patients among dialysis patients presenting to the emergency department. Although albumin reference ranges in healthy individuals are generally considered to be independent of sex, it is suggested that higher albumin levels may be observed in male patients with chronic kidney disease

undergoing dialysis. These findings need to be supported by studies conducted with larger patient cohorts.

Keywords:
Chronic kidney disease, dialysis, emergency department, albumin, hemoglobin, sex differences

Introduction:

Chronic kidney disease (CKD) is characterized by the progressive and irreversible loss of kidney function and is considered a major public health problem worldwide [1]. In the advanced stages of CKD, patients frequently progress to end-stage renal disease and require renal replacement therapies, particularly hemodialysis or peritoneal dialysis, to sustain life [2].

Patients receiving dialysis treatment are at high risk for numerous clinical conditions, including fluid and electrolyte imbalances, metabolic disturbances, cardiovascular complications, infections, and vascular access problems [3]. Therefore, emergency department visits among this patient group are more frequent compared to the general population and constitute a significant healthcare burden [4].

Although hyperkalemia, fluid overload, infections, and vascular access complications are commonly reported among the reasons for emergency department visits in dialysis patients in the literature, it is also known that these patients may present to the emergency department not only due to complications related to kidney disease but also due to other acute medical conditions [5]. Therefore, the evaluation of emergency department visits among dialysis patients is important not only for identifying complications related to kidney disease but also for revealing other accompanying acute clinical conditions.

In this study, it was aimed to evaluate the demographic characteristics and clinical reasons for presentation of patients diagnosed with chronic kidney disease who were receiving dialysis treatment and who presented to the emergency department for any reason within the past month.

Materials and Methods:

This study was conducted to retrospectively evaluate patients with chronic kidney disease (CKD) who presented to the emergency department of Malatya Training and Research Hospital between February 15, 2026, and March 15, 2026. Within the specified date range, patients with CKD who presented to the emergency department with any complaint and had received dialysis treatment at least once were identified through the hospital record system.

A total of 31 CKD patients were identified during the study period. Patients with chronic kidney disease who had presented to the emergency department and had previously received dialysis treatment at least once were included in the study. Patients with incomplete clinical or

laboratory data, as well as those whose study data could not be accessed, were excluded. Based on these criteria, 23 patients were included in the study.

Demographic characteristics of the patients (age, sex), along with laboratory parameters [C-reactive protein (CRP), albumin, neutrophil, amylase, lymphocyte, platelet, hemoglobin, monocyte, creatinine, urea, potassium, and sodium], were obtained from the hospital's electronic medical record system.

Statistical Analysis:
The normality of data distribution was assessed using the Shapiro–Wilk test. Variables with normal distribution were presented as mean ± standard deviation, whereas non-normally distributed variables were expressed as median (interquartile range, IQR). For comparisons between groups according to sex, the Independent Samples t-test was used for normally distributed variables, while the Mann–Whitney U test was applied for variables that did not show a normal distribution. Statistical significance was accepted as $p < 0.05$.

Results:
A total of 23 patients who presented to the emergency department within the past month and were receiving dialysis treatment due to chronic kidney disease were included in the study. Of these patients, 11 (47.8%) were male and 12 (52.2%) were female. The normality of data distribution was assessed using the Shapiro–Wilk test. Age (years) ($p = 0.041$), CRP (mg/L) ($p < 0.001$), hemoglobin (g/dL) ($p = 0.002$), and creatinine (mg/dL) ($p = 0.001$) were found not to follow a normal distribution, whereas albumin (g/dL) ($p = 0.197$), neutrophil ($\times 10^3/\mu L$) ($p = 0.369$), amylase (U/L) ($p = 0.417$), lymphocyte ($\times 10^3/\mu L$) ($p = 0.322$), platelet ($\times 10^3/\mu L$) ($p = 0.290$), monocyte ($\times 10^3/\mu L$) ($p = 0.252$), urea (mg/dL) ($p = 0.469$), potassium (mmol/L) ($p = 0.611$), and sodium (mmol/L) ($p = 0.428$) were determined to be normally distributed.

Variables with normal distribution were presented as mean ± standard deviation, whereas non-normally distributed variables were expressed as median and interquartile range (IQR). Descriptive statistics are presented in Table 1.

Table 1. Demographic and Laboratory Characteristics of the Patients

Variables	n	p value	Mean	SD	Median	IQR	
						25%	75%
Age (years)	23	0.041			58	55	79
CRP (mg/L)		<0.001			8,1	4,4	28
Albumin (g/dL)		0.197	3,31	0,6			
Neutrophil		0.369	5,35	1,7			

(×10³/µL)						
Amylase (U/L)	0.417	76,6	42,8			
Lymphocyte (×10³/µL)	0.322	1,99	0,79			
Platelet (×10³/µL)	0.290	239	63,1			
Hemoglobin (g/dL)	0.002			10,1	8,8	12,1
Monocyte (×10³/µL)	0.252	0,66	0,16			
Creatinine (mg/dL)	0.001			4	1,7	5,4
Urea (mg/dL)	0.469	98,2	36,6			
Potassium (mmol/L)	0.611	4,1	0,78			
Sodium (mmol/L)	0.428	136	3			

In the sex-based analysis, albumin levels were found to differ significantly between male and female patients, with higher albumin levels observed in males compared to females (3.59 ± 0.43 g/dL vs. 3.04 ± 0.64 g/dL, $p = 0.026$). In addition, hemoglobin levels were also higher in male patients than in female patients, showing a statistically significant difference between sexes ($p = 0.023$).

In contrast, no statistically significant differences were observed between male and female patients in terms of age, CRP, neutrophil, amylase, lymphocyte, platelet, monocyte, creatinine, urea, potassium, and sodium levels ($p > 0.05$).

Discussion:

In this study, laboratory parameters of chronic kidney disease patients receiving dialysis who presented to the emergency department were compared according to sex. Our findings demonstrated that albumin and hemoglobin levels were significantly higher in male patients compared to female patients, whereas no significant differences were observed between sexes for other laboratory parameters.

Serum albumin levels in chronic kidney disease and dialysis patients are considered an important indicator of nutritional status and inflammation, and low albumin levels have been shown in numerous studies to be associated with increased mortality [1,3]. Although reference ranges for albumin in healthy individuals are generally considered similar for both men and women, some studies have reported that albumin levels may be higher in male dialysis

patients. This difference is thought to be related to variations in muscle mass, protein metabolism, and hormonal differences between sexes. Greater muscle mass in males and the anabolic effects of androgens on protein synthesis may contribute to relatively higher hepatic albumin production. Additionally, protein-energy malnutrition and chronic inflammation, which are common in dialysis patients, may have a greater impact on albumin levels in female patients.

In our study, hemoglobin levels were also found to be higher in male patients. This finding is consistent with the literature. Although anemia is common in chronic kidney disease, hemoglobin levels are generally higher in men compared to women. This is primarily attributed to the erythropoiesis-stimulating effects of androgens and physiologically lower baseline hemoglobin levels in women.

In contrast, no significant differences were observed between male and female patients regarding CRP, neutrophil, amylase, lymphocyte, platelet, monocyte, creatinine, urea, potassium, and sodium levels. In dialysis patients, these biochemical parameters are often influenced more by acute clinical conditions, inflammation, and dialysis adequacy rather than by sex [3–5].

In conclusion, our study demonstrates that although albumin levels in dialysis patients are generally interpreted within fixed reference ranges similar to healthy individuals, they may be higher in male patients. This suggests that sex-specific physiological differences should be considered when interpreting albumin values in patients with chronic kidney disease.

Limitations:

This study has several limitations. First, it was conducted as a single-center study with a limited number of patients (n = 23). Additionally, the study had a retrospective design, and data were obtained from the hospital record system. Furthermore, only dialysis patients who presented to the emergency department within the past month were evaluated, and factors that could influence albumin levels, such as nutritional status or comorbid conditions, were not analyzed in detail. Therefore, the findings need to be supported by studies involving larger patient cohorts.

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Invasive Rhino-Orbital-Cerebral Mucormycosis Presenting with Isolated Ocular Findings in an Immunocompetent Patient: A Case Report

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Abstract
Mucormycosis is an angioinvasive fungal infection characterized by tissue necrosis and high mortality, typically affecting immunocompromised individuals. It is classically observed in patients with diabetes mellitus or other immunosuppressive conditions and commonly presents with nasal or palatal necrotic eschar. Herein, we report a case of rhinocerebral mucormycosis in a 69-year-old immunocompetent patient with no known comorbidities or immunosuppressive history, who presented with isolated progressive ocular symptoms, including ptosis, vision loss, and ophthalmoplegia, in the absence of overt nasal necrosis. The lack of classical clinical features and the patient's immunocompetent status posed significant challenges for early diagnosis.

Introduction
Mucormycosis is a rapidly progressive and life-threatening opportunistic fungal infection caused by organisms of the order *Mucorales* (e.g., *Rhizopus*, *Mucor*).[1,2] In clinical practice, it is most frequently encountered in high-risk populations, including patients with uncontrolled diabetes mellitus, diabetic ketoacidosis, hematologic malignancies, renal failure, or those receiving prolonged immunosuppressive therapy.[1]
Rhinocerebral mucormycosis (RCM), the most common clinical form, typically presents with symptoms resembling sinusitis, such as headache, periorbital edema, and fever, along with the hallmark finding of black necrotic eschar on the nasal or palatal mucosa. [1]However, rare cases have been reported in immunocompetent individuals, underscoring the aggressive nature of the disease. Nonspecific symptoms and atypical patient profiles may delay diagnosis, directly contributing to increased mortality.[1–3]

Case Presentation

A 69-year-old male with no known chronic medical conditions and no history of regular medication use presented to the emergency department with complaints of left-sided ptosis and vision loss. His history revealed a one-week prodrome of fever, malaise, headache, facial fullness, and nasal congestion prior to presentation.

On initial evaluation, vital signs were stable, and the patient was conscious, cooperative, and oriented. Neurological examination revealed no neck stiffness, and motor strength was intact in all extremities; however, the gag reflex was diminished. Ophthalmological examination demonstrated marked ptosis of the left eye. The left eye was deviated laterally in the primary position and appeared mydriatic, with restricted upward and medial gaze. Direct light reflex was absent in the left eye, while the right eye examination was unremarkable. Examination of the oral cavity, oropharyngeal mucosa, and both the hard and soft palate revealed no necrotic lesions or eschar typically associated with mucormycosis.

Laboratory investigations showed markedly elevated C-reactive protein (CRP) levels and neutrophil-predominant leukocytosis. Paranasal sinus computed tomography (CT) demonstrated significant loss of aeration in the bilateral anterior and posterior ethmoid air cells as well as the sphenoid sinuses. Cranial magnetic resonance imaging (MRI) performed at an external center revealed a contrast-enhancing lesion filling the nasal cavity adjacent to the anterior cranial fossa at the level of the ethmoid roof, along with a millimetric diffusion-restricted focus in the anterior portion of the right temporal lobe. Further cranio-orbital MRI showed prominent contrast enhancement in the left retrobulbar prenodal soft tissue, dural enhancement at the skull base bilaterally, and a diffusion-restricted lesion in the right temporal lobe suggestive of cerebritis or abscess formation. Diffusion-weighted imaging also demonstrated an acute infarction in the watershed zone between the anterior cerebral artery (ACA) and middle cerebral artery (MCA) territories. Cerebrospinal fluid analysis obtained via lumbar puncture showed no evidence of meningitis. (Figure 1)

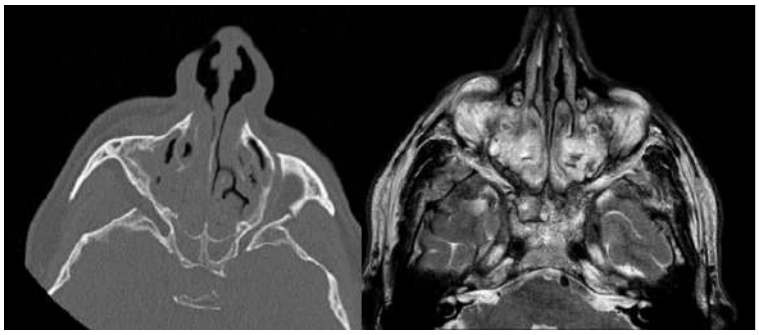

Figure 1. Paranasal sinus CT demonstrating loss of aeration in the paranasal sinuses and thinning of the bony structures of the sphenoid sinus (1). Contrast-enhanced brain MRI (T1-weighted) showing a hypointense area in the right temporal lobe suggestive of a purulent collection (2).

The patient was managed with a multidisciplinary approach involving neurology, infectious diseases, ophthalmology, and otorhinolaryngology teams. During follow-up, the development of vision loss in the contralateral (right) eye prompted urgent surgical exploration and debridement by the otorhinolaryngology team. Intraoperatively, the sphenoid sinus and adjacent structures appeared extensively necrotic, and radical debridement was performed. Histopathological examination of the debrided tissue confirmed invasive mucormycosis with characteristic *Mucorales* hyphae. (Figure 2)
Based on infectious diseases consultation, systemic liposomal amphotericin B was promptly added to the existing antimicrobial therapy. Despite aggressive management, the patient's condition deteriorated due to rapid intracranial progression. He required intubation and intensive care support but ultimately succumbed to the disease.

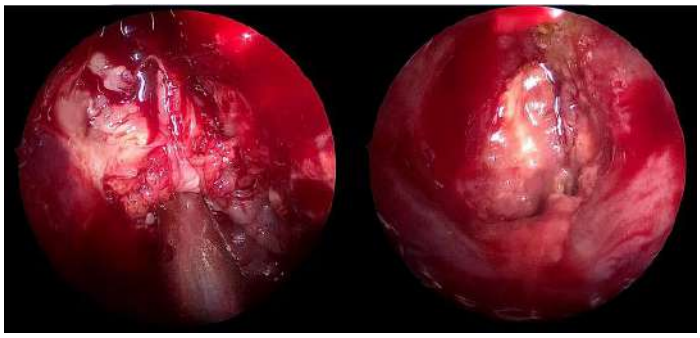

Figure 2 Intraoperative view showing inflamed ethmoid cells and necrotic mucosa of the sphenoid sinus.

Discussion

Mucormycosis is typically encountered in clinical conditions associated with impaired immunity, such as diabetes mellitus, diabetic ketoacidosis, and hematological malignancies.[1,4] Two main aspects make our case exceptionally rare and noteworthy in the medical literature: (1) the absence of any identifiable comorbidity or history of immunosuppression, and (2) the presentation of the disease with completely isolated progressive ocular findings, rather than the classical necrotic eschar of the palate and nasal cavity.

Following the inhalation of spores, the infection usually begins in the nasal turbinates and paranasal sinuses; immunocompetent individuals generally eliminate these spores rapidly.[1,2] However, in this case, despite an immunocompetent status, the disease exhibited an angioinvasive character and followed a fulminant course, demonstrating that progressive fungal dissemination may rarely occur even in healthy hosts. Similarly, previous studies have discussed that in orbital mucormycosis cases observed in immunocompetent patients, isolates such as *Apophysomyces elegans* may lead to an aggressive clinical course, or that transient, patient-specific defects in cellular immunity may predispose to this condition.[2,4,5]

From a diagnostic perspective, the greatest challenge was that the examination of the oral cavity and palate was completely normal at the initial presentation. [1]The most important major physical examination finding that raises suspicion for mucormycosis is palatal necrosis. The absence of this finding may often lead clinicians toward alternative diagnoses such as bacterial sinusitis or neurological vascular syndromes. The clinical presentation in our case (ptosis, pupillary abnormality, extraocular muscle involvement, and vision loss) clearly represents orbital apex syndrome, indicating the spread of infection from the sinuses to the retrobulbar space and orbital apex via the lamina papyracea or inferior orbital fissure.[1,2,4,6]

Due to the pathogen's high vascular tropism, angioinvasion affected the carotid artery and its branches, leading to vascular occlusions (acute infarction) and severe intracranial sequelae such as focal cerebritis or abscess formation. Indeed, the acute ACA–MCA watershed infarction and the temporal lobe lesion observed on diffusion-weighted MRI represent the radiological manifestations of this vascular occlusion. Although the classical treatment triad—correction of the underlying condition (which could not be identified in this case), aggressive surgical debridement, and systemic liposomal amphotericin B—was implemented, the extent of intracranial involvement and the spread toward the cavernous sinus via the sphenoid structures resulted in high mortality.[2,3,7,8]

Conclusion

Rhinocerebral mucormycosis should not be considered solely within the immunosuppressed or diabetic patient population. In otherwise healthy and immunocompetent patients, particularly in the presence of treatment-resistant and rapidly progressive isolated orbital or neurological findings such as ptosis, ophthalmoplegia, and vision loss, invasive fungal rhinosinusitis should be included among the primary differential diagnoses, even in the absence of classical findings such as palatal necrosis.[4] The fulminant nature of the disease demonstrates that even minimal clinical delay can lead to dramatic consequences, and that radiological and surgical evaluation must be performed urgently

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Atropine-Induced Delirium in Organophosphate Poisoning

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INTRODUCTION

Delirium is an acute neuropsychiatric syndrome marked by sudden disturbances in consciousness, attention, and cognition. In the emergency department (ED), infections, metabolic disorders, and medications are common causes (1). Drugs with anticholinergic properties, such as atropine, carry a high risk of central nervous system effects (2,3). Atropine is essential in organophosphate poisoning treatment but may rarely cause central anticholinergic toxicity and delirium, which can mimic worsening poisoning and complicate management (4). This case highlights atropine-induced delirium, its differential diagnosis, and the importance of recognizing central adverse effects during therapy.

CASE PRESENTATION

A 48-year-old male with suspected organophosphate exposure received atropine at 1 mg every 5 minutes (cumulative dose 50 mg) at an outside ED. Approximately 1.5 hours later, he developed acute agitation and altered mental status and was transferred to our ED. On admission, he was confused, GCS 12, vitals stable. Examination revealed chemical burns, warm dry skin, and mydriasis. Laboratory results, including ABG, CRP, WBC, pseudocholinesterase, and troponin, were unremarkable aside from neutrophilia. Other causes of delirium—hypoxia, sepsis, metabolic disturbances, ongoing organophosphate toxicity—were excluded. The temporal relationship with atropine administration and anticholinergic signs supported the diagnosis of atropine-induced delirium. The patient was admitted to the anesthesia ICU for close monitoring and supportive care.

DISCUSSION

Anticholinergic delirium results from central muscarinic receptor blockade, presenting with agitation, confusion, mydriasis, and fluctuating consciousness (1,3). While atropine is lifesaving in organophosphate poisoning, high cumulative doses may trigger central toxicity (4). Rapid onset of delirium after atropine, coupled with exclusion of alternative causes, confirms the diagnosis (1,3). Unrecognized atropine-induced delirium may be mistaken for poisoning progression, potentially leading to harmful dose escalation. Management focuses on supportive care, dose reassessment, and, if necessary, short-term sedatives. Physostigmine may be considered in selected cases but carries risks (5).

Atropine-induced delirium should be considered in any patient with altered mental status during organophosphate poisoning treatment, even at therapeutic doses, to avoid mismanagement and ensure appropriate supportive care(4).

KEYWORDS

Anticholinergic toxicity, Atropine, Delirium, Organophosphate poisoning

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Comparison of Lipid Profiles in Diabetic and Non-Diabetic Patients

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Objective: This study aimed to compare lipid profile parameters between diabetic and non-diabetic patients and to evaluate the effects of diabetes on lipid metabolism.

Materials and Methods: This is a retrospective and descriptive study including a total of 31 patients from an existing dataset. The patients were divided into two groups based on the presence of diabetes mellitus: diabetic and non-diabetic patients. Laboratory analyses evaluated total cholesterol, LDL-C, HDL-C, and triglyceride levels, while TG/HDL and LDL/HDL ratios were calculated for a more detailed assessment of atherogenic risk.

Results: No statistically significant differences were observed between diabetic and non-diabetic patients regarding total cholesterol ($p = 0.184$), LDL cholesterol ($p = 0.212$), HDL cholesterol ($p = 0.098$), and triglyceride ($p = 0.071$) levels. However, upon evaluating atherogenic indices, the TG/HDL ratio was found to be significantly higher in diabetic patients ($p = 0.041$). The LDL/HDL ratio did not show a statistically significant difference between the groups ($p = 0.089$).

Conclusion: Although statistical significance was not achieved in the majority of classical lipid parameters, atherogenic changes are observed in the lipid profiles of diabetic patients. In particular, the significantly higher TG/HDL ratio highlights the importance of this index as a key indicator of diabetic dyslipidemia and increased cardiovascular risk.

INTRODUCTION

Diabetes mellitus (DM) is a common endocrine disease characterized by chronic hyperglycemia, leading to numerous metabolic and vascular complications. Today, the rapid increase in the prevalence of diabetes has made it important to better understand the systemic effects of the disease and to identify complications at an early stage. Cardiovascular diseases are one of the most important causes of morbidity and mortality in diabetes, and this condition is largely associated with accompanying metabolic disorders (1).

Dyslipidemia, which is frequently seen in diabetic patients, occurs as a result of insulin resistance and impaired lipid metabolism. This condition is generally characterized by high

triglyceride levels, low HDL cholesterol, and normal or slightly increased LDL cholesterol, and it is defined as "diabetic dyslipidemia". Furthermore, LDL particles becoming smaller and denser contribute to an increase in atherogenic potential (2,3). These changes in the lipid profile play a fundamental role in the development of atherosclerosis and significantly increase the risk of cardiovascular events in diabetic patients. Therefore, in addition to classical lipid parameters such as total cholesterol, LDL, HDL, and triglycerides, atherogenic indices such as the TG/HDL and LDL/HDL ratios have also been considered important predictors in recent years (3,4).

Although it has been shown in the literature that the lipid profile is impaired in diabetic patients and this situation is closely related to cardiovascular risk, these parameters need to be compared across different populations to demonstrate their clinical significance (5). In this study, we aimed to compare lipid profile parameters in diabetic and non-diabetic patients and to evaluate the effects of diabetes on lipid metabolism.

Materials and Methods

This study was planned as a retrospective and descriptive study. A total of 31 patients present in the available dataset were included in the study. The patients were divided into two groups based on the presence of diabetes mellitus: diabetic patients and non-diabetic patients. Diabetes status was determined according to the diagnoses found in the patients' medical histories. The patients' age, gender, height, weight, body mass index, and accompanying diseases were recorded.

In the laboratory examination, parameters pertaining to the lipid profile were evaluated. Within this scope, total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglyceride levels were analyzed. In addition, the triglyceride/HDL (TG/HDL) ratio and the LDL/HDL ratio were calculated to evaluate atherogenic risk in more detail.

Data were obtained retrospectively from patient files and the registry system. Patients with missing data were excluded from the evaluation based on the relevant parameter. Statistical analysis was performed using SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). The conformity of continuous variables to a normal distribution was evaluated with the Shapiro-Wilk test. Data showing a normal distribution were presented as mean ± standard deviation, while data not showing a normal distribution were presented as median (minimum-maximum) or median (interquartile range). For the comparison of continuous variables between two independent groups, the independent samples t-test was used for data showing a normal distribution, and the Mann-Whitney U test was used for data not showing a normal distribution. Categorical variables were expressed as numbers and

percentages and were compared between groups using the chi-square test. The statistical significance level was accepted as $p<0.05$.

Results

In the comparison of lipid profile parameters between diabetic and non-diabetic patients, total cholesterol levels were observed to be higher in diabetic patients, but this difference was not found to be statistically significant (201.4 ± 52.3 mg/dL vs 182.7 ± 47.8 mg/dL, p = 0.184). Similarly, although LDL cholesterol levels were found to be higher in the diabetic group, statistical significance was not achieved (124.6 ± 34.2 mg/dL vs 110.3 ± 31.5 mg/dL, p = 0.212). HDL cholesterol levels were observed to be lower in diabetic patients, but this difference was also not found to be significant (41.2 ± 9.1 mg/dL vs 46.5 ± 10.4 mg/dL, p = 0.098). While triglyceride levels were found to be higher in diabetic patients, this increase did not reach the border of statistical significance (196.3 [IQR: 150–305] mg/dL vs 158.4 [IQR: 103–230] mg/dL, p = 0.071).

When atherogenic indices were evaluated, the TG/HDL ratio was found to be significantly higher in diabetic patients (4.8 [IQR: 3.2–6.7] vs 3.1 [IQR: 2.1–4.5], p = 0.041). On the other hand, although the LDL/HDL ratio was found to be higher in the diabetic group, no statistically significant difference was observed (3.1 ± 0.9 vs 2.6 ± 0.8, p = 0.089).

Discussion

In this study, when the lipid profile was compared between diabetic and non-diabetic patients, it was observed that total cholesterol, LDL, and triglyceride levels were higher, while HDL levels were lower in the diabetic group. However, statistical significance could not be obtained for the vast majority of these parameters. In contrast, finding the TG/HDL ratio, one of the atherogenic indices, to be significantly higher in diabetic patients is a striking finding.

The impairment in lipid metabolism in diabetic patients is fundamentally due to insulin resistance. The decrease in the regulatory effect of insulin on lipoprotein lipase activity leads to the accumulation of triglyceride-rich lipoproteins. This situation brings about the clinical picture of "diabetic dyslipidemia," which is particularly characterized by hypertriglyceridemia and low HDL cholesterol (6). The finding that triglyceride levels were higher and HDL levels were lower in diabetic patients in our study is consistent with these pathophysiological mechanisms.

Although no statistically significant difference was detected in LDL cholesterol levels, higher values were observed in the diabetic group; there are similar results in the literature (7). The reason for this may not solely be an increase in the amount of LDL, but also the fact that LDL

particles become smaller and denser (small dense LDL), thereby gaining more atherogenic properties. Therefore, classical LDL measurements alone may not adequately reflect cardiovascular risk.

The most striking finding in our study is that the TG/HDL ratio was found to be significantly higher in diabetic patients. The TG/HDL ratio has been recognized as a strong marker for insulin resistance and atherogenic dyslipidemia in recent years (8). It has been shown that an increase in this ratio is closely associated with endothelial dysfunction and the development of atherosclerosis. Therefore, the significance of the TG/HDL ratio in our study supports the notion that cardiovascular risk is higher in diabetic patients.

The fact that the LDL/HDL ratio was higher in the diabetic group but did not reach statistical significance may be related to the limited sample size. Similarly, the fact that the observed differences in total cholesterol and triglyceride levels did not reach a significant level can also be explained by this situation.

It has been shown in many studies in the literature that the lipid profile is impaired in diabetic patients, and that hypertriglyceridemia and low HDL levels, in particular, increase the risk of cardiovascular events. (9) The findings of our study are also generally consistent with this literature. However, in our study, the emergence of the TG/HDL ratio as a stronger distinguishing parameter compared to classical lipid parameters emphasizes the importance of this index in terms of clinical use.

There are some limitations to this study. Firstly, the relatively small sample size limits the statistical power. Furthermore, the retrospective and single-center nature of the study limits the generalizability of the results. Additionally, lipid parameters were evaluated through single-time measurements, and there are no long-term follow-up data available.

In conclusion, atherogenic changes are observed in the lipid profile of diabetic patients, and the TG/HDL ratio, in particular, stands out as an important indicator of diabetic dyslipidemia and increased cardiovascular risk. These findings need to be supported by prospective studies with larger samples.

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Complete atrioventricular block accompanying inferior myocardial infarction: A case report

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Abstract

In this case report, we present a patient with complete atrioventricular block secondary to inferior myocardial infarction on the background of multivessel coronary artery disease. A 67-year-old male patient with a history of diabetes mellitus was brought to the emergency department by emergency medical services after experiencing syncope. His vital signs on presentation were temperature 36.9°C, blood pressure 50/30 mmHg, pulse 40/minute, respiratory rate 16/minute, and oxygen saturation 95%. He was conscious, oriented, and cooperative. His fingerstick blood glucose was 650 mg/dL. Electrocardiography revealed bradycardia, ST elevation in leads II, III, and aVF, and third-degree atrioventricular complete block. The patient was taken to the resuscitation room and an external pacemaker (30 mV, 90 bpm) was applied, with the voltage gradually increased. A total of 1.5 mg of atropine was administered. After pacing, his blood pressure rose to 120/70 mmHg and his pulse rate to 90/min. Laboratory tests showed hs-troponin 201 ng/L, creatine kinase-MB 13.9 U/L,

creatinine 1.5 mg/dL, and potassium 5.1 mmol/L. Following cardiology consultation, the patient was admitted to the coronary care unit. During coronary angiography, the patient experienced multiple episodes of ventricular tachycardia, and recurrent cardioversion was performed. Coronary angiography revealed 80% stenosis in the proximal left anterior descending artery, 100% occlusion in the post-D1 segment, 90% stenosis in the proximal OM1 branch of the circumflex artery, a long segment lesion of 95% in the proximal right coronary artery, and 100% thrombosis in the right coronary artery posterior descending branch. Stents were placed in the proximal RCA and the branches. A temporary pacemaker was inserted. The patient was followed for 4 days in the intensive care unit and 3 days in the ward. The patient, who did not develop malignant arrhythmia, was discharged in good health. It should be remembered that rapid diagnosis of conduction disorders and myocardial infarction associated with right coronary artery occlusion, and especially early reperfusion therapy, will reduce mortality rates.

Keywords: Complete atrioventricular block, External pacemaker, Inferior myocardial infarction.

Introduction

Acute inferior myocardial infarction (MI) usually occurs as a result of right coronary artery (RCA) occlusion and is often accompanied by bradycardia, hypotension, and atrioventricular (AV) conduction disturbances. Third-degree AV block is observed in approximately 5–10% of inferior MI cases and is an important complication associated with increased mortality (1-2).

Since the RCA typically supplies the sinoatrial and AV nodal arteries, ischemic damage can temporarily or permanently disrupt these conduction pathways (3). Cases of complete AV block accompanying inferior MI due to vasospasm, congenital coronary anomalies, or multivessel disease have been reported in the literature (4-6). In this case report, we present a patient with complete AV block secondary to inferior MI on the background of multivessel coronary artery disease.

Case report

A 67-year-old male patient with a history of diabetes mellitus was brought to the emergency department by emergency medical services after experiencing syncope. His vital signs on presentation were temperature 36.9°C, blood pressure 50/30 mmHg, pulse 40/minute, respiratory rate 16/minute, and oxygen saturation 95%. He was conscious, oriented, and cooperative. His fingerstick blood glucose was 650 mg/dL. Electrocardiography (ECG) revealed bradycardia, ST elevation in leads II, III, and aVF, and third-degree AV complete block (Figure 1). The patient was taken to the resuscitation room and an external pacemaker (30 mV, 90 bpm) was applied, with the voltage gradually increased. A total of 1.5

mg of atropine (IV) was administered. After pacing, his blood pressure rose to 120/70 mmHg and his pulse rate to 90/min. Laboratory tests showed hs-troponin 201 ng/L, creatine kinase-MB 13.9 U/L, creatinine 1.5 mg/dL, and potassium 5.1 mmol/L. Following cardiology consultation, the patient was admitted to the coronary care unit. During coronary angiography, the patient experienced multiple episodes of ventricular tachycardia (VT), and recurrent cardioversion was performed. Coronary angiography revealed 80% stenosis in the proximal left anterior descending artery (LAD), 100% occlusion in the post-D1 segment, 90% stenosis in the proximal OM1 branch of the circumflex artery, a long segment lesion of 95% in the proximal RCA, and 100% thrombosis in the RCA posterior descending (PD) branch. Stents were placed in the proximal RCA and the branches. A temporary pacemaker was inserted. The patient was followed for 4 days in the intensive care unit and 3 days in the ward. The patient, who did not develop malignant arrhythmia, was discharged in good health.

Discussion

AV complete block accompanying inferior MI usually results from AV nodal ischemia due to RCA occlusion (1,3). The RCA is the artery that supplies the AV node and the proximal part of the His bundle. Critical lesions in the RCA can cause significant conduction delays, particularly in dominant right coronary systems (7,8). In a case of AV complete block associated with inferior MI, early reperfusion has been reported to reduce mortality by restoring conduction (3). In our case, the severe proximal lesion in the RCA impaired the perfusion of the AV nodal artery, leading to the development of complete block. Additionally, the presence of a lesion in the LAD further worsened the clinical picture by reducing myocardial reserve. Early diagnosis and the application of an external pacemaker ensured hemodynamic stabilization and prevented possible fatal outcomes.

Conclusion

It should be remembered that rapid diagnosis of conduction disorders and myocardial infarction associated with RCA occlusion, and especially early reperfusion therapy, will reduce mortality rates.

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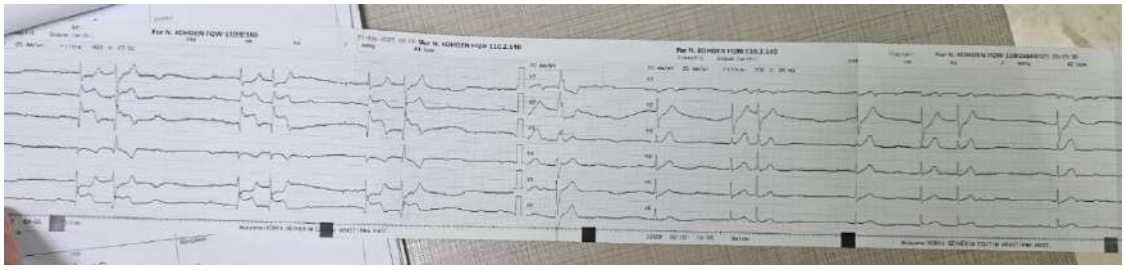

Figure 1. ECG taken at the time of application

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Hematocrit and LDL Cholesterol Relationship At High Altitude:Early Cardiovascular Risk Phenotype In Young Men

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Introduce and Purpose

The aim of this study was to evaluate the relationship between hematocrit levels and lipid profile in young male individuals living at high altitude and to investigate whether the coexistence of altitude-dependent erythrocytosis and atherogenic lipid profile constitutes a potential cardiovascular risk phenotype.

Chronic hypobaric hypoxia can lead to erythrocytosis and vascular hemodynamic features that increase blood viscosity. In this situation, particularly high LDL consumption can

accelerate atherogenic growths such as endothelial dysfunction and LDL oxidation. However, the relationship between this hematological adaptation and lipid metabolism in young populations living at high altitudes has not been sufficiently investigated.

Although no significant linear relationship was found between hematocrit and LDL cholesterol levels in young male individuals living at high altitudes, it is thought that these two parameters coexist at a high prevalence in the same individuals, potentially constituting a pro-atherogenic risk phenotype.

Therefore, our study aims to evaluate the frequency of erythrocytosis and dyslipidemia co-occurrence in high-altitude populations and the relationship between these parameters.

Materials and Method

This study was planned as a retrospective and cross-sectional study analyzing laboratory data obtained during routine clinical practice.

The study was conducted at the Şehit Aydoğan Aydın State Hospital in Şırnak Province, located in southeastern Turkey at an altitude of approximately 1300–1400 meters above sea level. Individuals living in this region are chronically exposed to moderately high altitude conditions.

The study population consisted of 200 male individuals aged 30–45 years who had been living at high altitude for at least one year.

Known cardiovascular disease, chronic kidney disease, chronic liver disease, active infection, and the presence of secondary causes of erythrocytosis were considered exclusion criteria. Additionally, female individuals were excluded from the study to eliminate gender-dependent physiological variability in hematological and lipid parameters and to obtain a homogeneous study population.

The study covers the period from January 2025 to March 2026..

Data such as age, hemoglobin, hematocrit, red blood cell count (RBC), platelet count, CRP, HbA1c, and LDL cholesterol levels were obtained retrospectively from the hospital's electronic medical record system.

All blood samples were taken after fasting and analyses were performed using standard methods in the same central laboratory.

Definitions

Erythrocytosis: Hematocrit ≥49% according to World Health Organization criteria

High LDL cholesterol: LDL cholesterol levels were considered high if they were ≥100 mg/dL according to the European Society of Cardiology (ESC/EAS) guidelines. In addition, LDL levels ≥130 mg/dL were considered severely high.

Findings and Conclusion

The mean age of the participants was 38.8 ± 8.7 years. The mean hemoglobin level was 16.88 ± 0.99 g/dL and the mean hematocrit level was 49.8 ± 2.76%. The mean LDL cholesterol level was 120.7 ± 29.4 mg/dL.

In 74% (n=148) of participants, hematocrit was found to be ≥49%, while in 77.5% (n=155), LDL cholesterol was ≥100 mg/dL. The percentage of individuals with LDL ≥130 mg/dL was 37.5% (n=75). Both high hematocrit (≥%49) and high LDL cholesterol (≥100 mg/dL) were observed in 59% (n=118) of participants.

Correlation Analysis

The relationship between hematocrit and LDL cholesterol levels was evaluated using Spearman correlation analysis. No statistically significant correlation was found between the two variables ($r = 0.08$, $p = 0.24$).

Determinants of High-Risk Phenotype

In a multivariate logistic regression analysis conducted to evaluate the independent determinants of the high-risk phenotype (coexistence of high hematocrit and high LDL cholesterol), erythrocyte count was found to be an independent determinant (OR: 1.75, 95% CI: 1.20–2.55; p=0.01).

When participants were divided into two groups based on their hematocrit levels and compared, it was found that the high hematocrit group (≥49%) had a significantly higher red blood cell count (5.90 ± 0.42 vs. 5.43 ± 0.37 $\times 10^6/\mu L$, $p<0.001$). Furthermore, the average age was found to be lower in this group (37.8 ± 7.7 vs. 41.8 ± 10.7 year, p=0.016).

No statistically significant difference was found between the groups in terms of LDL cholesterol levels (118.8 ± 29.9 mg/dL vs. 126.1 ± 27.6 mg/dL, p=0.113). Similarly, although the incidence of LDL ≥100 mg/dL tended to be lower in the high hematocrit group, this difference did not reach statistical significance (74.3% vs. 86.5%, p=0.105).

When the relationship between hematocrit and LDL cholesterol levels was evaluated using **scatter plot** analysis, no significant linear correlation was found. However, the distribution of data points indicated heterogeneity in LDL levels across a wide hematocrit range.

However, the high prevalence of high hematocrit and high LDL cholesterol together indicates that these two parameters can frequently coexist in the same individuals. This finding suggests that hematological and metabolic cardiovascular risk factors may tend to cluster in young individuals living at high altitudes. The findings may indicate that not only metabolic factors but also hematological adaptations should be considered in cardiovascular risk assessment.

The findings should be interpreted not as defining a "relationship" in the classical sense, but rather as an epidemiological co-occurrence pattern reflecting the tendency of cardiovascular risk factors to cluster at the individual level. Indeed, modern cardiovascular risk models increasingly emphasize the prognostic significance of the clustering of risk factors in the same individual, rather than their individual effects. This co-occurrence observed in our study can be explained by a hemodynamic-metabolic interaction.

This study is expected to contribute to the identification of an early cardiovascular risk phenotype by revealing the coexistence of erythrocytosis and dyslipidemia in young populations living at high altitudes. The findings may demonstrate that not only metabolic factors but also hematological adaptations should be considered in cardiovascular risk assessment.

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Approach to "Simple" Trauma in the Emergency Department: Intracranial Injury Detected Following Wire Penetration

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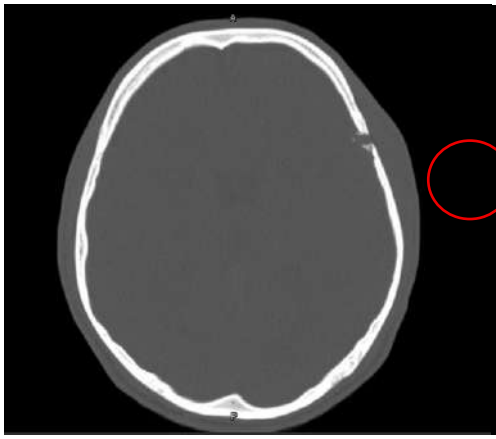
Introduction

Although minor head traumas are frequent among emergency department visits, penetrating injuries caused by occupational accidents require special attention. Particularly, flying fragments (projectile objects) during metal cutting carry significant kinetic energy due to their high velocity, despite having a small mass. In such injuries, the entry wound on the skin can be small and misleading; however, there is a high risk of fracture in the underlying bone tissue and parenchymal damage (1). In the literature, even for patients with a Glasgow Coma Scale (GCS) of 15, "suspected open or depressed skull fracture" is defined as a high-risk factor under the Canadian CT Head Rule (CCHR) (2). This presentation reports a case of a patient who was clinically stable but radiologically diagnosed with a parietal fracture and contusion caused by a metal fragment that flew off while cutting wire at the workplace.

Case Report

A 19-year-old male patient presented to the emergency department on foot with the complaint of being struck in the head by a snapped piece of wire that flew off while he was cutting metal wire at his workplace. The patient did not report any loss of consciousness, vomiting, or seizures at the time of the incident. The patient's general condition was good, and he was evaluated as conscious, oriented, and cooperative (GCS: 15). The neurological examination revealed no motor or sensory deficits, nor any cranial nerve pathology. Scalp examination showed a penetrating entry hole of approximately 1 cm in diameter, with irregular borders and no active bleeding, in the left parietal region.

Because the injury was caused by a high-velocity object and had a penetrating nature, a non-contrast Brain Computed Tomography (CT) was performed. CT Findings**:** A minimally displaced fracture line was detected in the left parietal bone (Figure 1), along with a minimal contusion area in the brain parenchyma (at the cortical level) immediately adjacent to this line (Figure 2). No epidural or subdural hematoma was observed. The patient was consulted with the neurosurgery clinic. A prophylactic tetanus vaccine was administered, and broad-spectrum antibiotic therapy (Ceftriaxone) was initiated to prevent the risk of intracranial infection. The patient was admitted to the neurosurgery clinic.



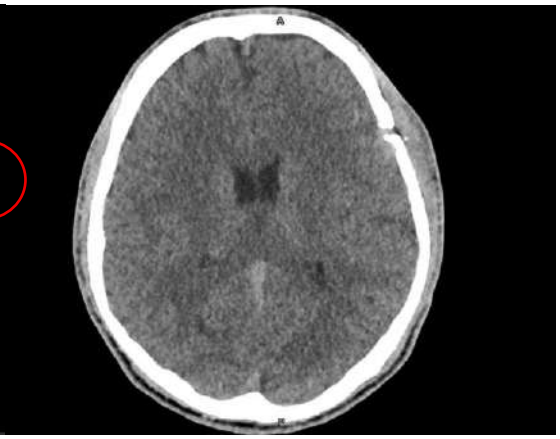


Figure 1 Figure 2

Discussion

This case demonstrates that the "mechanism" detail in the anamnesis (flying off while cutting wire) is far more decisive than the physical examination findings. Even though the wire fragment was very light, the release of tension at the moment of cutting caused it to reach high velocities like a bullet, thus disrupting the integrity of the skull.

In clinical practice, a 1 cm incision might easily be evaluated as a simple laceration and the patient discharged after suturing. However, the presence of a "displaced fracture" and "contusion" in this case introduces the risk of a dural tear and exposure of the brain parenchyma to the external environment (pneumocephalus, brain abscess, meningitis) (3). Therefore, in patients with a history of a high-velocity flying fragment, the threshold for cranial imaging should be kept low, even if the GCS is 15. This approach is critical in preventing overlooked intracranial infections and late-stage neurological sequelae.

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3019

Evaluation of Emergency Department Triage in a Tertiary Hospital Over the Years

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Introduction:

Triage systems are vital in emergency departments to prevent delays in patient care and efficiently distribute the increasing workload. In Türkiye, the number of emergency department admissions is constantly increasing, affecting all aspects of emergency department service delivery and triage practices.

AIM:

To compare emergency department triage characteristics, patient flow, and hospitalization rates in a tertiary referral hospital between 2024 and 2025.

Material and Methods:

A retrospective review was conducted for all emergency department admissions to Malatya Training and Research Hospital between January-December 2024 and January-December 2025. Data on total admissions, repeat admissions, ambulance arrivals, triage categories, observation rates, and hospitalization rates were analyzed and compared across years.

Results

A total of 902,347 patients visited the emergency department in 2024, and 899,585 patients in 2025. Repeat visits decreased from 7,912 in 2024 to 6,271 in 2025. The number of patients brought by ambulance services (112) remained at 38,778 in both years.

The number of patients referred to the green zone increased significantly from 83,007 in 2024 to 421,841 in 2025, while visits to the yellow zone decreased from 805,331 to 463,531. The number of hospitalized patients increased from 23,187 in 2024 to 25,071 in 2025.

Table 1

Değişken	2024 ort.	2025 ort.	p	
Yatış	1932.3	2089.3	0.002	*
112	1231.1	1782.8	0.017	
Gözlem	33167.0	29888.7	0.012	
Toplam	58288.2	59755.5	0.046	
Yeşil	6917.3	35153.4	<0.001	
Sarı	67110.9	38627.6	<0.001	

Monthly average comparisons showed significant differences between years: hospitalization rates (1932.3 vs 2089.3, p=0.002), ambulance arrivals (1231.1 vs 1782.8, p=0.017), observation admissions Table 1 shows the differences in admissions by region: 29,888.7 (33,167.0 vs. 29,888.7, p=0.012), total number of patients (58,288.2 vs. 59,755.5, p=0.046), green zone admissions (6,917.3 vs. 35,153.4, p<0.001), and yellow zone admissions (67,110.9 vs. 38,627.6, p<0.001).

The region-based evaluation showed that the mean values were 21,035.3 ± 14,951.7 for the green zone, 52,869.3 ± 15,952.2 for the yellow zone, and 1,175.9 ± 95.3 for the red zone.

Discussion:

Malatya Training and Research Hospital is a rapidly developing emergency department. The significant increase in green zone triage, the decrease in repeat admissions, and the increase in hospitalization rates despite similar total patient numbers suggest that triage accuracy will improve and there will be more resident physicians by 2025. However, the very low hospitalization rate (approximately 2%) in both years may also indicate a significant burden of non-emergency emergency department visits.

Keywords

Emergency department, triage, hospitalization

3036

Cold Agglutinin Disease-Related Hemolytic Anemia and Associated Hepatobiliary Dysfunction in the Emergency Department

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ABSTRACT

Introduction and Purpose: Cold agglutinin disease (CAD) and cold agglutinin syndrome (CAS) are rare forms of autoimmune hemolytic anemia characterized by complement-mediated hemolysis triggered by cold exposure. Patients frequently present with nonspecific symptoms, which may delay diagnosis, particularly in the emergency department. Laboratory inconsistencies such as hemoglobin–hematocrit discordance and erythrocyte agglutination may provide important diagnostic clues. This report presents a case of cold agglutinin–related hemolytic anemia associated with hepatobiliary dysfunction following prolonged cold exposure.

Case Presentation: A 61-year-old woman presented to the emergency department with bilateral leg pain and cramps, right upper quadrant abdominal pain, and dyspnea. Her medical history included diabetes mellitus, hypertension, and chronic obstructive pulmonary disease. Symptoms developed after an approximately 18-hour bus journey from a warm to a colder region, during which the patient experienced significant cold exposure. Physical examination revealed mild jaundice and right upper quadrant tenderness. Initial laboratory findings showed hemoglobin–hematocrit inconsistency, markedly elevated mean corpuscular hemoglobin concentration, leukocytosis, increased lactate dehydrogenase, and hyperbilirubinemia accompanied by elevated hepatobiliary enzymes. Reanalysis of the blood sample after warming at 37°C corrected the hematologic discrepancies. Peripheral blood smear demonstrated erythrocyte agglutination, supporting the diagnosis of cold agglutinin disease.

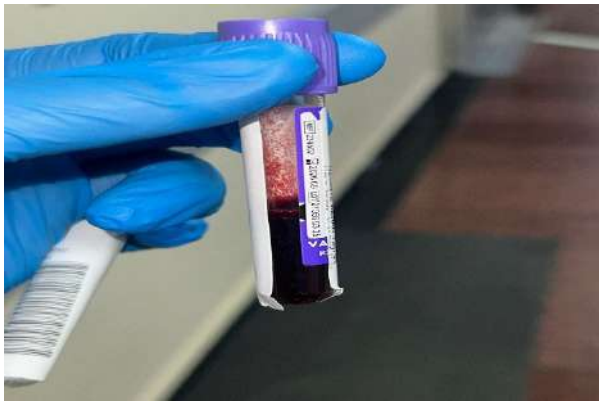


Figure 1. Erythrocyte agglutination in a hemogram tube

Results and Conclusion: This case demonstrates that cold agglutinin–related hemolytic anemia may present with atypical symptoms and multisystem involvement, including hepatobiliary enzyme elevation. Recognition of laboratory inconsistencies and a history of cold exposure are essential for early diagnosis. Considering CAD in patients presenting with hemolysis and unexplained biochemical abnormalities in the emergency department may prevent diagnostic delay and facilitate appropriate management.

Keywords: Cold agglutinin disease, autoimmune hemolytic anemia, hepatobiliary dysfunction, emergency department, hemolysis

INTRODUCTION

Cold agglutinins are seen in primary Cold Agglutinin Disease (CAD) and secondary Cold Agglutinin Syndrome (CAS). Primary CAD is considered a clonal B-cell lymphoproliferative disorder, while secondary CAS arises due to an underlying cause such as a specific infection, autoimmune disorder, or lymphoid malignancies (1).

Rather than clear diagnostic parameters, clinical and laboratory inconsistencies are noteworthy. The generally accepted diagnostic criteria include the presence of hemolysis findings, a positive Coombs test for C3d, and a cold agglutinin titer >64 at 4°C (1-3). In most patients, the diagnosis is made long after the onset of symptoms (1,2).

The primary treatment is protection from cold, but anemia caused by hemolysis should be treated according to the situation. While no specific treatment is required in mild cases, transfusion and plasmapheresis should be considered in cases of severe anemia. In addition, treatment should be individualized according to the underlying disease, and a regimen including rituximab should be planned (4).

CASE PRESENTATION

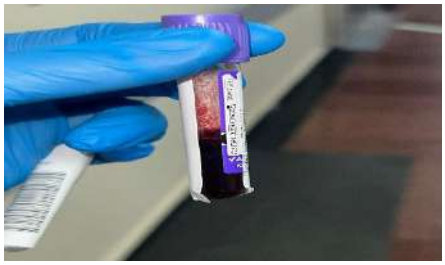
A 61-year-old female patient presented to the emergency department with complaints of pain and cramps starting in the groin and spreading to the legs for 2 days, new-onset right upper quadrant pain, and shortness of breath. She had known diagnoses of diabetes mellitus (DM), hypertension (HT), and chronic obstructive pulmonary disease (COPD) and stated that she took her medications regularly and had no recent medication changes. She had a history of cholecystectomy. It was learned that the patient had traveled by bus for 18 hours that day. It was learned that this trip was from a warm climate to a cold climate and that the patient felt very cold during breaks during the trip.

During the physical examination of the patient, his short stature and body mass index (BMI) >30 (obese) were the first elements that attracted attention during inspection. Vital signs were

body temperature 36.7 °C, blood pressure 110/60 mmHg, pulse 67 beats/min, respiratory rate 15/min, and saturation 93%. Inspection revealed that the patient's skin color and sclerae were slightly jaundiced. Palpation revealed tenderness in the right upper quadrant. Examination of the lower extremities revealed no noticeable difference in temperature or diameter, and no skin findings were observed. Breath sounds were normal on auscultation, and no pathological sounds were heard.

Laboratory findings: hemoglobin (Hgb): 11.1 g/dL, hematocrit (Htc): 1.2, mean corpuscular volume (MCV): 166 fL, mean corpuscular hemoglobin concentration (MCHC): 925 g/dL, platelets (PLT): 235,000/mm3, leukocytes (WBC): 18,100/mm3, neutrophils: 15,600/mm3, lactate dehydrogenase (LDH): 727 U/L, total bilirubin (TBIL): 4.8 mg/dl, direct bilirubin (DBIL): 1.3 mg/dl, alanine aminotransferase (ALT): 103 U/L, aspartate aminotransferase (AST): 106 U/L, gamma-glutamyl transferase (GGT): 561 U/L, red cell distribution width-standard deviation (RDW-SD): 276 fL, C-reactive protein (CRP): 35 mg/L.

Due to the patient's current physical examination findings, elevated liver function tests, and signs of hemolysis, a hepatobiliary ultrasound (USG) was performed. The USG showed: liver craniocaudal length 180 mm (hepatomegaly), grade 2 increased parenchymal echogenicity (hepatosteatosis), and a 17x9 mm hypoechoic appearance with no detectable vascularity on color Doppler ultrasound (RDUS) at the portal hilum level (lymph node) were observed. This picture suggested cholestatic/hepatocellular damage accompanied by an increase in indirect bilirubin due to hemolysis. The patient's abnormal MCHC value, inconsistent Hgb-Htc values, the presence of hemolysis, and the onset of symptoms after a long journey to a cold region raised suspicion of cold agglutinin disease. A repeat blood sample was taken from the patient, placed in an ethylenediaminetetraacetic acid (EDTA) tube, warmed to 37°C, and processed in the laboratory. Laboratory results for the second sample: TBIL: 5 mg/dl, DBIL: 1.7 mg/dl, ALT: 94 U/L, AST: 108 U/L, GGT: 549 U/L, LDH: 731 U/L, CRP: 35 mg/dl, WBC: 17000/mm3, NEU: 14,000/mm3, Hgb: 9.7 g/dl, Htc: 11.9, PLT: 206,000/mm3, MCV: 159 fL, MCHC: 81 g/dl, RDW-SD: 288 fL. The peripheral smear requested from the patient was examined at 100x magnification; an increase in segmented neutrophils and agglutination in erythrocytes were observed in the leukocyte formula. Cells with atypical lymphoid characteristics and dark basophilic cytoplasm were observed in some areas. The platelet count was normal. Erythrocyte agglutination was observed in the blood sample tube taken without heating the blood



(Figure 1). **Figure 1. Erythrocyte agglutination in a hemogram tube**

Based on the patient's findings, a new diagnosis of cold agglutinin disease was considered. In the emergency department, measures were taken to prevent cold exposure. The patient was actively warmed; the room temperature was increased, heated blankets were used, and all intravenous fluids were administered warm. Care was taken to preserve peripheral circulation. Due to the significant anemia detected at the time of admission, a red blood cell suspension transfusion was planned. Considering the presence of cold agglutinins, blood products were warmed prior to transfusion, and intravenous lines were kept warm during transfusion. During transfusion, the patient was closely monitored clinically and laboratory-wise for signs of hemolysis. The patient was referred to a referral center for planning further investigations and treatment.

DISCUSSION

Cold agglutinin syndrome (CAS) is a rare subtype of autoimmune hemolytic anemia and often presents with nonspecific clinical findings that lead to delayed diagnosis (2,3). Patients frequently present with fatigue, dyspnea, acrocyanosis, or symptoms that worsen after exposure to cold (1,3). In the emergency department, the picture often mimics infection, hepatobiliary disease, or thromboembolic events (2,5). Therefore, CAS is a condition that can easily be overlooked, especially on initial evaluation (3,5).

In the presented case, one of the most important clues leading to the diagnosis was laboratory inconsistencies. The marked MCV elevation detected in the initial hemogram, the MCHC value reported above physiological limits, and the hemoglobin-hematocrit inconsistency are consistent with the classic preanalytical findings described in the literature for CAD. The aggregation of erythrocytes in the presence of cold agglutinins can lead to incorrect measurements, which are often interpreted as device errors. However, the normalization of values after re-testing at 37°C, for example, is critical for diagnosis (1,2,6). This situation demonstrates that emergency physicians must evaluate laboratory results not only numerically but also from a pathophysiological perspective.

Another important point to note in this case is the long bus journey prior to the onset of symptoms. In CAD, cold exposure can trigger hemolysis by increasing IgM-mediated erythrocyte agglutination in peripheral circulation. Prolonged immobility and peripheral vasoconstriction may also facilitate this process. Exposure to cold environments, outdoor exposure, and conditions with reduced peripheral circulation have been identified in the literature as factors that trigger hemolytic attacks (1,3). In this respect, our case is noteworthy in that it demonstrates the initiating role of environmental factors in the clinical picture.

One of the important features that distinguishes this case from many CAD cases in the literature is the accompanying marked hepatobiliary dysfunction. In addition to increased

indirect bilirubin, elevated transaminases and cholestatic enzymes were detected in the patient. Although the fundamental pathophysiology of CAD is indirect hyperbilirubinemia due to hemolysis, in cases of intense hemolysis, the liver's inability to cope with the bilirubin load and the increased inflammatory response can lead to elevated hepatic enzymes. However, CAD cases with marked hepatobiliary enzyme elevation are rare in the literature (3,7,8). This situation demonstrates that the systemic effects of hemolysis are not limited to hematological parameters and require a multisystemic evaluation.

A significant proportion of CAD cases are associated with underlying secondary causes. Infections, autoimmune diseases, and especially lymphoproliferative diseases are among the frequently reported etiological factors (1,3). In our case, the presence of atypical lymphoid cells in the peripheral smear suggested a secondary cause, and further investigations led to a diagnosis of Waldenström macroglobulinemia.

The most important approach in the management of CAD in the emergency department is to prevent cold exposure and actively warm the patient. Care should be taken as unnecessary or inappropriate blood transfusions may increase agglutination. When transfusion is necessary, warmed blood products should be preferred and intravenous lines should also be kept warm (1,3). In this case, considering the diagnosis in the emergency department and referring the patient to a referral center demonstrates that early awareness plays a critical role in preventing complications.

In conclusion, this case highlights the importance of carefully evaluating laboratory inconsistencies in patients presenting to the emergency department with nonspecific symptoms. A history of cold exposure, hemolytic findings, and erythrocyte agglutination in the peripheral smear should be considered warning signs for CAD. Furthermore, the accompanying hepatobiliary dysfunction is noteworthy as it demonstrates the systemic effects of the disease. In these respects, our case contributes to the literature by drawing attention to the atypical and multisystemic presentation of CAD in the emergency department.

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3051

Hepatic Portal Venous Gas (PVG) due to Alkaline Corrosive Agent Ingestion in a Patient with Prior Sleeve Gastrectomy: A Case Report of an Uncommon Presentation

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Abstract

Hepatic portal venous gas (PVG) has traditionally been regarded as a radiologic harbinger of catastrophic intra-abdominal pathology, most notably bowel ischemia requiring urgent surgery.

We report a unique case of a 36-year-old woman presenting with hematemesis after accidental ingestion of an alkaline household cleaning agent (sodium percarbonate), set apart by her history of a prior sleeve gastrectomy. The patient unknowingly ingested approximately 50 mL of the substance after it was mistaken for water and subsequently developed recurrent hematemesis. Contrast-enhanced Computed Tomography revealed PVG within the hepatic portal venous system, reflecting significant mucosal injury but notably without free air, free fluid, or other radiological signs of perforation. Despite this unexpected radiologic finding often linked with severe intra-abdominal pathology, the patient remained hemodynamically stable, had normal lactate levels, and exhibited no peritoneal signs. Conservative management

led to an uncomplicated outcome. We delineate an evidence-informed algorithmic framework for PVG clinical evaluation and management, underscoring the diagnostic nuances in this singular clinical scenario.

Keywords: *portal venous gas; alkaline corrosive, caustic ingestion, sleeve gastrectomy, emergency algorithm*

Introduction

Hepatic portal venous gas (PVG) was first described in 1955 in association with neonatal necrotizing enterocolitis[1], and later became strongly linked to mesenteric ischemia and bowel necrosis, carrying mortality rates as high as 75% in early surgical series.[2] For decades, its radiographic detection was considered a mandate for urgent surgical exploration.[3] However, the widespread use of multidetector computed tomography (CT) has fundamentally shifted the epidemiology and clinical interpretation of PVG.[4,5]

It is now recognized as a non-specific radiological sign occurring across a broad clinical spectrum, ranging from life-threatening bowel infarction to benign, self-limiting processes such as iatrogenic injury (e.g., endocolonic procedures), inflammatory disorders, and states of increased intraluminal pressure. This evolution presents a clinical dilemma: how to respond when PVG is detected incidentally in a stable patient. While a universal management algorithm is lacking, current paradigms emphasize risk stratification based on clinical stability,[5] laboratory markers (e.g., lactate levels), and CT evidence of transmural injury rather than the presence of gas alone. Three primary pathophysiological mechanisms facilitate PEG formation: (1) mucosal barrier disruptions, allowing intraluminal gas to enter the mesenteric venous circulation; (2) increased intraluminal pressure promoting gas translocation; and (3) invasion by gas-forming bacteria in the bowel wall or portal system. Consequently, the primary challenge in emergency settings has transitioned from mere detection to effective risk stratification, specifically distinguishing ischemic from non-ischemic ideologies.

Conversely, alkaline corrosive agents can induce liquefaction necrosis, allowing deep penetration into the gastrointestinal wall and greatly increasing the risk of transmural injury, acute perforation, and long-term effects such as strictures or malignant transformation.[6] Patients with altered gastrointestinal anatomy, including those who have undergone bariatric surgery, may be predisposed to atypical injury patterns and complications following corrosive exposure. PVG is an uncommon but significant radiologic finding that may reflect severe mucosal injury, ischemia, or increased intraluminal pressuree.[7] Clinical severity depends on the agent's chemical properties, ingested volume, duration of mucosal contact, and individual patient factors. Notably, those with prior sleeve gastrectomy may exhibit atypical injury patterns and unique complications. In these contexts, PVG serves as a critical marker of severe mucosal damage, ischemia, or pathologically elevated intraluminal pressure. However, recent evidence suggests that PVG following caustic injury can sometimes be transient and manageable with conservative treatment in stable patients.[7]

We present a case where PVG was identified as an incidental CT finding during the workup of accidental alkaline caustic ingestion in a patient with prior sleeve gastrectomy, complicated by

hematemesis and Zargar grade 2B gastrointestinal injury. This case highlights diagnostic and management challenges in this unique context and outlines an evidence-informed algorithmic framework for clinical evaluation and management.

Case Presentation

A 36-year-old woman with no comorbidities presented after accidentally ingesting about 50 ml of an alkaline cleaning agent (Vanish Oxi-actionÒ) given in error as water. She then drank 500 mL of water and vomited, with fresh blood followed by intermittent hematemesis. Her medical history included a laparoscopic sleeve gastrectomy 6 years earlier. She was not on regular medications and had no known allergies.

On presentation, the patient was alert and hemodynamically stable. Physical examination revealed a soft, non-tender abdomen without peritoneal signs. Cardiopulmonary examination was unremarkable. During observation, she reported intermittent retrosternal odynophagia and a single episode of bilious vomiting, without recurrent hematemesis.

Laboratory tests showed mild anemia (hemoglobin 11.4 g/dL). Renal and hepatic function were normal, including serum creatinine (0.81 mg/dL; eGFR >90 mL/min/1.73 m^2), transaminases (AST 14 U/L, ALT 13 U/L), and bilirubin. Electrolytes were unremarkable, except for a mild hyponatremia on venous sampling (Na^+ 134 mmol/L) that corrected on repeat (139 mmol/L). Venous blood gas analysis showed a normal pH (7.399), a mildly reduced pCO_2 (36.7 mmHg), bicarbonate of 22.2 mmol/L, and normal lactate (1.8 mmol/L), with no acid–base or metabolic disturbance. Inflammatory markers were normal, and serial CBCs showed no leukocytosis. Urinalysis showed marked hematuria (+++; 153 erythrocytes/ high-power field) without leukocyturia or nitrituria. Serial labs showed no systemic inflammation, metabolic imbalance, or organ dysfunction.

Conservative management included nil per os, intravenous proton pump inhibitor (80 mg bolus then 40 mg every 6 hours), IV fluids, and close monitoring. Neutralizing agents, nasogastric tube placement, and corticosteroids were avoided per recommendations. Airway patency was monitored. Otolaryngology, Internal Medicine, and Infectious Diseases were consulted. Otolaryngology confirmed no laryngeal or pharyngeal involvement. Internal Medicine planned an upper GI endoscopy to be performed within the recommended 6–24-hour window.[8] Endoscopy showed linear erosions from the gastroesophageal junction, diffuse gastric edema and hyperemia, and linear ulcerations in the duodenum's second portion. Findings were consistent with Los Angeles grade B esophagitis, pangastritis, and erosive duodenitis, correlating with Zargar grade 2B corrosive injury (Figures 1 and 2).[6]

Contrast-enhanced abdominal computed tomography demonstrated gas-compatible densities within the portal venous system of the liver, consistent with portal venous gas, without evidence of free intraperitoneal air, free fluid, pneumoperitoneum, intestinal wall non-enhancement, or solid organ injury (Figure 3). Serial abdominal examinations revealed no signs of peritonitis or perforation. In the absence of fever, leukocytosis, hemodynamic instability, or radiologic evidence of perforation or infection, empiric antibiotic therapy was

not initiated following infectious diseases consultation. The patient was admitted to the gastroenterology service for continued close monitoring. The clinical course was uneventful.

Discussion

Portal venous gas (PVG) is an uncommon finding in corrosive ingestion and has traditionally been associated with bowel ischemia or necrosis. However, emerging evidence and contemporary CT-based series report markedly lower mortality rates, particularly when ischemia is excluded. Recent analyses indicate that: (1) mortality correlates strongly with elevated serum lactate levels; (2) hemodynamic instability is a stronger predictor of adverse outcomes than the radiological burden of PVG; and (3) CT features such as bowel wall non-enhancement, mesenteric occlusion, pneumatosis intestinalis, and free intraperitoneal air carry greater prognostic significance. Thus, PVG should be interpreted within a clinical–radiological–laboratory triad rather than as an independent predictor of mortality.[9]

Three principal mechanisms are traditionally implicated in PVG formation: (1) Mucosal barrier disruption permitting intraluminal gas to enter the mesenteric venous circulation, (2) increased intraluminal pressure, facilitating transportation of luminal gas, and (3) gas-forming bacterial invasion. In our case, two main mechanisms likely contributed to PVG formation: first, the ingested alkaline substance (Vanish®) contains non-ionic surfactant (5%) and 5% sodium percarbonate. Alkaline agents cause liquefaction necrosis, leading to mucosal injury and increased permeability. Endoscopy confirmed significant mucosal damage (Zargar grade 2B). Disruption of the mucosal barrier allows intraluminal gas to enter the portal venous circulation. Second, sodium percarbonate decomposes into hydrogen peroxide, which further breaks down into water and oxygen. It has been reported that 1 ml of hydrogen peroxide can release a substantial volume of oxygen gas (100 ml). This oxygen production increases intraluminal pressure. The patient's history of sleeve gastrectomy adds further clinical relevance. Altered gastric anatomy and accelerated transit may facilitate distal gastrointestinal exposure to corrosive agents, potentially explaining the prominent duodenal involvement and development of portal venous gas.[10]

Importantly, the patient was hemodynamically stable, had normal lactate levels, and showed no CT signs of bowel ischemia, including wall nonenhancement or free air. Recent studies indicate that mortality is strongly associated with hemodynamic instability and elevated lactate levels rather than with PVG alone. Therefore, PVG should be interpreted as a clinical sign that requires careful evaluation of the overall condition.[5]

Surgical consultation remains the inviolable default in any patient with peritoneal signs, hemodynamic compromise, lactic acidosis, or imaging features of bowel ischemia or perforation. In selected patients, PVG may result from severe mucosal injury and increased intraluminal pressure, permitting gas translocation into the portal circulation.[8] The absence of systemic inflammation, lactic acidosis, and hemodynamic compromise in this patient supports a benign, transient mechanism rather than true intestinal ischemia. In such cases, conservative management with close surveillance may be appropriate.[8]

The clinical algorithm for portal venous gas (PVG) is not about whether it is present, but about why it is present and whether the underlying cause requires surgery. The following algorithm structures this evaluation across four sequential steps.

Step 1 - Immediate clinical assessment. The first and most important question is whether the patient is in immediate danger. Any combination of hemodynamic instability (hypotension, signs of shock), peritoneal signs (acute abdomen, abdominal guarding, rigidity, rebound), or signs of septic shock constitutes a surgical emergency, irrespective of the suspected etiology. These patients require immediate surgical consultation and resuscitation.

Step 2 – Identify the causative etiology. In the stable patient, the clinical history should be used to classify the likely cause as high-risk or low-risk. High-risk causes – including mesenteric ischemia, necrotizing enterocolitis, closed-loop small bowel obstruction, and portal vein thrombosis – are associated with a high probability of requiring surgical or vascular intervention. Low-risk causes – including post-endoscopic insufflation, inflammatory bowel disease, caustic ingestion, and blunt abdominal trauma without solid organ injury – may be managed conservatively when other parameters are reassuring.

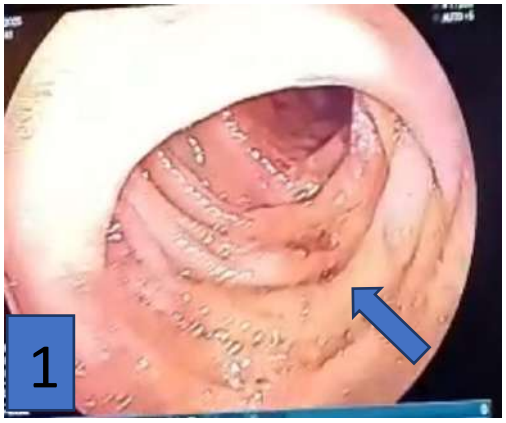
Step 3 – Re-evaluate CT morphology. Even in a clinically stable patient with a low-risk etiology, specific CT findings should independently prompt surgical consultation; pneumatosis intestinalis, free intraperitoneal air (pneumoperitoneum), focal bowel wall non-enhancement suggesting transmural ischemia, or significant free peritoneal fluid, PVG in isolation – without these accompanying features – carries a substantially lower surgical risk.

Step 4 – Biochemical assessment. Serum lactate is the most clinically useful single biomarker for identifying occult mesenteric ischemia. Elevated lactate, leukocytosis, or evidence of organ dysfunction should prompt escalation of care regardless of apparent clinical stability. Normal biochemistry in a stable patient with a low-risk etiology and a reassuring CT supports a conservative management strategy with close serial monitoring. The resulting clinical pathway is illustrated in Figure 4.

Current evidence does not support routine use of antibiotics, corticosteroids, or neutralizing agents in the absence of infection or perforation.[11,12] Optimal management relies on early risk stratification, timely endoscopic evaluation, supportive care, and multidisciplinary collaboration.

Conclusion

This case demonstrates that PVG can arise as a transient and clinically benign finding in the context of severe caustic ingestion, driven by alkaline liquefactive necrosis, pressure-mediated gas dissection through a disrupted mucosal barrier, and potentially altered gastrointestinal anatomy secondary to prior sleeve gastrectomy. PVG should prompt a structured, stepwise clinical evaluation. Hemodynamic stability, peritoneal signs, etiological context, CT morphology (without signs of irreversible ischemia), and serum lactate together determine the appropriate management and early surgical consultation. We support a risk-based approach and highlight the critical role of early risk stratification.



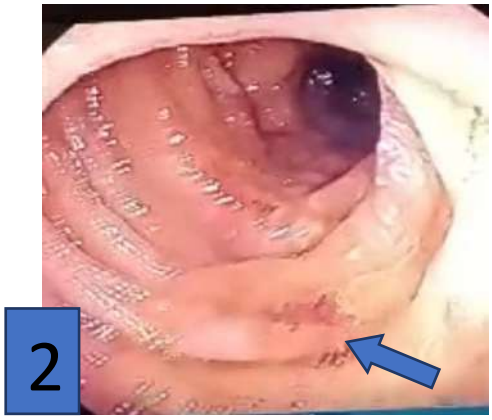


Figure 1. and Figure 2. Upper gastrointestinal endoscopic evaluation. A. Endoscopic findings included linear erosions extending proximally from the gastroesophageal junction, diffuse gastric mucosal edema and hyperemia, and deep and superficial linear ulcerations in the second portion of the duodenum. These findings were consistent with Los Angeles grade B esophagitis, pangastritis, and erosive duodenitis, corresponding to Zargar grade 2B corrosive injury

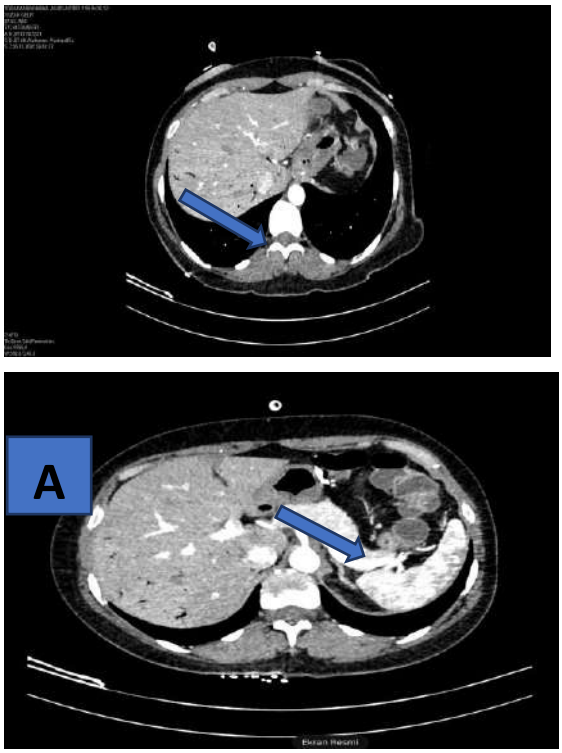


Figure 3. A and B Contrast-enhanced abdominal computed tomography. Hepatic portal vein gas is localized in the left lobe of the liver, and closer to the liver capsule (the arrows show the gas)

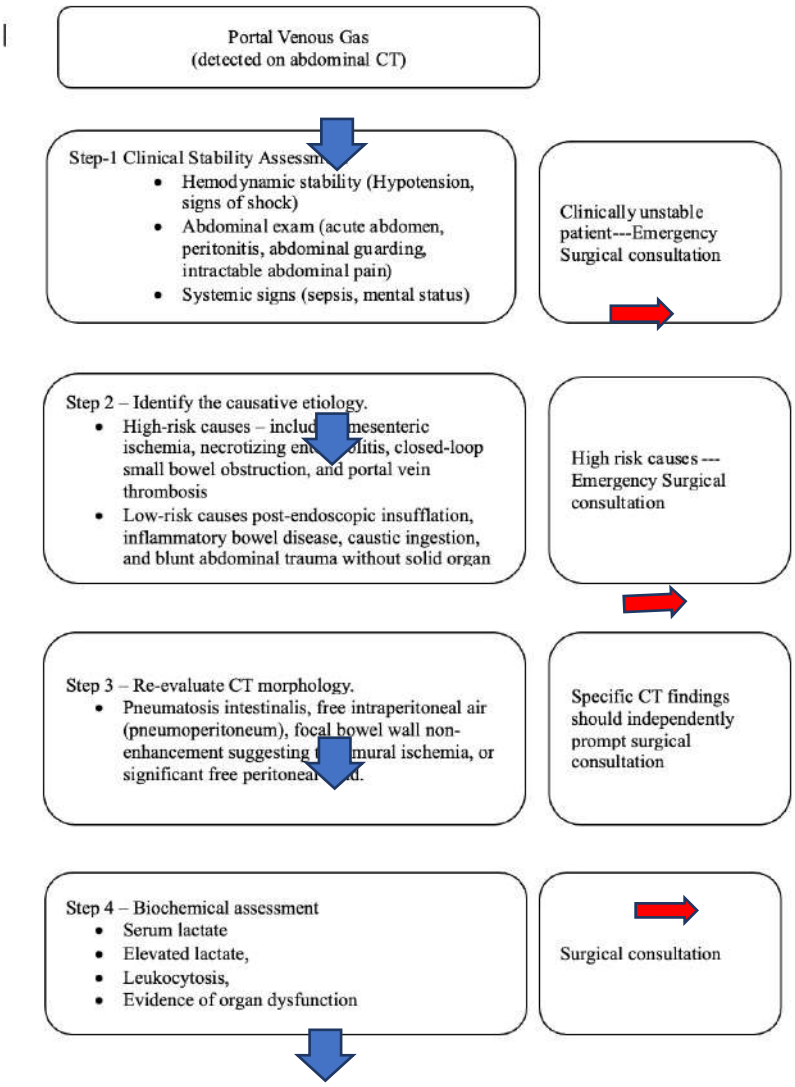


Figure 4. Proposed algorithmic approach to portal venous gas (PVG); stepwise clinical decision framework

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Sex-Based Differences in Carboxyhemoglobin Levels, Venous Blood Gas Parameters, and Hematologic Indices in Acute Carbon Monoxide Poisoning

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Abstract

Introduction: Carbon monoxide (CO) poisoning is one of the leading causes of unintentional poisoning-related deaths worldwide [1]. Although its pathophysiology is well-defined, how gender-related variability in clinical and laboratory findings affects diagnosis and prognosis has not been sufficiently investigated.

Objective: This study aimed to characterize gender-based differences in carboxyhemoglobin (COHb), venous blood gas parameters, and hematological biomarkers in patients diagnosed with acute CO poisoning.

Methods: This retrospective study included patients with isolated CO poisoning who presented to the emergency department of Malatya Training and Research Hospital between March 2025 and February 2026. Statistical analyses were performed using SPSS software. Means ± standard deviation (SD) were calculated for parametric variables, and median (IQR) values were calculated for non-parametric variables. A t-test was used to compare parametric variables between gender groups, and the Mann–Whitney U test was used for non-parametric variables. Relationships between non-parametric variables were assessed using Spearman's rank correlation analysis.

Results: A total of 32 patients were included; 17 were male and 15 were female. The mean age was 48 years. All patients were discharged alive. Carboxyhemoglobin and hemoglobin levels were significantly higher in males. In females, venous PO_2, platelet count, and lymphocyte count were higher compared to males. Moderately strong negative correlations were observed between age and albumin and lymphocyte levels, while a positive and significant relationship was found between lymphocyte and monocyte levels.

Conclusion: In acute CO poisoning, gender significantly influences key laboratory findings, particularly carboxyhemoglobin, venous PO_2, and hematological parameters. These findings highlight the need for the development of gender-specific cut-off values in diagnostic and prognostic assessments, while also providing a robust foundation for future prospective studies.

Keywords: carbon monoxide poisoning, gender, carboxyhemoglobin, blood gases, hematological parameters.

Introduction

CO is a colorless, odorless, and non-irritating gas produced by the incomplete combustion of carbon-containing fuels. Due to its approximately 240-fold higher affinity for hemoglobin compared to oxygen, it binds to hemoglobin, leading to functional anemia, tissue hypoxia, and multi-organ dysfunction [2]. This can result in serious clinical consequences even at low environmental concentrations [3]. Despite advanced supportive therapies and hyperbaric oxygen therapy, CO poisoning continues to cause preventable morbidity and mortality, particularly in low-resource settings and during winter months [4].

The clinical presentation is typically characterized by headache, dizziness, nausea, and confusion; however, a weak correlation is often observed between symptom severity and measured COHb levels [5,6]. This suggests that this biomarker alone has limited prognostic rather than diagnostic value. In this context, additional laboratory markers such as

inflammatory cell counts, acid-base balance, and metabolic parameters have emerged as complementary tools for risk stratification and treatment monitoring.

In this study, we observed gender-based differences in 32 CO poisoning patients: while COHb and hemoglobin levels were higher in males, venous PO_2 and platelet count were significantly higher in females. These observations highlight that biological sex may not merely be a confounding factor in the interpretation of basic hematological and blood gas parameters, but rather a variable that can add diagnostic and prognostic clarity. The findings reported in this analysis aim to contribute to the development of gender-specific reference ranges, improve risk modeling, and provide a solid foundation for future studies in acute CO poisoning.

Materials and Methods

This study was conducted by retrospectively reviewing the records of patients with CO poisoning who presented to the emergency department of Malatya Training and Research Hospital between March 2025 and February 2026. Data were obtained from the hospital's information management system.

Inclusion and Exclusion Criteria

Only patients over 18 years of age were included in the study. Patients with a COHb level ≥ 9% measured at the time of emergency department admission or within the first 6 hours were included. Patients with COHb levels < 9% and those with symptoms attributable to intracranial or thoracic pathologies were excluded from the study.

Parameters Evaluated

Differences based on gender were evaluated for the following parameters: age (years), albumin (g/L), C-reactive protein (CRP, mg/L), platelets (PLT, $\times 10^9$/L), hemoglobin (HGB, g/dL), neutrophils ($\times 10^9$/L), monocytes ($\times 10^9$/L), lymphocytes ($\times 10^9$/L), pH, pCO_2 (mmHg), pO_2 (mmHg), and carboxyhemoglobin (%).

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics 27 software. The normal distribution of data was assessed using the Shapiro-Wilk test. Variables with a parametric distribution were expressed as mean ± standard deviation, and non-parametric data were expressed as median (minimum–maximum). Relationships between parameters were evaluated using Spearman's correlation test. We used the classification proposed by Mukaka for interpreting correlation coefficients: <0.30 negligible, 0.30–0.49 weak, 0.50–0.69 moderate, 0.70–0.89 strong, and ≥0.90 very strong correlation. A p-value of <0.05 was considered statistically significant.

Results

Of the total 32 patients, 53.1% were male (n=17) and 46.9% were female (n=15). The mean age was 48.3±16.7 years. All patients were discharged alive (mortality rate 0%). Carboxyhemoglobin levels were significantly higher in males (29.1±10.2% vs. 24.6±8.7%, p=0.024); hemoglobin levels were also higher in males (14.7±1.9 g/dL vs. 12.8±2.2 g/dL, p=0.02). In females, venous PO_2 (p=0.005), platelet count (p=0.004), and lymphocyte count (p=0.06) were higher compared to males. Moderately strong negative correlations were observed between age and albumin (r=-0.574, p<0.001) and lymphocyte (r=-0.473, p=0.006) levels, while a positive and significant relationship was found between lymphocyte and monocyte levels (r=0.635, p<0.001).

Discussion

CO poisoning is one of the leading causes of unintentional poisoning-related deaths worldwide [1]. In this study, significant gender-related laboratory differences were identified in cases of acute carbon monoxide poisoning. The finding of higher carboxyhemoglobin and hemoglobin levels in males can be explained both by the greater total hemoglobin mass in males and by potential differences in exposure patterns. In contrast, the higher venous PO_2 and platelet levels observed in females suggest the influence of gender-related physiological and hormonal differences on tissue oxygenation and hematological response.

The trend towards higher lymphocyte levels in females supports the notion that immune responses may vary by gender; however, the failure to reach statistical significance may be related to the limited sample size. In the correlation analysis, the negative relationship between age and both albumin and lymphocyte levels is consistent with the literature indicating that nutritional status and immune response decline with aging [7]. Furthermore, the positive correlation between lymphocytes and monocytes suggests that these may be co-regulated components of the inflammatory response.

These findings demonstrate that in acute CO poisoning, gender is not merely a demographic variable but can also be an important determinant in the interpretation of laboratory parameters. However, considering the retrospective design and limited sample size of this study, these results need to be validated by larger, prospective studies.

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Pediatric Oropharyngeal Penetrating Injury Caused by a Kitchen Mixer Attachment: A Case Report

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Introduction

Oropharyngeal traumas in childhood constitute a significant portion of emergency department admissions. This group, standardized in the literature as "pencil injuries," typically occurs when children fall while running with a foreign object in their mouths(1). However, the spectrum of domestic accidents can take on a much more complicated clinical presentation with the involvement of complex-designed objects such as kitchen utensils(5). Due to its close proximity to vital vascular structures, cranial nerves, and the airway, the oropharyngeal region carries a risk of deep tissue damage even in seemingly superficial traumas(3,4). In this case report, we analyze the management process of a transfixed palate trauma in a 3-year-old patient caused by a spiral mixer attachment, which posed a high risk for removal due to its geometric structure, in light of current literature.

Case Presentation

A 3-year-old female patient was brought to our emergency department following a penetrating trauma that occurred when she put a spiral metal attachment of a kitchen mixer into her mouth while playing at home. Upon initial assessment, the patient's vitals were stable, oxygen saturation was 100%, she was conscious, and her general condition was good. Laboratory investigations, including hemogram, biochemistry, and coagulation parameters, were found to be completely within normal limits.

Figure 1 Physical Examination Image

Clinical examination revealed that the metal spiral attachment entered through the inner aspect of the right cheek mucosa, passed behind the uvula, and re-directed into the mouth by penetrating through the soft palate (transfixed) (Figure 1). Following an ENT (Ear, Nose, and Throat) consultation, it was noted that facial nerve functions were normal, and no ecchymosis, crepitus, or active bleeding was detected in the cervical region. Radiological examinations confirmed the trajectory of the metallic foreign body (Figure 2); no additional pathology was detected regarding vascular proximity or bone integrity.

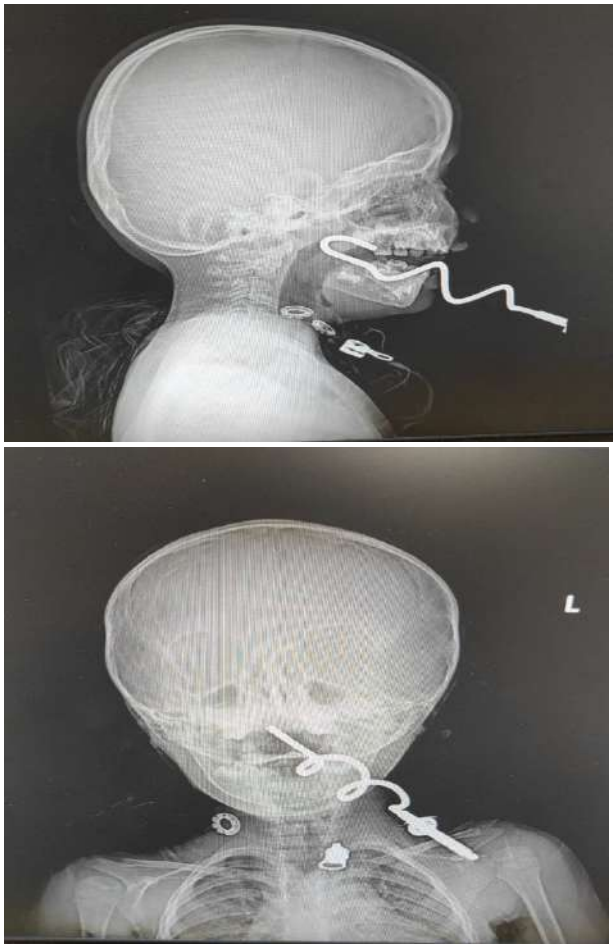

Figure 2 Radiological Imaging

The patient was taken to minor surgery under sterile conditions under general anesthesia/deep sedation. The most critical stage of the procedure was breaking the "screw" effect created by the spiral structure within the tissue. Following the spiral geometry, the object was removed in one piece using rotational maneuvers toward the nasopharynx without widening the tissue tear. The 0.5 cm perforation area in the palate was sutured with 4/0 Vicryl. The patient, who developed no postoperative complications, was discharged from the emergency department with the necessary recommendations.

Discussion

In large series studies on pediatric oropharyngeal injuries (Zonfrillo et al.), whether the injury is midline or lateral is the most important prognostic criterion. Lateral wall injuries may necessitate absolute CT angiography due to the risk of internal carotid artery dissection and late-term thrombosis(3,4). In our case, the predominantly midline location of the injury lowered the vascular risk profile. However, attention should be drawn to the concept of the "lucid interval" highlighted in the literature; patients who are initially stable in oropharyngeal

traumas may return hours later with retropharyngeal abscesses or vascular complications. yaş grubunda ciddi maksillofasiyal travma kaynağı olabileceğine dair uyarıları desteklemektedir[5].

The primary challenge distinguishing this case from similar ones in the literature is the geometry of the object. While a pulling maneuver may be sufficient for straight objects (lollipop sticks, etc.), the literature (Hengerer et al.) suggests the "atraumatic vectorial removal" method for spiral objects(2). In our case, we preserved tissue integrity by rotating the object on its own axis. Furthermore, pediatric studies on home safety support warnings that kitchen appliances (mixers, blenders, etc.) can be a source of serious maxillofacial trauma in this age group(5).

Conclusion

This case demonstrates that in pediatric penetrating traumas, not only the depth but also the geometric form of the object determines the surgical strategy. Normal laboratory and radiological findings should not mislead the physician; each case must be evaluated within its own anatomical trajectory. Keeping kitchen utensils out of children's reach is the most fundamental public health intervention to prevent such avoidable traumas.

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Keywords: Pediatric trauma, Oropharyngeal injury, Foreign body, Domestic accidents.

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THE EFFECT OF ELECTROLYTE DISORDERS ON MORTALITY AND HOSPITAL STAY IN PATIENTS PRESENTING TO THE EMERGENCY DEPARTMENT WITH ACUTE EXACERBATION OF COPD

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Abstract

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a condition characterised by chronic respiratory symptoms and abnormalities in the airways (bronchitis, bronchiolitis, alveolar emphysema), which frequently leads to progressive airway obstruction. COPD exacerbations constitute the leading cause of mortality and hospital admissions among COPD patients. This study investigated the effects of electrolyte disturbances on mortality and length of hospital stay in patients with COPD exacerbations.

Materials and Methods: This prospective study included patients who presented to the Emergency Medicine Clinic and were admitted to hospital due to an acute exacerbation of COPD. Following the application of exclusion criteria, 328 patients formed the study sample. The patients' age, gender, vital signs (temperature, pulse, blood pressure, oxygen saturation), comorbid conditions, haematological parameters (complete blood count, biochemistry, CRP, blood gas, lactate, etc.), inflammation parameters and radiological imaging were examined. Biochemical parameters including glucose, BUN, creatinine, uric acid, albumin and electrolyte levels were recorded. Patients' admission status to the ward or intensive care unit and length of stay were recorded. Mortality rates among hospitalised patients were analysed.

Results: A total of 328 patients were included in the study. Of the patients, who had a mean age of 71.5 ± 9.8 years, 91 were women and 237 were men. The patients were divided into two groups according to mortality status: those with mortality and those without. There were 21 (6.4%) patients with mortality. The mean age of patients with mortality was higher, and their intensive care and total hospitalisation durations were longer, whilst their ward stay duration was shorter. In terms of laboratory values, the haemoglobin and albumin levels were lower in the group with mortality, whilst glucose, urea and phosphorus levels were higher. When patients were divided into three groups based on electrolyte levels (low, normal and high) and evaluated, significant differences in mortality were observed between the groups for sodium, potassium, chloride and phosphorus levels, whereas no significant differences were found for magnesium and calcium levels. When patients were divided into three groups based on low, normal and high electrolyte levels and assessed in terms of length of hospital stay, a significant difference was found between the groups for chloride and phosphorus levels,

whereas no significant difference was found for sodium, potassium, calcium and magnesium levels.

Conclusion: In patients with acute exacerbations of COPD, electrolyte imbalances influence the length of hospital stay and mortality rates. We observed that hyperphosphataemia and hypochloraemia prolong the length of hospital stay, whilst hypernatraemia, hypokalaemia, hypochloraemia and hyperphosphataemia also increase the risk of mortality. Consequently, correction of these electrolyte imbalances is necessary to reduce the length of hospital stay and the risk of mortality.

Keywords: Electrolyte imbalances, hospitalization duration, COPD, mortality

Introduction

COPD is a disease that can result from the inhalation of toxic gases harmful to the lungs, is characterised by chronic respiratory symptoms, and typically leads to progressive airway obstruction (1). COPD is one of the leading causes of mortality in both developed and developing countries (2). According to data from the World Health Organisation, it caused 3.5 million deaths in 2021 (3). Considering factors such as changes in the age distribution of the global population and the rise in tobacco use in the coming years, it is anticipated that the prevalence of COPD will continue to increase (4). The fundamental pathogenesis of the disease is irreversible airflow limitation, particularly during exhalation (1). Advanced age, genetic factors, cigarette smoke, occupational exposures, low socioeconomic status and childhood respiratory infections are among the risk factors for COPD (5). In individuals with a history of exposure to COPD risk factors, a diagnosis of COPD should be considered and a spirometry test performed if they present with symptoms such as shortness of breath, chronic cough and/or sputum production (6). Electrolyte imbalances are commonly observed in patients with COPD exacerbations due to the medical treatments administered, acid-base disturbances and associated comorbidities. Electrolyte imbalances can exacerbate the severity of exacerbations by impairing respiratory function and causing weakness in the respiratory muscles, and may influence length of hospital stay and mortality (7,8)

Materials and Methods

The study commenced following the granting of ethical approval by the Ethics Committee for Non-Interventional Research at Elazığ Fethi Sekin City Hospital, dated 21 November 2024 and bearing session number 2024/4-6. This study was conducted as a prospective study. The study included patients who applied to the Department of Respiratory Medicine between 1 December 2024 and 31 May 2025 and admitted to the Respiratory Medicine Ward, Respiratory Intensive Care Unit, or Anaesthesia and Resuscitation Intensive Care Units due to an acute exacerbation of COPD. For this prospective study, 328 patients constituted the sample size within the specified period. The patients' age, gender, vital signs (temperature,

pulse, blood pressure, oxygen saturation), comorbid conditions, haematological parameters (complete blood count, biochemistry, CRP, blood gas, lactate levels, etc.), inflammation markers and radiological imaging were examined. The patients' admission status to the ward and intensive care unit, as well as their length of stay, were recorded. The mortality rates of hospitalised patients were examined. The study investigated whether electrolyte imbalances in hospitalised patients had an effect on mortality and length of hospital stay.

Statistical Analysis

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) for Windows 22.0 (IBM SPSS Inc., Chicago, IL). The normality of the data was assessed using the Kolmogorov-Smirnov test. Numerical variables exhibiting a normal distribution are presented as mean ± standard deviation, whilst those not exhibiting a normal distribution are presented as median (minimum–maximum). Categorical variables are presented as counts and percentages. For comparisons between two groups, the Student's t-test was used for numeric variables exhibiting a normal distribution, and the Mann-Whitney U test was used for numeric variables not exhibiting a normal distribution. In comparisons involving more than two groups, the one-way analysis of variance (ANOVA) was used for numerical variables that followed a normal distribution, and the Kruskal-Wallis test was used for those that did not. For comparisons of categorical data, the chi-square test and Fisher's exact chi-square test were employed. In the statistical analyses, a p-value of <0.05 was considered statistically significant.

Results

A total of 328 patients were included in our study. The mean age of the patients was 71.5 ± 9.8 years. When the patients were divided into two groups according to mortality status, 21 (6.4%) had died. The group with mortality was older than the group without mortality. No statistically significant differences were observed between the two groups in terms of gender and comorbidities (hypertension, coronary artery disease, diabetes mellitus, chronic kidney disease, malignancy, stroke) (Table 1).

Table 1. Demographic characteristics of patients

	Total (n=328)	No mortality (n=307)	Mortality (n=21)	p
Gender				0,273
Female, n (%)	91 (27,7)	83 (27)	8 (38,1)	
Male, n (%)	237 (72,3)	224 (224)	13 (61,9)	
Age ± SD	71,5 ± 9,8	71 ± 9,8	78 ± 8,2	**0,002**

Hypertension				0,227
None, n (%)	151 (46)	144 (46,9)	7 (33,3)	
Present, n (%)	177 (54)	163 (53,1)	14 (66,7)	
Coronary Artery Disease				0,250
None, n (%)	179 (54,6)	165 (53,7)	14 (66,7)	
Present, n (%)	149 (45,4)	142 (46,3)	7 (33,3)	
Diabetes Mellitus				0,586
None, n (%)	256 (78)	238 (77,5)	18 (85,7)	
Present, n (%)	72 (22)	69 (22,5)	3 (14,3)	
Chronic Kidney Disease				0,087
None, n (%)	320 (97,6)	301 (98)	19 (90,5)	
Present, n (%)	8 (2,4)	6 (2)	2 (9,5)	
Malignancy				0,634
None, n (%)	307 (93,6)	288 (93,8)	19 (90,5)	
Present, n (%)	21 (6,4)	19 (6,2)	2 (9,5)	
Stroke				0,966
None, n (%)	313 (95,4)	293 (95,4)	20 (95,2)	
Present, n (%)	15 (4,6)	14 (4,6)	1 (4,8)	

It was observed that the group with mortality had a shorter length of stay on the ward but a longer duration of intensive care and total hospital stay compared to the group without mortality. It was found that the group with mortality had lower blood pressure (systolic and diastolic) and oxygen saturation levels, and higher body temperature and pulse rates (Table 2).

Table 2. Comparison of patients' lengths of stay and vital signs

	Total (n=328)	No mortality (n=307)	Mortality (n=21)	p
Number of days spent in the ward, median (min–max)	8 (0-51)	8 (0-36)	0 (0-51)	**<0,001**
Number of days spent in intensive care, median (min–max)	0 (0-127)	0 (0-127)	10 (0-115)	**<0,001**
Total length of stay, median (min-max)	11 (1-127)	10 (1-127)	16 (2-123)	**0,025**
Systolic blood pressure, median (min-max)	130 (90-200)	135 (90-200)	110 (95-160)	**<0,001**
Diastolic blood pressure, median (min-max)	80 (50-130)	80 (50-120)	65 (50-130)	**<0,001**
Body temperature, median (min-max)	36,4 (36-38,6)	36,4 (36-38,6)	36,7 (36-37,9)	**0,046**

Pulse rate, median (min-max)	96 (42-150)	95 (42-147)	119 (71-150)	**<0,001**
SO2 ± SD	70,8 ± 9,7	71,2 ± 9,6	64,5 ± 9,2	**0,002**

SO2: Saturation

When patients were divided into three groups—low, normal and high blood sodium levels—and analysed, a significant difference in mortality was observed between the groups. In pairwise comparisons, it was observed that the length of hospital stay was longer in the normonatraemia group compared to the hypernatraemia group. It was found that the length of intensive care unit stay was longer in the hyponatraemia and hypernatraemia groups compared to the normonatraemia group (Table 3).

Table 3. Comparison of groups by serum sodium level

	Hyponatraemia (n=38)	Eunatraemia (n=284)	Hypernatraemia (n=6)	p
Mortality None, n (%) Present, n (%)	 33 (86,8) 5 (13,2)	 270 (95,1)# 14 (4,9)#	 4 (66,7)# 2 (33,3)#	**0,004**
Length of stay in the ward, median (min–max)	7 (0-28)	8 (0-51) #	0 (0-8) #	**0,001**
Length of stay in intensive care, median (min–max)	5 (0-127) #	0 (0-115) #^	15 (0-70)^	**<0,001**
Total length of stay, median (min–max)	11(3-127)	10 (1-123)	15 (8-70)	0,293

#^ It shows which groups in the current row show a significant difference.

When patients' serum potassium levels were assessed, a significant difference in mortality was observed between the groups (hypokalemia, normokalemia, hyperkalemia). In other comparisons, no significant differences were found between the groups in terms of the number of days spent in the ward, in intensive care, or the total length of stay (Table 4).

Table 4. Comparison of groups according to serum potassium levels

	Hypokalaemia (n=11)	Normokalaemia (n=301)	Hyperkalaemia (n=16)	p
Mortality None, n (%) Present, n (%)	 8 (72,7) # 3 (27,3) #	 285 (94,7) # 16 (5,3) #	 14 (87,5) 2 (12,5)	**0,008**
Length of stay in the ward,	8,1 ± 9,2	9,1 ± 7,1	6,2 ± 7	0,277

median (min–max)				
Length of stay in intensive care, median (min–max)	9,5 ± 14,9	4,4 ± 12,4	8,1 ± 4,5	0,214
Total length of stay, median (min–max)	17,6 ± 14	13,5 ± 14	14,3 ± 8,4	0,601

[#] It shows which groups in the current row show a significant difference.

When patients' serum calcium levels were assessed, no significant difference was observed between the groups (hypocalcaemia, normocalcaemia, hypercalcaemia) in terms of mortality and total length of hospital stay (Table 5).

Table 5. Comparison of groups according to serum calcium levels

	Hypocalcaemia (n=121)	Normocalcaemia (n=190)	Hypercalcaemia (n=17)	p
Mortality None, n (%) Present, n (%)	 109 (90,1) 12 (9,9)	 181 (95,3) 9 (4,7)	 17 (100) 0 (0)	0,103
Length of stay in the ward, median (min–max)	7 (0-31) [#]	8 (0-51)	11 (1-36) [#]	**0,039**
Length of stay in intensive care, median (min–max)*	0 (0-127) [#] (6,6 ± 16,4)	0 (0-72) [#] (3,8 ± 9,2)	0 (0-15) (1,9 ± 4)	**0,031**
Total length of stay, median (min–max)	11 (2-127)	10 (1-123)	11 (5-39)	0,236

[#] This shows which groups in the current row show a significant difference. *As the median values for the patients were the same, the mean values are also given in brackets.

When patients' blood chloride levels were assessed, a significant difference in mortality was observed between the groups (hypochloraemia, normochloraemia, hyperchloraemia). In pairwise comparisons based on total length of stay, the total length of stay in the hypochloraemia group was found to be longer than that in the normochloraemia group (Table 6).

Table 6. Comparison of groups according to blood chloride levels

	Hypochloremia (n=72)	Normochloremia (n=228)	Hyperchloremia (n=28)	p
Mortality None, n (%) Present, n (%)	 63 (87,5) [#] 9 (12,5) [#]	 218 (95,6) [#] 10 (4,4) [#]	 26 (92,9) 2 (7,1)	**0,049**
Length of stay in the ward, median (min–max)	7 (0-36)	8 (0-51)	8 (0-26)	0,401
Length of stay in intensive	6 (0-127) [#^]	0 (0-115) [#]	0 (0-11) ^	**<0,001**

care, median (min–max)				
Total length of stay, median (min–max)	12 (3-127) #	10 (1-123) #	10 (2-26)	**0.025**

#^ It shows which groups in the current row show a significant difference.

When patients' serum magnesium levels were assessed, no significant difference was observed between the groups (hypomagnesaemia, normomagnesaemia, hypermagnesaemia) in terms of mortality and total length of hospital stay (Table 7).

Table 7. Comparison of groups according to serum magnesium levels

	Hypomagnesaemia (n=51)	Normomagnesaemia (n=261)	Hypermagnesaemia (n=16)	p
Mortality None, n (%) Present, n (%)	 45 (88,2) 6 (11,8)	 248 (95) 13 (5)	 14 (87,5) 2 (12,5)	0,115
Length of stay in the ward, median (min–max)	8 (0-51)	8 (0-36)	6 (0-20)	0,142
Length of stay in intensive care, median (min–max)	0 (0-72)	0 (0-115) #	7 (0-127) #	**0,002**
Total length of stay, median (min–max)	11 (3-123)	10 (1-117)	12 (3-127)	0,544

It shows which groups in the current row show a significant difference.

When patients' serum phosphorus levels were assessed, a significant difference in mortality was observed between the groups (hypophosphataemia, normophosphataemia and hyperphosphataemia). In pairwise comparisons based on the total number of hospital days, it was found that the total number of hospital days in the hyperphosphataemia group was higher than that in the normophosphataemia group (Table 8).

Table 8. Comparison of groups according to serum phosphorus levels

	Hypophosphataemia (n=22)	Normophosphataemia (n=270)	Hyperphosphataemia (n=36)	p
Mortality None, n (%) Present, n(%)	 22 (100) 0 (0)	 256 (94,8) # 14 (5,2) #	 29 (80,6) # 7 (19,4) #	**0,002**
Length of stay in the ward, median (min–max)	10 (0-33)	8 (0-51)	6 (0-36)	0,144
Length of stay in intensive care,	0 (0-23) #	0 (0-72) ^	9 (0-127) #^	**<0,001**

median (min–max)				
Total length of stay, median (min–max)	11 (2-36)	10 (1-123) #	14 (3-127) #	**0,026**

#^ It shows which groups in the current row show a significant difference.

Discussion

Investigating the risk factors that influence the prognosis of COPD exacerbations is crucial for reducing mortality and hospital admission rates. Factors influencing mortality in patients with COPD exacerbations include age, gender, smoking status, body mass index, electrolyte levels, liver and kidney function, and associated comorbidities (3). Electrolyte imbalances are commonly observed in COPD patients. The causes of this condition include comorbidities, associated polypharmacy, and mixed acid-base disorders (7). Electrolyte disturbances observed during the acute exacerbation phase of COPD can worsen airway function and lead to weakness in the respiratory muscles. Consequently, electrolyte imbalances observed during this period may exacerbate exacerbations (7,8). With this study, we aimed to contribute to the literature by investigating the effects of electrolyte imbalances on mortality and length of hospital stay in patients presenting to the emergency department and subsequently hospitalised due to acute COPD exacerbations.

Electrolyte imbalances are common in hospitalised patients. Hyponatraemia, the most common electrolyte imbalance in hospitalised patients, is also a predictor of poor prognosis in COPD exacerbations. A study by Garcia-Sanz et al. demonstrated that, among patients hospitalised for COPD exacerbations, those with hyponatraemia had longer hospital stays (9). Fan and colleagues investigated the effects of serum sodium levels on long-term mortality in COPD patients in intensive care. This study found that both hyponatraemia and hypernatraemia were significantly associated with mortality (10). In our study, consistent with the literature, we also found that dysnatraemia was associated with mortality. When evaluating the length of hospital stay in our study, we found that the normonatraemic group had the longest length of stay, whilst the hypernatraemic group had the shortest.

In their study investigating the effects of serum electrolytes on mortality in patients hospitalised due to COPD exacerbations, Ogan et al. found that hypokalaemia and hypocalcaemia were significantly associated with mortality in patients with COPD exacerbations, whereas serum magnesium and phosphorus levels were not associated with mortality (7). There are few studies in the literature regarding the effects of potassium abnormalities on the length of hospital stay in patients with COPD exacerbations. In a study conducted by Bagde et al. in 2025, it was demonstrated that hypokalaemia and hyponatraemia affected the length of hospital stay and altered mortality in patients with COPD exacerbations (11). Consistent with the literature, our study also demonstrated that hypokalaemia is a risk factor for mortality in patients hospitalised for COPD exacerbations; however, in our study,

no significant association was found between potassium abnormalities and the number of days spent in the ward, intensive care unit, or total hospital stay.

Calcium is the most abundant mineral in the human body and plays a role in numerous physiological functions. Imbalances in calcium homeostasis, however, have been linked to many diseases. Nevertheless, there are few studies in the literature examining the relationship between serum calcium levels and COPD exacerbations. A study by Wan et al. demonstrated that hypercalcaemia is associated with an increased risk of COPD incidence and mortality (12). A retrospective study by Ogan et al. found that hypokalaemia and hypocalcaemia during COPD exacerbations were associated with mortality (7). In contrast, Deep et al.'s study found no significant association between serum calcium and mortality during COPD exacerbations (8). As can be seen, the relationship between serum calcium and mortality during COPD exacerbations remains inconclusive in the literature. There are studies in the literature indicating that both hypocalcaemia and hypercalcaemia are associated with mortality (7,12). In our study, however, no significant association was found between serum calcium and mortality. We believe that longer-term, multicentre, and prospective studies are required to understand the effects of serum calcium on mortality during COPD exacerbations.

Chloride is the most abundant anion outside the cell and is one of the key anions required to maintain the balance between the intracellular and extracellular environments. However, the role of serum chloride in mortality has not been extensively investigated (13). In a study by Sun et al., a strong association was found between in-hospital mortality and serum chloride levels in patients with COPD exacerbations (14). Huang and colleagues, who investigated the role of serum chloride in the prognosis of COPD, demonstrated that low serum chloride levels are independently associated with COPD exacerbations (15). In our study, consistent with the literature, we found that hypochloraemia is a significant risk factor for mortality. Furthermore, similar to the literature, our study found that hypochloraemia is a significant factor influencing the length of stay in intensive care and the total number of hospital days.

Magnesium is an important cation that induces relaxation in bronchial smooth muscle cells. Causes of low magnesium levels in COPD patients include the use of corticosteroids, low dietary intake and the use of beta-2 agonists. A study by Kshirsagar and Patil demonstrated that low magnesium levels are a risk factor for COPD exacerbations (16). In studies by Makwana et al. investigating the correlation between serum magnesium levels and COPD exacerbations, it was shown that patients with hypomagnesaemic exacerbations had a statistically significant longer hospital stay (17). In our study, however, consistent with the literature, no statistically significant relationship was observed between magnesium levels and mortality during exacerbation periods. When the total number of hospital days was evaluated in our study, unlike in the literature, no statistically significant difference was observed between the groups. We believe this stems from the fact that the exclusion criteria in our study differed from those in the study by Makwana et al.

It is known that serum phosphate plays a role in various physiological functions, such as energy metabolism, bone mineralisation and intracellular signalling. Disorders of phosphorus metabolism are particularly common in critically ill patients admitted to intensive care. A study by Du et al. demonstrated that abnormal serum phosphate levels play a significant role in mortality among critically ill COPD patients (18). In the literature, there are few studies examining the effects of serum phosphorus levels on the duration of hospitalisation in patients with COPD exacerbations. Farah and colleagues investigated the effects of hypophosphataemia on disease prognosis in COPD patients. In this study, it was found that hypophosphataemia in patients with COPD exacerbations led to an increase in length of hospital stay, the need for ventilation, and the duration of intensive care unit (ICU) stay (19). In our study, consistent with the literature, hyperphosphataemia was found to be associated with mortality in patients hospitalised for COPD exacerbations. However, when total length of stay and length of stay in intensive care were evaluated in our study, it was observed that, unlike in the literature, the length of stay in intensive care and total length of hospitalisation were longer in the hyperphosphataemia group.

Conclusion

Electrolyte imbalances may occur in patients with COPD exacerbations due to associated comorbidities, acid-base disturbances and the medical treatments administered. Electrolyte imbalances can impair lung function and lead to weakness in the respiratory muscles, thereby increasing the severity of the exacerbation. In patients with acute COPD exacerbations, electrolyte imbalances affect the duration of hospitalisation and mortality. We observed that hyperphosphataemia and hypochloraemia prolong the length of hospital stay, whilst hypernatraemia, hypokalaemia, hypochloraemia and hyperphosphataemia also increase the risk of mortality. Consequently, correction of these electrolyte imbalances is necessary to reduce the length of hospital stay and the risk of mortality.

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Relationship Between Hemogram and Biochemical Parameters and Hospital Length of Stay in Lung Cancer Patients Evaluated in the Emergency Department

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Introductıon

Lung cancer is one of the leading causes of cancer-related mortality worldwide and ranks among the most common malignancies in both men and women. It is particularly prevalent in the elderly population, with most patients being diagnosed at an advanced stage (1). According to global cancer statistics, lung cancer continues to be a major contributor to cancer-related deaths (2). Consequently, a significant proportion of patients present to emergency departments due to acute exacerbations of symptoms or complications associated with the disease.

Systemic inflammation has been shown to be closely associated with disease progression and prognosis in patients with lung cancer (3). Therefore, routine laboratory parameters, including hemogram and biochemical markers, play an important role in clinical assessment. Hematological parameters, particularly neutrophils, lymphocytes, and platelets, have been demonstrated to be related to inflammation. Additionally, several studies have reported that biochemical markers such as C-reactive protein and albumin may also be associated with disease prognosis (4).
Furthermore, parameters such as neutrophils and lymphocytes are considered indirect indicators of the inflammatory response occurring within the tumor microenvironment.

The evaluation of clinical characteristics and laboratory parameters in lung cancer patients presenting to the emergency department can provide valuable information for predicting clinical outcomes such as disease course and hospital length of stay. However, studies examining the relationship between hematological and biochemical parameters obtained during emergency department visits and clinical outcomes in this patient population are limited (5).

Materıals And Methods

This study was retrospectively conducted on patients diagnosed with lung cancer who presented to our acute emergency department within the past month. Patients under 18 years of age and those with incomplete data were excluded from the study.

Statıstıcal Analysıs

Statistical analysis of the data obtained in this study was performed using IBM SPSS Statistics version 27 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro–Wilk test. Categorical data were presented as

frequencies and percentages. For continuous variables, those with a parametric distribution were expressed as mean ± standard deviation, while non-parametric variables were presented as interquartile range (IQR) or minimum–maximum values. The independent samples t-test was used for parametric data, and the Mann–Whitney U test was applied for non-parametric data. Correlation analyses were conducted for quantitative variables. A p-value of <0.05 was considered statistically significant.

Results

Of the patients, 63.6% were male and 36.4% were female. The mean age was 67.4 ± 12.1 years. The patients' biochemical and hematological parameters were as follows: amylase 56.8 ± 13.1, hemoglobin 13.1 ± 1.9, neutrophils 5.3 (3.1–7.05), lymphocytes 1.208–2.198, platelets 195–319, C-reactive protein (CRP) 5.95–63.35, albumin 33–38, and HALP score 182–566. There was no statistically significant difference between gender and age (p = 0.305). Similarly, no significant differences were observed between gender and the following laboratory parameters: amylase (p = 0.748), hemoglobin (p = 0.829), neutrophils (p = 0.562), lymphocytes (p = 0.681), monocytes (p = 0.784), platelets (p = 0.733), CRP (p = 0.733), albumin (p = 0.890), hospital length of stay (p = 0.274), or HALP score (p = 0.733). A moderate negative correlation was found between neutrophil count and hospital length of stay (r = -0.497, p = 0.019).

Dıscussıon

Lung cancer is one of the leading causes of cancer-related mortality worldwide and is particularly more prevalent in the elderly population. A significant proportion of patients are diagnosed at an advanced stage and may present to emergency departments due to exacerbation of symptoms or disease-related complications (6). Therefore, the evaluation of demographic and laboratory characteristics of lung cancer patients presenting to the emergency department can provide valuable insights into the disease course.

In our study, the mean age of the patients was 67.4 ± 12.1 years. The literature reports that lung cancer is most commonly observed in the elderly population (7). One of the main reasons for this is prolonged exposure to carcinogens. In particular, the cumulative effect of long-term smoking and the increase in DNA damage with age contribute to an elevated risk of lung cancer. Therefore, we consider that the incidence of lung cancer is higher in older age groups.

In our study, 63.6% of the patients were male. The literature indicates that lung cancer has historically been more common in men (8). The primary reason for this is the historically higher prevalence of smoking among males. Smoking is considered the most important risk factor for lung cancer and is thought to be responsible for a large proportion of cases.

Systemic inflammation is known to be closely associated with disease progression and prognosis in patients with lung cancer. Inflammatory processes play a key role in the

formation of the tumor microenvironment and can influence tumor growth and metastasis development (9). Therefore, in recent years, hematological parameters obtained from the complete blood count have been investigated as potential biomarkers for assessing the inflammatory response.

In our study, no significant differences were observed between gender and biochemical or hematological parameters. The literature reports that inflammatory markers in lung cancer patients are generally associated with disease stage, tumor burden, and systemic inflammation, rather than gender (10). Therefore, the absence of a significant relationship between gender and laboratory parameters in our study can be considered consistent with the existing literature.

In our study, a moderate negative correlation was observed between hospital length of stay and neutrophil levels. Neutrophils are key components of the inflammatory response and are considered important indicators of tumor-associated inflammation. The literature reports that parameters such as neutrophils and lymphocytes reflect the inflammatory response within the tumor microenvironment and may be associated with disease prognosis (11). Therefore, evaluating the relationship between neutrophil levels and clinical outcomes can provide valuable insights into disease progression.

Conclusıon

In conclusion, this study evaluated the demographic characteristics and laboratory parameters of lung cancer patients presenting to the emergency department. No significant differences were observed between genders for most hematological and biochemical parameters, and no statistically significant relationships were found between gender, age, and laboratory parameters.

However, a moderate negative correlation was identified between hospital length of stay and neutrophil levels, suggesting that neutrophil counts, as reflections of the inflammatory response, may be associated with clinical outcomes. Additionally, the HALP score, calculated from complete blood count parameters, may serve as a potential biomarker for evaluating inflammatory processes and clinical outcomes.

Nevertheless, due to the limited sample size, further studies with larger patient populations are needed to validate and more clearly interpret these findings.

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The Diagnostic Challenge of Metabolic Disturbances: Thiazide-Induced Hyponatremia as a Stroke Mimic

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Introduction

Acute ischemic stroke is a leading cause of global disability and mortality, necessitating rapid diagnostic and therapeutic interventions within a narrow clinical window (1). The "Stroke Code" system is designed to expedite the administration of reperfusion therapies, yet this time-sensitive environment inherently increases the risk of misidentifying non-vascular conditions that present with similar neurological deficits. These conditions, categorized as stroke mimics, account for approximately 22% to 45.75% of all stroke-related emergency evaluations (2). Among the various etiologies of stroke mimics, metabolic disturbances represent a critical subgroup, with hyponatremia being the most prevalent electrolyte abnormality encountered in acute care settings (3).

Hyponatremia is defined as a serum sodium concentration below 135 mmol/L and is frequently observed in the geriatric population due to age-related changes in water homeostasis and a high burden of comorbidities (4). Thiazide diuretics, a cornerstone of hypertension management, are the most common pharmacological cause of drug-induced hyponatremia (5). Thiazide-induced hyponatremia (TIH) can range from mild, asymptomatic states to severe encephalopathy characterized by seizures, coma, and, notably, focal neurological deficits (6). Focal manifestations such as hemiparesis, aphasia, and facial asymmetry can precisely mirror the clinical presentation of a large vessel occlusion, leading to diagnostic confusion during initial stroke assessments (7).

The pathophysiology of neurological symptoms in hyponatremia involves an osmotic gradient that drives water into the intracellular compartment of the brain, resulting in cerebral edema (8). While this process typically results in generalized central nervous system dysfunction, FNDs are reported in up to 18% of patients with severe hyponatremic encephalopathy (1).

Case Presentation

A 62-year-old female presented to the emergency department via "Stroke Code" with sudden onset right-sided hemiparesis and motor aphasia. Her medical history included type 2 diabetes and hypertension treated with thiazide diuretics. Clinical evaluation revealed a GCS of 12 and an NIHSS score of 14, featuring 2/5 motor strength and a positive right Babinski sign.

Vital signs and blood glucose (105 mg/dL) were within normal limits. Noncontrast Brain CT, Brain Diffusion MRI, and CT angiography ruled out acute hemorrhage and large vessel occlusion. However, laboratory results identified severe hypotonic hyponatremia (Sodium: 114 mmol/L, Serum Osmolality: 240 mOsm/kg). Thrombolytic therapy was withheld, and a controlled 3% hypertonic saline infusion was initiated. Within four hours, sodium levels reached 120 mmol/L, leading to the complete resolution of neurological deficits (NIHSS 0, 5/5 motor strength). The presentation was attributed to thiazide-induced hyponatremia exacerbated by excessive water intake.

Discussion

The patient, a 62-year-old female, presented with an NIHSS score of 14, exhibiting acute right-sided weakness and motor aphasia. While the presentation mirrored a large vessel occlusion, laboratory evaluation revealed a serum sodium of 114 mmol/L, and imaging ruled out acute ischemia, confirming a metabolic stroke mimic.

Severe hyponatremia (120 mmol/L) frequently manifests as confusion or seizures, but focal neurological deficits (FNDs) occur in up to 18% of cases (9). Aphasia is the most frequent focal symptom, seen in 37.6% of hyponatremic mimics (10). These deficits stem from regional cerebral edema and localized neuronal dysfunction (11). Thiazide-induced hyponatremia (TIH) results from NCC inhibition in the distal tubule, which impairs the kidney's ability to excrete free water while stimulating ADH secretion (6, 12). Risk factors include advanced age, female gender, and SSRI co-administration (6, 13).

Management focuses on controlled sodium correction using hypertonic (3%) saline boluses (8). For this patient, raising sodium to 120 mmol/L led to a complete resolution of her NIHSS score within four hours (11). To prevent osmotic demyelination syndrome (ODS), correction should not exceed 8 mmol/L per day (8). Although IV thrombolysis (IVT) is relatively safe in stroke mimics—with SICH rates between 0.4 % and 1.0 %—rapid electrolyte screening is essential to avoid unnecessary medication (14). Long-term prevention requires discontinuing thiazides to prevent recurrence (7).

Conclusion

This case highlights that severe hyponatremia can precisely mirror an acute ischemic stroke by presenting with significant focal neurological deficits, such as hemiparesis and aphasia. In this patient, the combination of thiazide diuretic use and excessive water intake led to a critical drop in sodium levels, resulting in an NIHSS score of 14.

The rapid resolution of symptoms following controlled hypertonic saline infusion underscores the importance of early metabolic screening within the "Stroke Code" protocol. While time-

sensitive reperfusion therapies are vital for true strokes, identifying "stroke mimics" caused by electrolyte imbalances is essential to avoid unnecessary interventions. Clinicians should maintain a high index of suspicion for metabolic disturbances in geriatric patients on diuretics to ensure prompt, targeted treatment and prevent potential complications like osmotic demyelination syndrome.

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Traumatic Gallbladder Perforation Following Blunt Abdominal Trauma: A Rare but Fatal Complication

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Introduction

Motor vehicle collisions are among the most common causes of emergency department admissions and may present with a wide clinical spectrum ranging from minor injuries to severe trauma requiring urgent surgery (1). In patients with multiple trauma, certain intra-abdominal injuries may be overlooked during the initial assessment due to competing priorities. In abdominal trauma, the liver and spleen are the most frequently injured organs (2). Traumatic gallbladder perforation is extremely rare; however, when it occurs, early diagnosis is crucial because it may lead to serious complications such as biliary peritonitis and sepsis (3).

Case Report

A 76-year-old male with chronic kidney disease on hemodialysis, hypertension, and coronary artery disease was brought to the emergency department one hour after a motor vehicle collision. He was conscious and cooperative; blood pressure was 75/45 mmHg, heart rate 90 beats/min, and SpO_2 98%. Abdominal examination revealed right upper quadrant tenderness without peritoneal signs.

Venous blood gas showed lactate 6.9 mmol/L, consistent with metabolic acidosis. Serial hemograms demonstrated a decrease in hemoglobin (11.8 g/dL to 9.3 g/dL) and leukocytosis. Elevated AST and ALT were attributed to blunt hepatic injury.

Thoracic CT revealed a manubrium sterni fracture, multiple rib fractures, and minimal bilateral pneumothorax. Contrast-enhanced abdominal CT demonstrated gallbladder wall disruption with displacement of gallstones and intraluminal contents outside the gallbladder fossa. Grade 2 hepatic contusions and free intra-abdominal fluid were also present, consistent with traumatic gallbladder perforation.

Emergency exploratory laparotomy and cholecystectomy were performed. Intraoperatively, he developed hemodynamic instability requiring inotropic support and erythrocyte transfusion. In the ICU, metabolic acidosis and elevated lactate persisted despite broad-spectrum antibiotics. His condition deteriorated, leading to cardiac arrest and death despite advanced life support

Discussion

Traumatic gallbladder injuries account for approximately 1.9–2.1% of blunt abdominal trauma cases and may be diagnosed late due to their rarity (5). Clinical findings are often subtle, and contrast-enhanced CT is the primary diagnostic modality (6). Indirect signs such as pericholecystic fluid, wall contour disruption, and intraluminal high-density material should raise suspicion (6). When perforation is suspected, surgical exploration and cholecystectomy are generally recommended (3).

This case emphasizes that rare gallbladder injuries should be considered in trauma patients and that early imaging plays a vital role in diagnosis and management.

Keywords: Biliary peritonitis, Blunt abdominal trauma, Gallbladder perforation

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Fatal Pneumomediastinum Following Suspected Odontogenic Infection

Konuşmacı Şeyma Nur Çalışır

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1. Başakşehir Çam ve Sakura Şehir Hastanesi

Introduction

Pneumomediastinum is an uncommon condition characterized by the presence of air in the mediastinum and is usually benign and self-limiting (1). However, secondary pneumomediastinum associated with trauma, esophageal injury, or deep neck infections—particularly of odontogenic origin—may spread to the mediastinum and result in life-threatening complications such as pneumothorax and empyema (2).

Material and Method

A 49-year-old woman presented to the emergency department with throat swelling and dyspnea. Her medical history was significant for diabetes mellitus and hyperlipidemia. Her symptoms had progressively worsened over the preceding three days. She reported a history of tooth extraction three months earlier, with no recent history of trauma or any additional invasive procedure. On initial physical examination, she was found to be in severe respiratory distress. Non-crepitant swelling extending from the submandibular region was noted. Her vital signs revealed an oxygen saturation of 85% on room air, a heart rate of 128 beats/min, and an arterial blood pressure of 142/91 mmHg. Laboratory evaluation demonstrated elevated acute-phase reactants. Thoracic computed tomography showed air densities extending from the right cervical region into the mediastinum, pneumomediastinum, an associated right focal pneumothorax, and a 3.5-cm pleural effusion. During follow-up, the patient developed respiratory failure and was subsequently intubated. Following intubation, tube thoracostomy was performed, and pleural fluid analysis was consistent with empyema. Pleural fluid culture

grew Streptococcus anginosus. Despite intensive care management, her clinical condition progressively deteriorated, and she died during her stay in the intensive care unit.

Result and Conclusion

Pneumomediastinum is a rare emergency department presentation, most commonly seen in children and young adults (3). This case is remarkable because of its complicated course with pneumothorax and empyema. Intraoral procedures may cause deep neck infection and secondary pneumomediastinum (3 ,4). Although the dental extraction had been performed three months earlier, isolation of S. anginosus, an oral flora organism associated with abscess formation, together with cervical-to-mediastinal air on imaging, suggests an insidious oral and deep neck infectious source (5,6,7). Clinicians should consider infectious complications, pneumothorax, and empyema in pneumomediastinum patients with a history of dental procedures. Early recognition, close monitoring, and timely airway management are crucial for preventing mortality.

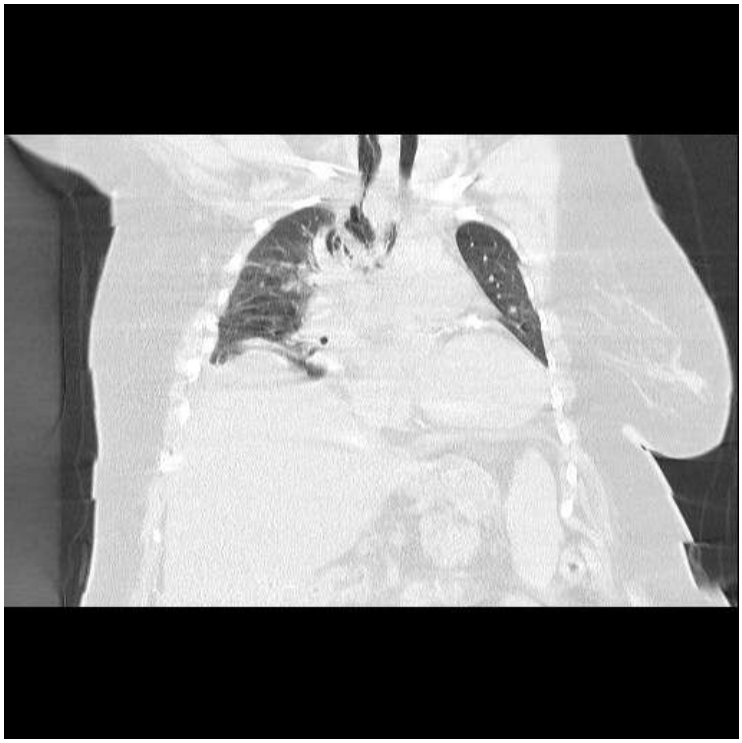

Figure 1. Coronal chest CT image revealed pneumomediastinum extending from the right cervical region, associated with a focal right pneumothorax and a 3.5-cm pleural effusion.

Figure 2. Transverse *chest CT image revealed pneumomediastinum extending from the right cervical region, associated with a focal right pneumothorax and a 3.5-cm pleural effusion.*

Figure 3. Posteroanterior chest radiograph

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Diagnostic Performance of a YOLOv8n-Based Artificial Intelligence Model for Detecting Nasal Bone Fractures on Lateral Nasopharyngeal Radiographs in the Emergency Department

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Background:
Nasal bone fractures are among the most frequently encountered facial injuries in emergency departments (EDs) (1). While lateral nasopharyngeal radiographs are widely used due to their accessibility and cost-effectiveness, interpretation often depends on the experience of the clinician or radiologist, which may lead to diagnostic variability, particularly in busy or high-volume ED settings (2). Recent advances in artificial intelligence (AI), especially deep learning models like YOLOv8n, offer the potential to enhance diagnostic accuracy and provide real-time decision support for fracture detection (3-6) This study aimed to assess the diagnostic performance and practical utility of a YOLOv8n-based AI model for identifying nasal bone fractures on lateral radiographs in a real-world ED population.

Methods:
We conducted a retrospective analytical study including 1,099 adult patients who presented with nasal trauma between January 2021 and December 2023. Radiographs were randomly divided into training (80%) and independent test (20%) datasets. Fracture status was determined based on combined clinical evaluation and radiological interpretation. The YOLOv8n model was trained for 50 epochs using standard data augmentation techniques. Performance metrics evaluated included accuracy, precision, recall (sensitivity), F1-score, and mean average precision at an intersection-over-union threshold of 0.5 (mAP@0.5). Additionally, confusion matrix analysis was performed to provide a detailed understanding of classification outcomes.

Results:
Among the patients, 62.9% were male, with a median age of 31 years. The overall fracture rate was 23.4%. Trauma mechanism was significantly associated with fracture presence (p=0.001), with assault-related injuries showing the highest fracture rate (34.5%). On the independent test set, the AI model demonstrated an accuracy of 86.8%, precision of 65.2%, recall of 69.8%, F1-score of 0.67, and mAP@0.5 of 0.711. Confusion matrix analysis revealed 30 true positives, 161 true negatives, 13 false negatives, and 16 false positives, highlighting the model's balanced performance in detecting fractures and non-fracture cases.

Conclusion:
The YOLOv8n-based AI model showed clinically acceptable performance for detecting nasal bone fractures on lateral radiographs and could serve as a rapid screening and decision-support tool in high-volume ED settings (6). Its integration into clinical workflows may improve efficiency, reduce diagnostic variability, and potentially mitigate medicolegal risks (3). Further multicenter prospective studies are warranted to validate these findings and explore real-time deployment in emergency care environments.

Keywords: Emergency department, nasal fracture, artificial intelligence, YOLOv8, deep learning

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Opinions of Pamukkale University Faculty of Medicine Students Regarding Emergency Medicine Residency Training

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Introduction

Emergency Medicine (EM) serves as one of the most critical and dynamic cornerstones of the healthcare system, yet it represents a complex decision point in the career planning of medical students. In both global and national contexts, trends in selecting EM residency through the Examination for Specialty in Medicine (TUS) are shaped by multi-dimensional factors such as professional satisfaction, lifestyle flexibility offered by the shift system, workload, stress, and security concerns. It is well-documented that elements like early clinical exposure, the presence of role models, and the perceived prestige of the branch are also decisive in students' specialty choices. This study, planned within the scope of the Special Study Module (SSM) by three second-year medical students , aims to comprehensively determine the perspectives of Pamukkale University Faculty of Medicine students regarding EM residency training, their reasons for choosing or avoiding this branch, and their inclinations toward alternative specialties.

Methods

In this study, the perceptions, trends, and underlying reasons for specialty choices regarding Emergency Medicine residency among 150 students at Pamukkale University Faculty of Medicine were evaluated. Our findings revealed that 38.7% of students believe they lack sufficient information about specialty fields in the TUS, while 40.7% remain undecided. This suggests that the specific awareness of medical students—especially 1st and 2nd-year students who constitute the majority (71.3%) of this study —regarding specialty branches has not yet fully matured during the preclinical stages. A comprehensive study by Ray et al. [1] emphasized that students' inclusion of branches like EM, where rotations typically occur in the later stages of education, into their career plans may be delayed. Our results support the notion that a large portion of students have not yet reached the decision-making phase and require more institutional guidance regarding the practical workflows of specialty fields.

Results

When examining the inclination toward choosing EM residency, only 2.7% of participants "definitely" and 18% "partially" considered it. In contrast, a significant majority of 56.7% stated they were not considering EM. A 14-year longitudinal study by Eken et al. [2] on medical students found that the rates of choosing EM as a future career fluctuated, with the perception of work being the primary determinant in these decisions. The low rates of definite preference in our study align with the specific challenges perceived in the EM branch within general literature, indicating that its position within TUS competition does not fully align with the expectations of today's students.

An analysis of the reasons provided by students who would consider EM shows that "Professional satisfaction" (29%) and "Short-term patient follow-up" (25.8%) were the most frequently reported motivations. Additionally, "Interest/Passion" (16.1%) and "Teamwork environment" (6.5%) emerged as other significant drivers. By nature, EM offers opportunities for rapid diagnosis, acute intervention, and immediate results. Alshahrani et al. [3] demonstrated that students choosing EM possess different values compared to those leaning toward surgical or internal branches, prioritizing hospital-centeredness and case variety. Our findings regarding "short-term patient follow-up" and "professional satisfaction" confirm that students prefer a dynamic workflow where they can take immediate action and see tangible results, rather than long-term chronic patient management. These characteristic features of EM practice continue to be a primary attraction for students inclined toward crisis management.

On the other hand, the reasons cited by students who definitely or partially do not consider EM reflect a landscape shaped by the physical and psychological rigors of the profession. "Heavy workload" (28.2%) , "Irregular working hours" (24.7%) , and "Stressful environment" (22.4%) were recorded as the fundamental reasons for rejection. These were accompanied by the "Pressure of rapid decision-making" (17.6%) and "Security concerns" (5.9%). This picture is highly consistent with current international literature. Dorsey et al. [4] established that the concept of a "controllable lifestyle" has become the most critical determinant in the specialty choices of recent medical graduates. In our study, the complaints centered on workload and shift-based systems clearly indicate that physician candidates do not wish to move away from a controllable lifestyle. Furthermore, the role of security concerns in the decision-making process is an important signal that the working environment in emergency departments needs to be made more secure.

The alternative specialty preferences of students not considering EM strongly support the "controllable lifestyle" thesis. The most preferred branches among this group were Ophthalmology (10.6%), Plastic, Reconstructive and Aesthetic Surgery (8.2%), and

Dermatology (7.1%). All these branches generally have fewer acute cases and lower shift burdens, possess regular outpatient hours, and are areas where placements are made with high TUS scores. National survey studies by Alfaqih et al. [5] have noted a similar trend, reporting an increased demand for branches offering lifestyle flexibility and a decrease in preference for high-stress branches. The fact that a significant proportion of students in our findings focused on comfortable and predictable branches is a concrete indicator of the tendency to avoid burnout syndrome.

Conclusion

In conclusion, these data obtained specifically from medical faculty students indicate that the perception of EM residency involves dual-sided dynamics. While professional satisfaction and short-term patient follow-up are the strongest aspects of the branch, heavy workload, irregular hours, and intense stress weaken its competitiveness in TUS preferences. To maintain interest in EM residency training in the future, it is of great importance to improve working conditions and introduce students to the branch accurately through early clinical rotations.

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Mobile Multiplace Hyperbaric Oxygen Unit for Crush Injuries in Earthquake Disasters: A National Capacity Analysis from Türkiye and a Deployment Model

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Introduction and Objective

Türkiye is located on active seismic fault lines and has experienced large-scale earthquakes causing significant morbidity. Crush injuries and acute compartment syndrome are major contributors to limb loss in disaster settings. Hyperbaric oxygen therapy (HBOT) enhances tissue oxygenation, reduces edema, improves leukocyte function, and has been reported to decrease amputation rates in selected cases of acute traumatic ischemia. However, access to HBOT during disasters may be limited due to geographic clustering of fixed centers. This study aimed to analyze the national hyperbaric treatment capacity in Türkiye and propose a deployable mobile multiplace HBOT system for early on-site intervention in disaster zones.

Materials and Methods

The number and geographic distribution of HBOT centers in Türkiye were evaluated using publicly available institutional data. Distribution patterns were analyzed in relation to earthquake-prone regions. Potential delays associated with intercity transfer of crush victims were conceptually assessed. Based on identified capacity gaps, a conceptual design model for a truck-mounted mobile multiplace hyperbaric chamber was developed to allow rapid deployment to affected areas.

Results and Conclusion

A total of 57 HBOT centers operate across 28 provinces in Türkiye, with significant concentration in major metropolitan areas. In large-scale disasters, transportation of crush victims to distant centers may result in treatment delays. A mobile multiplace HBOT unit could provide early intervention directly within disaster zones, potentially reducing amputation rates and improving limb salvage outcomes. Such a system may also support provinces without fixed HBOT infrastructure or areas where facilities are damaged during disasters. Integrating mobile hyperbaric systems into national disaster preparedness planning may strengthen acute trauma management capacity in Türkiye.

Keywords: hyperbaric oxygen therapy, crush injury, disaster medicine, earthquake, mobile medical systems

Introduction

In Türkiye, a seismically active country, large-scale earthquakes have repeatedly resulted in crush injuries associated with substantial morbidity, systemic complications, and limb loss. Crush injuries and acute compartment syndrome are among the most severe medical consequences of disasters, frequently leading to tissue ischemia, systemic inflammatory response, renal failure, and amputation if not managed promptly (1,2). Early intervention aimed at restoring tissue oxygenation and reducing edema is critical in preventing irreversible necrosis.

Hyperbaric oxygen therapy (HBOT) is an established adjunctive treatment in crush injuries and acute traumatic ischemia. By increasing dissolved oxygen in plasma under elevated atmospheric pressure, HBOT enhances oxygen delivery independent of hemoglobin concentration, reduces tissue edema via vasoconstriction without impairing oxygenation, improves leukocyte oxidative killing, inhibits anaerobic bacterial growth, and promotes angiogenesis and collagen synthesis (3,4). Clinical and experimental studies suggest that early HBOT may reduce amputation rates and improve limb salvage in crush-related injuries (2,5).

National Hyperbaric Capacity in Türkiye

Currently, 57 HBOT centers operate across 28 provinces in Türkiye, encompassing both public and private institutions. A substantial proportion of these facilities are concentrated in major metropolitan regions, resulting in geographic imbalance in national hyperbaric capacity (Figure 1). Such clustering may delay access to treatment during large-scale disasters, particularly in earthquake-prone areas lacking local infrastructure. Furthermore, seismic events may compromise hospital functionality and render fixed hyperbaric units temporarily inoperable. Intercity transfer of critically injured patients may therefore prolong HBOT initiation, potentially adversely affecting outcomes in time-sensitive ischemic conditions (1,2).

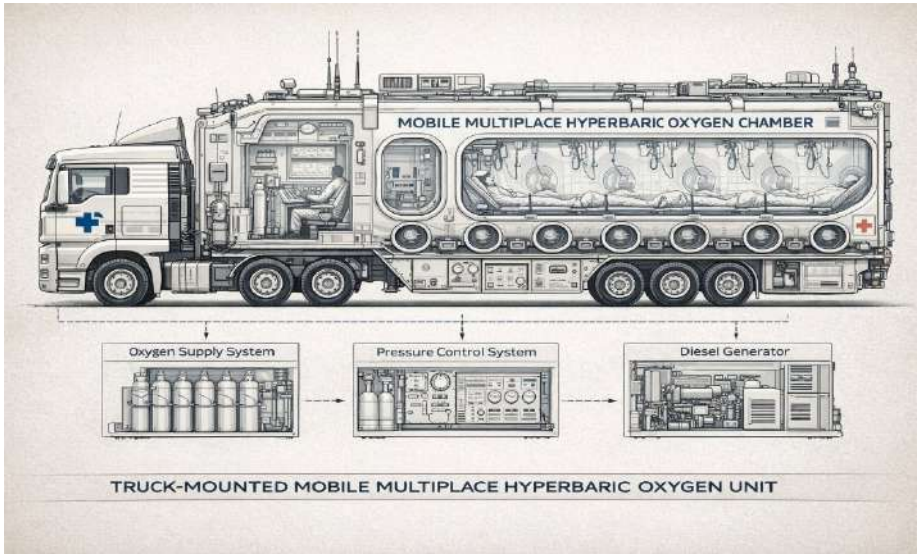
Figure 1. Geographic distribution of the 57 hyperbaric oxygen therapy (HBOT) centers across 28 cities in Türkiye included in the study. Additional HBOT facilities in Batman, Samsun,Aydın, Ordu, and Zonguldak are currently in the planning phase and are therefore not included in the present analysis.

Proposed Mobile Deployment Model

To address these limitations, a truck-mounted mobile multiplace hyperbaric chamber system is proposed as a deployable disaster-response solution (Figure 2). Mobile medical systems are increasingly recognized as essential components of disaster preparedness frameworks (6). The proposed model includes a multiplace chamber capable of treating multiple patients per session at 2.0–2.8 ATA, an integrated medical oxygen supply system, independent power generation, and reinforced structural design suitable for transportation and rapid setup. The system is designed for operational readiness within 6–8 hours of arrival and integration into regional emergency medical coordination networks.

Such a system could function as a primary HBOT facility in provinces lacking fixed infrastructure, as a supplementary unit when local capacity is overwhelmed, or as a temporary replacement if existing chambers are damaged. Integration of mobile hyperbaric systems into national disaster planning may enhance early intervention capacity and improve limb salvage outcomes in earthquake-related crush injuries.

Figure 2. Technical schematic representation of a truck-mounted mobile multiplace hyperbaric oxygen unit designed for rapid disaster deployment. The system includes a multiplace chamber (2.0–2.8 ATA capacity), integrated oxygen supply module, pressure control system, and independent power generator.



Conclusion

Crush injuries remain a leading cause of preventable limb loss in earthquake disasters. Despite the presence of established HBOT centers in Türkiye, geographic concentration and disaster-related infrastructure damage may significantly delay treatment initiation. Deployment of a mobile multiplace hyperbaric oxygen unit could enable time-critical on-site HBOT initiation during the early phase of reversible ischemic injury. Integration of mobile hyperbaric systems into national disaster preparedness planning may strengthen national disaster response capacity and contribute to improved limb salvage outcomes in earthquake-related traumatic ischemia.

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Postpartum Posterior Reversible Encephalopathy Syndrome Presenting with Tonic-Clonic Seizures: A Case Report

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a neurological syndrome characterized by headache, altered consciousness, seizures, visual symptoms, and characteristic imaging findings [1–3]. In terms of pathophysiology, the development of vasogenic edema due to impaired cerebral autoregulation, endothelial dysfunction, and loss of blood-brain barrier integrity is considered the primary mechanism [1,2].

In obstetric patients, PRES is most commonly associated with preeclampsia and eclampsia; it may occur during the antepartum, intrapartum, or postpartum periods [4,5]. According to World Health Organization data, preeclampsia affects approximately 3–8% of pregnancies and may progress to eclampsia [7]. Additionally, blood pressure may rise again in the first few days postpartum; therefore, the coexistence of postpartum seizures and hypertension should be carefully evaluated from a clinical perspective [5,8].

In this case report, a patient presenting with a history of tonic-clonic seizures and hypertensive encephalopathy in the postpartum period, who was diagnosed with PRES based on magnetic resonance imaging findings, is presented.

Case Presentation

A 26-year-old female patient was brought to the emergency department by ambulance due to a seizure during the postpartum period. It was learned that the patient had undergone a cesarean section four days earlier due to a twin pregnancy. Her medical history indicated no known chronic conditions other than goiter. She had experienced a tonic-clonic seizure lasting approximately 3 minutes at an external facility. After receiving two doses of 5 mg diazepam intravenously 10 minutes apart, followed by a loading dose of 6 mg magnesium sulfate in 150 ml of 5% dextrose administered intravenously over 30 minutes, she was transferred to our center.

A video recording taken by his relatives during the seizure showed loss of consciousness, ocular deviation, and tonic-clonic convulsions. During the examination in the emergency department, the patient was confused, disoriented, and uncooperative; he responded to painful stimuli but did not provide meaningful responses to verbal commands. Arterial blood pressure

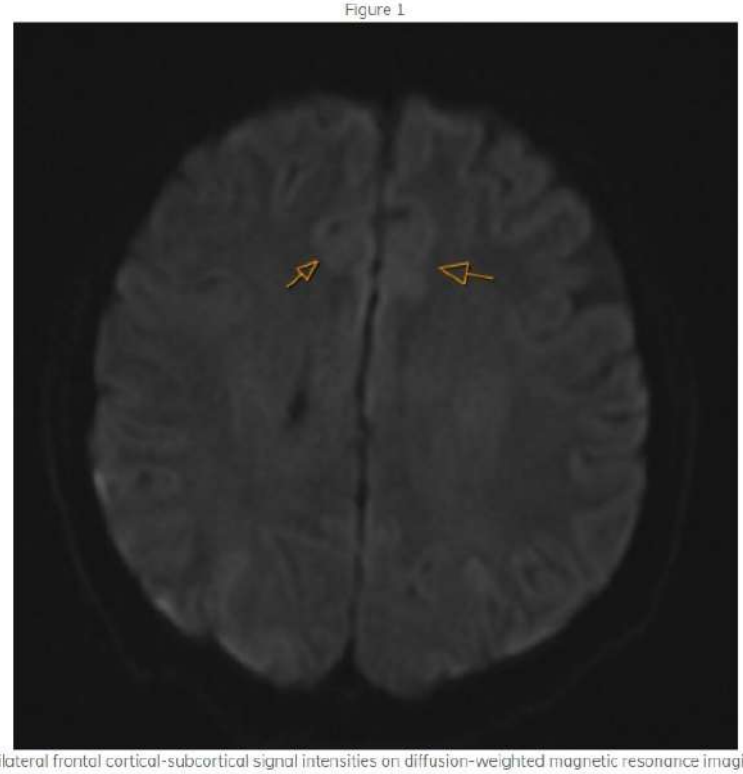
was measured at 170/120 mmHg, pulse rate at 105 bpm, temperature at 36.4°C, and oxygen saturation at 94%. No significant focal motor deficits were detected.

On obstetric examination, the uterus appeared postpartum; no free fluid was detected in the Douglas or Morison spaces. No active vaginal bleeding was observed, and no additional obstetric complications requiring urgent surgical intervention were suspected. The Pfannenstiel incision site was clean. Approximately 1200 mL of urine was drained via the Foley catheter.

Laboratory findings: hemoglobin 9.3 g/dL, white blood cells 11.15 × 10^3/µL, platelets 175 × 10^3/µL, C-reactive protein 5.09 mg/dL, lactate dehydrogenase 327 U/L, uric acid 9.2 mg/dL, albumin 2.6 g/dL, calcium 8.5 mg/dL, creatine kinase 212 U/L, and arterial lactate 3.68 mmol/L. Coagulation parameters were within normal limits.

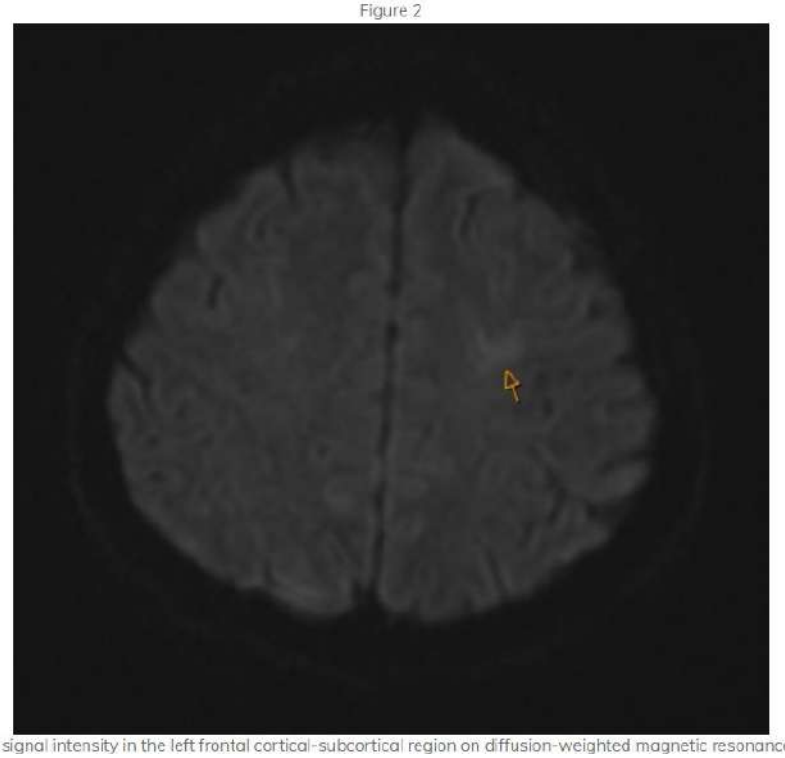
No acute intracranial pathology was detected on non-contrast cranial computed tomography. Consequently, further neuroimaging was performed. Diffusion-weighted magnetic resonance imaging did not reveal diffusion restriction suggestive of acute infarction; signal intensifications were noted in bilateral parieto-occipital cortical-subcortical areas and frontal regions. The findings were reported as consistent with PRES (Figure 1, Figure 2).

Figure 1



Bilateral frontal cortical-subcortical signal intensities on diffusion-weighted magnetic resonance imaging

Figure 2



Increased signal intensity in the left frontal cortical-subcortical region on diffusion-weighted magnetic resonance imaging

The patient was diagnosed with postpartum PRES following a comprehensive evaluation of the postpartum period, severe hypertension, recurrent seizures, encephalopathy, and imaging findings. The patient was admitted to the neurology intensive care unit for close monitoring, and blood pressure was brought under control. Approximately one week later, the patient was discharged in good health.

Discussion

This case demonstrates that PRES can present with seizures and altered consciousness in the postpartum period. The classic clinical spectrum of PRES includes headache, seizures, encephalopathy, and visual symptoms; however, the clinical presentation may vary among cases [1–3]. In obstetric patients, particularly during the postpartum period, the PRES and eclampsia spectra should be evaluated in conjunction when severe, newly onset hypertension and seizures are present [4,5].

From a diagnostic perspective, this case is notable because magnetic resonance imaging (MRI) revealed the typical PRES pattern despite a normal cranial computed tomography (CT) scan. CT may be normal in the early stages or may have limited sensitivity; in contrast, MRI is superior in demonstrating vasogenic edema, particularly with FLAIR and diffusion sequences [1–3]. The absence of diffusion restriction has weakened the likelihood of ischemic infarction and has been interpreted in favor of PRES [1,2].

Cerebral venous thrombosis must be considered in the differential diagnosis of postpartum seizures. Since the postpartum period is associated with hypercoagulability, venous imaging methods should be incorporated into the diagnostic process when clinically indicated [9]. However, in the present case, the presence of a posterior-predominant vasogenic edema pattern and marked hypertension has led to a diagnosis of PRES.

The primary approach to management is the rapid control of the triggering mechanism. Obstetric guidelines recommend the immediate treatment of acute-onset severe hypertension during pregnancy and the postpartum period, and the use of intravenous labetalol, intravenous hydralazine, or oral rapid-release nifedipine in appropriate cases [8]. In patients with eclampsia or suspected eclampsia, magnesium sulfate is the primary agent for both the treatment and prevention of seizures [7,8].

Although PRES is reversible in most cases with appropriate treatment, delayed diagnosis or inadequate management may lead to cerebral infarction, hemorrhage, status epilepticus, or permanent neurological sequelae [1–3,6]. Therefore, the coexistence of seizures and hypertension in postpartum patients should be considered not merely as symptomatic seizures but as an indicator of underlying potential neurovascular and obstetric emergencies.

Conclusion

In patients presenting with seizures, altered consciousness, and severe hypertension during the postpartum period, PRES must be considered in the differential diagnosis. A normal cranial computed tomography scan does not rule out the diagnosis; magnetic resonance imaging is definitive for diagnosis [1–3]. Early diagnosis, appropriate antihypertensive treatment, seizure control, and a multidisciplinary approach are critical for preventing irreversible neurological damage [4,8].

Ethics Committee Approval: Not applicable.

Informed Consent: Written informed consent was obtained from the patient for the publication of this case report.

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Acute Liver Failure and Hepatic Encephalopathy Developing on the Basis of Chronic Alcoholism

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Abstract

In this case report, we presented a 22-year-old female patient with chronic alcoholism who experienced a hepatic encephalopathy episode with progressive hyperbilirubinemia, hyponatremia, disorientation, and asterixis following alcohol intoxication and was referred to a liver transplant centre. A 22-year-old female patient was brought to the emergency department with a diagnosis of alcohol intoxication. She was uncooperative and agitated secondary to high-dose alcohol ingestion. Laboratory investigations revealed aspartate aminotransferase (AST) 466 U/L, alanine aminotransferase (ALT) 307 U/L, gamma-glutamyl transferase (GGT) 1254 U/L, total bilirubin 2.2 mg/dL, sodium 131 mEq/L, and an ethanol level of 469.3 mg/dL. Abdominal ultrasonography showed only Grade 2 steatosis, with no other significant pathology. The patient was admitted to the intensive care unit due to treatment-resistant active nausea and vomit. During follow-up, her ethanol level decreased to 198 mg/dL, and transaminase levels dropped by 20–30%. However, the total bilirubin level rose to 7.7 mg/dL, resulting in a marked icteric clinical state. Prognostic scoring yielded a Model for End-Stage Liver Disease score of 18. Visual and auditory hallucinations and asterixis developed, indicating the progression of hepatic encephalopathy. The patient, evaluated as stage 3 hepatic encephalopathy, was transferred to a centre with gastroenterology and liver transplant capabilities for further management. Hepatic encephalopathy must be included in the differential diagnosis of patients presenting with altered consciousness and a history of alcohol use. Furthermore, it should be kept in mind that liver transplantation may be required in patients with active hepatic encephalopathy and a Model for End-Stage Liver Disease score above 15.

Key words: Alcoholism, Hepatic encephalopathy, MELD.

Introduction

Hepatic encephalopathy (HE), defined as a neuropsychiatric syndrome resulting from the accumulation of neurotoxic substances due to liver dysfunction, presents with varying clinical manifestations depending on the severity of the underlying liver failure (1). In this case report, we presented a 22-year-old female patient with chronic alcoholism who experienced an HE episode with progressive hyperbilirubinemia, hyponatremia, disorientation, and asterixis following alcohol intoxication and was referred to a liver transplant centre.

Case Report

A 22-year-old female patient was brought to the emergency department with a diagnosis of alcohol intoxication. She was uncooperative and agitated secondary to high-dose alcohol ingestion. Her medical history revealed chronic alcoholism and early-stage liver cirrhosis. Upon initial assessment, her Glasgow Coma Scale (GCS) score was 15, and she was oriented. Vital signs at admission were as follows: blood pressure 120/80 mmHg, pulse 56 bpm, temperature 36.8°C, respiratory rate 16 breaths/minute, and oxygen saturation 95%. Her bedside capillary blood glucose was 120 mg/dL. Laboratory investigations revealed aspartate aminotransferase (AST) 466 U/L, alanine aminotransferase (ALT) 307 U/L, gamma-glutamyl transferase (GGT) 1254 U/L, total bilirubin 2.2 mg/dL, sodium 131 mEq/L, and an ethanol level of 469.3 mg/dL. Abdominal ultrasonography showed only Grade 2 steatosis, with no other significant pathology. The patient was admitted to the intensive care unit due to treatment-resistant active nausea and vomit. During follow-up, her ethanol level decreased to 198 mg/dL, and transaminase levels dropped by 20–30%. However, the total bilirubin level rose to 7.7 mg/dL, resulting in a marked icteric clinical state. Prognostic scoring yielded a Model for End-Stage Liver Disease (MELD) score of 18. Visual and auditory hallucinations and asterixis (flapping tremor) developed, indicating the progression of HE. The patient, evaluated as Stage 3 HE, was transferred to a centre with gastroenterology and liver transplant capabilities for further management.

Discussion

There is no specific biomarker for the diagnosis of HE; the diagnosis is primarily based on neuropsychiatric examination and the exclusion of other potential causes (2). Typically, focal neurological deficits are not seen in HE; if present, intracranial pathologies must be ruled out. Ammonia enters the systemic circulation as a result of the loss of the liver's detoxification capacity. In HE occurring on the basis of acute liver failure, unlike in cirrhosis, cytotoxic cerebral oedema and the consequent increase in intracranial pressure are the primary causes of mortality (3). In our case, the observed hyponatremia was considered a significant factor accelerating neurological deterioration. Furthermore, the presence of visual/auditory hallucinations and asterixis led to the classification of the patient as Stage 3 HE.

The MELD score is a crucial parameter used to predict the severity of liver disease and short-term mortality risk (4). Moreover, even a single episode of severe hepatic encephalopathy is considered a significant prognostic factor that may negatively impact long-term survival, even if partial improvement in liver function is achieved (5). In our case, it was determined that this was the patient's first HE episode. Additionally, a MELD score of 18 demonstrated that the patient was experiencing severe liver failure.

Conclusion

Hepatic encephalopathy must be included in the differential diagnosis of patients presenting with altered consciousness and a history of alcohol use. Furthermore, it should be kept in mind that liver transplantation may be required in patients with active hepatic encephalopathy and a MELD score above 15.

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Extreme High Anion Gap Metabolic Acidosis and Anuric Acute Kidney Injury Requiring Dialysis Triggered by Gastrointestinal Infection in Severe Anorexia Nervosa: A Case Report

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Abstract

Objective

Anorexia nervosa is characterized by severe malnutrition and restricted energy intake and is associated with systemic complications that may lead to electrolyte imbalances, acid–base disturbances, and multi-organ dysfunction. Prolonged starvation, vomiting, and hypovolemia may result in life-threatening metabolic conditions such as lactic acidosis, ketoacidosis, and acute kidney injury. In this case report, we present a patient with anorexia nervosa who developed extreme high anion gap metabolic acidosis and anuric acute kidney injury requiring emergency hemodialysis.

Case

A 24-year-old female patient with a history of anorexia nervosa since childhood presented to the emergency department with cessation of oral intake, recurrent vomiting, abdominal pain, and anuria. On admission, she appeared severely cachectic (BMI 11.7 kg/m²) and had marked hypotension (70/40 mmHg). Arterial blood gas analysis revealed a pH of 6.90, HCO_3^- of 6.4 mmol/L, and lactate level of 16 mmol/L. The calculated anion gap was 40.6 mmol/L, consistent with severe high anion gap metabolic acidosis. The presence of urinary ketones suggested a multifactorial metabolic acidosis involving both lactic acidosis and starvation

ketoacidosis. Due to anuric acute kidney injury and profound metabolic acidosis, emergency hemodialysis was initiated under vasopressor support. Significant improvement in acid–base parameters was observed following hemodialysis. Stool culture revealed the growth of *Salmonella* species, suggesting that gastrointestinal infection may have contributed to dehydration and metabolic decompensation.

Conclusion

In patients with anorexia nervosa, severe malnutrition and hypovolemia combined with triggering factors such as infection may lead to extreme metabolic acidosis and acute kidney injury. Early acid–base analysis, hemodynamic stabilization, and timely initiation of renal replacement therapy are critical in reducing mortality in such cases.

Keywords

Anorexia nervosa; Lactic acidosis; High anion gap metabolic acidosis; Starvation ketoacidosis; Acute kidney injury; Hemodialysis

Introduction

Anorexia nervosa is a severe eating disorder characterized by significant weight loss, marked restriction of energy intake, and distorted body image. However, it is not solely a psychiatric condition; it is also recognized as a systemic disease associated with severe malnutrition, electrolyte disturbances, cardiovascular complications, and metabolic disorders (1,2).

During prolonged starvation, the body initially utilizes glycogen stores for energy production, followed by fatty acid oxidation and ketogenesis. In the presence of severe malnutrition and hypovolemia, mitochondrial oxidative phosphorylation becomes impaired, and anaerobic metabolism predominates. This process leads to increased lactate production and may result in severe lactic acidosis (3,4).

Additionally, vomiting, laxative abuse, and gastrointestinal infections—commonly observed in anorexia nervosa—can cause significant fluid loss, leading to renal hypoperfusion and the development of acute kidney injury. Prolonged hypoperfusion may progress from prerenal azotemia to ischemic tubular injury, eventually requiring renal replacement therapy (7).

In this case report, we present a patient with anorexia nervosa who developed extreme high anion gap metabolic acidosis and anuric acute kidney injury triggered by gastrointestinal infection and severe dehydration, requiring emergency hemodialysis.

Case Presentation

A 24-year-old female patient presented to the emergency department with complaints of complete cessation of oral intake for the past two days, recurrent nausea and vomiting, and

abdominal pain. It was learned that the patient had been diagnosed with anorexia nervosa since childhood and had experienced significant weight loss recently.

On physical examination, the patient appeared severely cachectic (height 160 cm, weight 30 kg, BMI 11.7 kg/m²). Initial vital signs revealed blood pressure of 70/40 mmHg, heart rate of 110 beats/min, respiratory rate of 20 breaths/min, and oxygen saturation of 99%.

Arterial blood gas analysis demonstrated pH 6.90, pCO_2 19 mmHg, and HCO_3^- 6.4 mmol/L. The calculated anion gap, based on serum electrolytes (Na 139 mEq/L, Cl 92 mEq/L), was 40.6 mmol/L. Lactate level was 16 mmol/L. Urinalysis revealed positive ketones.

Biochemical evaluation showed a creatinine level of 5.13 mg/dL (baseline 0.72 mg/dL), consistent with acute kidney injury. The patient was anuric.

Aggressive fluid resuscitation was initiated; however, hypotension persisted, necessitating vasopressor support with norepinephrine infusion. Due to severe metabolic acidosis, anuria, and hemodynamic instability, emergency renal replacement therapy was indicated, and a four-hour hemodialysis session was performed.

Post-dialysis arterial blood gas analysis showed significant improvement (pH 7.33; HCO_3^- 18 mmol/L; lactate 9.4 mmol/L).

Infectious workup revealed growth of *Salmonella* species in stool culture. Blood cultures were negative. Urine culture showed 10^4 CFU/mL *Enterococcus faecalis*, which was considered of limited clinical significance.

The patient was admitted to the intensive care unit for close monitoring of fluid-electrolyte balance, renal function, and metabolic parameters.

Discussion

In this case, severe metabolic decompensation in the setting of anorexia nervosa was characterized by extreme high anion gap metabolic acidosis and anuric acute kidney injury. The initial pH value of 6.90 is considered near the lower limit compatible with life.

The metabolic acidosis in this patient is thought to be multifactorial. First, severe hypovolemia and tissue hypoperfusion played a central role in the development of Type A lactic acidosis (3,4).

Second, prolonged starvation and severe malnutrition contributed to metabolic acidosis through increased ketogenesis. The presence of urinary ketones supports the contribution of starvation ketoacidosis (8).

Gastrointestinal infections are important triggers of metabolic decompensation in patients with anorexia nervosa. In this case, *Salmonella* infection likely exacerbated vomiting and dehydration, contributing to hypovolemic shock.

Renal hypoperfusion due to hypovolemia was considered the primary mechanism underlying acute kidney injury. Prolonged hypoperfusion may lead to ischemic tubular injury following prerenal azotemia.

Severe metabolic acidosis (pH <7.1) is associated with decreased myocardial contractility, peripheral vasodilation, and impaired responsiveness to vasopressors. Therefore, in the presence of profound metabolic acidosis, anuria, and hemodynamic instability, renal replacement therapy constitutes a strong indication (6,7).

This case demonstrates that anorexia nervosa, when not managed appropriately, can lead to extreme metabolic disturbances and life-threatening complications.

Conclusion

Anorexia nervosa is not merely a psychiatric eating disorder but a serious systemic disease that can lead to severe metabolic complications. Triggering factors such as prolonged starvation, vomiting, and infection may predispose patients to high anion gap metabolic acidosis and acute kidney injury.

Therefore, early acid–base evaluation, close monitoring of renal function, and timely initiation of renal replacement therapy in patients with anorexia nervosa can be life-saving.

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Retrospective Analysis of Crimean-Congo Hemorrhagic Fever Cases Presenting to the Emergency Department

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Introduction

Crimean-Congo hemorrhagic fever (CCHF) is a life-threatening viral infection in endemic regions, often presenting with nonspecific symptoms in the emergency department (ED), which may delay diagnosis. Early recognition is crucial for timely management and infection control.

Methods

This retrospective study evaluated 15 patients with PCR-confirmed CCHF who presented or were referred to the Emergency Department of Manisa Celal Bayar University between 2012 and 2025. Demographic, clinical, laboratory, and initial treatment data were obtained from medical records.

Results

Fifty-three percent of patients were female, with a mean age of 48.3 ± 13.1 years. Most patients (93.3%) lived in rural areas, and 40% were engaged in farming or animal husbandry. The mean time from tick exposure to hospital admission was 9.08 (2–30) days. Fever and fatigue were present in all patients, while nausea–vomiting (46.6%), myalgia, and abdominal pain (40%) were common. Hemorrhagic manifestations were observed in 73.3% of cases.

At ED admission, leukopenia (86.6%), neutropenia (60%), and thrombocytopenia (80%) were prominent findings. Prolonged INR and aPTT were detected in 40% and 46.6% of patients, respectively. Median fibrinogen and D-dimer levels were 260 [220.5–292] g/L and 605 [115.75–1251.5] ng/mL. Elevated AST and ALT levels were observed in 86.6% and 73.3% of patients, respectively, and 71.4% had elevated CPK levels. A significant negative correlation was found between platelet count and AST/ALT levels ($p<0.05$).

All patients received ribavirin, and 66.6% required blood product transfusion. Intensive care unit admission was necessary in 20% of cases, with an in-hospital mortality rate of 6.6%.

Discussion

In conclusion, in endemic areas, the combination of exposure history, fever, thrombocytopenia, and elevated transaminases should prompt early suspicion of CCHF in the ED. Rapid diagnosis is essential to improve outcomes and prevent transmission.

Keywords Crimean-Congo hemorrhagic fever; Emergency department; Thrombocytopenia

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Evaluation of Inflammatory and Biochemical Markers in Hospitalized Acute Gastroenteritis Cases

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ABSTRACT

Objective: The present study aims to investigate the demographic characteristics and the impact of laboratory parameters (WBC, CRP, albumin, BUN/creatinine ratio) on the length of hospital stay in adult patients hospitalized with a preliminary diagnosis of acute gastroenteritis (AGE).

Materials and Methods: A total of 37 patients over the age of 18 with complete data records were included in the study. For data analysis, following normality testing, the Mann-Whitney U test, Independent Samples t-test, and Spearman correlation analysis were utilized. The threshold for statistical significance was set at $p < 0.05$.

Results: The mean age of the patients was 70.22±14.21 years, with males comprising 62.2% of the study population. The median length of hospital stay was determined to be 3 days. Correlation analyses revealed a very strong, positive, and highly significant relationship between the length of hospital stay and CRP levels ($r = 0.946$; $p < 0.001$). A strong positive correlation was observed between age and the BUN/creatinine ratio ($r = 0.699$; $p < 0.001$). Additionally, moderate, negative significant correlations were identified between albumin levels and age ($r = -0.431$; $p = 0.008$), as well as WBC counts ($r = -0.529$; $p = 0.001$). No statistically significant differences were found between genders regarding laboratory parameters and the length of stay.

Discussion and Conclusion: CRP levels serve as a robust predictor of the length of hospital stay, particularly in elderly patients with AGE. Furthermore, the age-related decline in renal perfusion (indicated by an elevated BUN/creatinine ratio) and decreased albumin levels underscore the critical need for close clinical monitoring in this vulnerable demographic.

Keywords: Acute Gastroenteritis, CRP, BUN/Creatinine Ratio, Length of Hospital Stay, Dehydration.

INTRODUCTION

Acute gastroenteritis (AGE) is a critical public health issue that causes billions of cases worldwide each year and remains one of the leading causes of childhood mortality, particularly in developing countries. Although global mortality rates have decreased thanks to current rehydration therapies, the disease still causes significant morbidity and loss of workforce. Similarly in Turkey, the increasing number of cases during seasonal transitions constitutes a significant portion of emergency department admissions. The high incidence rate increasingly compounds the burden of this disease on public health day by day (1).

AGE, which can often be perceived as a "routine" or "simple" clinical picture because it is frequently encountered in daily practice by physicians and healthcare professionals, is actually a condition that has been simplified thanks to modern medical capabilities but can be fatal if not properly treated. Especially in vulnerable groups such as the elderly, immunocompromised individuals, and children, the clinical picture can rapidly progress to severe dehydration, electrolyte imbalance, and acute kidney injury.

The decision for hospitalization is made for reasons such as impaired oral intake, intractable vomiting, signs of severe dehydration, or marked deterioration in laboratory parameters, and mortality risk becomes inevitable in cases where timely intervention is not performed (2). In this study, the relationship between demographic characteristics and inflammatory and biochemical markers of 37 hospitalized acute gastroenteritis cases was investigated. In addition to the age and gender distributions of the patients; the effects of leukocyte (WBC), C-reactive protein (CRP), albumin levels, and the BUN/creatinine ratio, which is a sensitive

indicator of renal perfusion, on the length of hospital stay were evaluated. The analysis of these parameters aims to highlight the necessity of predicting the clinical course and applying treatment procedures meticulously.

MATERIAL AND METHODS

The SPSS V26.0 software package was used for data analysis. Whether the obtained data exhibited a normal distribution was analyzed using Shapiro-Wilk tests. In the comparison of continuous variables between categorical groups, the Mann-Whitney U test was preferred because the data exhibited a non-parametric distribution. For parametric comparisons between groups, the Independent Samples T-test was applied. Spearman correlation analysis was performed to determine the degree and direction of possible relationships between continuous variables. In all analyses, the limit of statistical significance was accepted as $p < 0.05$.

INCLUSION CRITERIA

Patients who presented to the emergency department with a preliminary diagnosis of acute gastroenteritis (AGE) and were decided to receive inpatient treatment.

Adult individuals who are 18 years of age or older at the time of admission.

Cases whose laboratory parameters investigated within the scope of the study (WBC, CRP, albumin, BUN, creatinine) are entirely recorded, and whose admission-discharge periods are clearly documented.

EXCLUSION CRITERIA

Pediatric patients under 18 years of age.

Cases followed up on an outpatient basis (polyclinic or emergency observation) and discharged, without hospitalization.

Cases with missing data in their file records or whose laboratory results could not be accessed.

Patients with primary infections outside the gastrointestinal tract (e.g., pneumonia, urinary tract infection) or known to have a diagnosis of chronic inflammatory bowel disease.

RESULTS:

The mean age of the 37 cases included in the study was determined as 70.22 ± 14.21. Of the cases, 62.2% (n=23) were male, and 37.8% (n=14) were female. Descriptive statistics regarding the patients' laboratory values and length of hospital stay are presented in Table 1.

Table 1. Descriptive Statistics Regarding Demographic Characteristics, Laboratory Parameters, and Clinical Data of the Patients

	n	mean	±sd		n	median	IQR(25-75)
age	37	70,22	14,21	wbc	37	7,2	4,55-11,45
				crp	37	9,7	4,4-12,75
				albumin	37	3,1	2,9-3,3
				Bun/kre	37	50	32,58-64,72
				h-los	37	3	2,0-4,0

The median WBC value of the cases was at the level of 7.2 (IQR: 4.55 - 11.45). The median of the CRP value, an indicator of inflammation, was determined as 9.7 mg/L (IQR: 4.4 - 12.75). The median albumin value of the patients was measured as 3.1 g/dL (IQR: 2.9 - 3.3). The median of the BUN/Creatinine ratio, an indicator of the prerenal state and dehydration, was found to be 50 (IQR: 32.58 - 64.72). When the lengths of hospital stay of the cases were evaluated, the median length of stay was observed to be 3 days (IQR: 2.0 - 4.0).

In the analyses performed, no statistically significant difference was found between male and female patients in terms of WBC, CRP, albumin, BUN/creatinine ratio, and length of stay ($p > 0.05$). According to the results of the Spearman correlation analysis performed to determine the relationship between parameters:

A positive, very strong, and statistically highly significant correlation was detected between the length of stay and CRP levels ($r = 0.946$; $p < 0.001$).

It was observed that as age increased, the BUN/creatinine ratio increased, demonstrating a positive and strong correlation between the two variables ($r = 0.699$; $p < 0.001$).

A negative, moderately significant correlation was detected between the albumin level and age ($r = -0.431$; $p = 0.008$), as well as WBC ($r = -0.529$; $p = 0.001$).

DISCUSSION

In this study, the relationships between the laboratory parameters and the length of hospital stay in patients with acute gastroenteritis treated on an inpatient basis were investigated. The findings obtained are noteworthy in terms of reflecting the effects of disease severity, inflammatory response, and demographic characteristics of patients on the clinical picture.

In our study, the mean age of a total of 37 hospitalized AGE cases was determined as 70.22 ± 14.21 (3). A study that conducted a comprehensive review and evaluation for AGE cases also mentions that hospitalized AGE cases are generally in the advanced age group. We believe that the fact that hospitalized AGE cases consist of the advanced age group stems from simple-seeming clinical presentations suddenly progressing to a picture of profound dehydration due to declining renal functional reserve with advancing age, coupled with delays caused by the need for caregiver support for hospital admission.

Of our cases, 62.2% (n=23) were male and 37.8% (n=14) were female (4). Similar clinical studies on gastroenteritis cases have also shown male predominance, and a higher rate is present in our study. In our study, where we examined only AGE cases treated on an inpatient basis, this proportional highness in favor of the male gender can be explained by the delay of men in seeking medical help due to sociocultural structure and gender roles, and the deepening of dehydration and the progression of the infectious process due to this delay.

The median WBC value of the cases is at the level of 7.2 (IQR: 4.55 - 11.45). In contrast to the literature for AGE, which is an infectious disease, normal white blood cell values were detected in our study instead of elevated white blood cells (5). The absence of an elevation in white blood cells may indicate that our gastroenteritis cases are of viral origin; however, we do not have data regarding the agent. Furthermore, the fact that our sample consists of elderly patients and that these patients have reduced immune responses can also explain the normal white blood cell levels.

In our study, no statistically significant difference was found between female and male patients in terms of leukocyte (WBC), C-reactive protein (CRP), albumin, BUN/creatinine ratio, and lengths of stay ($p > 0.05$). This situation indicates that the inflammatory response and fluid-electrolyte imbalances developing during the course of acute gastroenteritis progress through similar mechanisms independently of gender. Many studies in the literature also support that gender is not a determining factor on the severity of acute gastrointestinal infections and the length of hospital stay (3).

The positive, very strong, and highly significant correlation ($r = 0.946$; $p < 0.001$) detected between the length of stay and CRP levels as a result of the analysis is one of the most striking findings of our study. CRP, as is known, is an important acute-phase reactant synthesized in the liver and reflects the severity of infection/inflammation (6). High CRP levels generally indicate bacterial agents or more invasive mucosal damage (7). In cases where CRP is elevated, the need for intravenous antibiotic therapy in addition to fluid replacement and the time required for the clinical picture to resolve directly explain the prolongation of the hospital stay. This finding demonstrates that CRP can be used as a very strong biomarker in predicting the length of stay in patients with acute gastroenteritis.

The strong positive correlation ($r = 0.699$; $p < 0.001$) showing that the BUN/creatinine ratio increases with age indicates the high risk of dehydration and prerenal azotemia in elderly patients. Severe fluid losses occur through diarrhea and vomiting in acute gastroenteritis. In elderly individuals, decreased sensation of thirst, physiological decline in basal kidney functions, and a lower total body fluid ratio cause fluid loss to impair renal perfusion much

more rapidly (8). An increased BUN/creatinine ratio indicates decreased renal blood flow due to hypovolemia and emphasizes how critically and aggressively fluid replacement therapy must be managed in the advanced age group.

In our study, moderate, significant negative correlations were found between albumin levels and age ($r = -0.431$; $p = 0.008$), as well as WBC ($r = -0.529$; $p = 0.001$). Age and Albumin: The decrease in albumin with advancing age reflects that the basal nutritional status (malnutrition) may be more inadequate in elderly patients and the age-related physiological decline in the protein synthesis capacity of the liver. WBC and Albumin: Albumin is also a "negative acute-phase reactant". In states of acute infection, while the liver prioritizes the synthesis of positive acute-phase proteins such as CRP, it decreases albumin production. Furthermore, increased capillary permeability secondary to inflammation causes albumin to leak extravascularly (9). The decrease in albumin levels in situations where WBC, hence inflammation, increases is an expected and classic indicator of the systemic inflammatory response (9).

CONCLUSION

In summary, in cases of acute gastroenteritis requiring hospitalization, the CRP level is a very strong determinant of the length of hospital stay. Advanced age is a major risk factor for severe dehydration and consequential prerenal acute kidney injury (high BUN/creatinine ratio). An increased inflammatory response (WBC elevation), on the other hand, manifests itself with a decrease in the levels of albumin, which is a negative acute-phase reactant. These findings demonstrate that patients presenting especially at an advanced age and with high CRP values should be monitored more closely, and their fluid-electrolyte and nutritional support should be rapidly planned.

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3787

Acute Pancreatitis with Inflammatory Progression Despite Early Regression of Pancreatic Enzymes and Imaging Findings of Mesenteric Inflammation: An Atypical Course Causing Diagnostic Challenge

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Introduction

Acute pancreatitis (AP) is an inflammatory disease of the pancreas that typically presents with epigastric pain and elevated pancreatic enzyme levels. Diagnosis is established when at least two of the following criteria are present: characteristic abdominal pain, serum amylase or lipase levels at least three times the upper limit of normal, and imaging findings consistent with pancreatitis (1). However, in clinical practice, not all patients meet these classic biochemical thresholds. Particularly in late-presenting cases, pancreatic enzyme levels may decline, creating diagnostic challenges (2).

Imaging modalities, especially contrast-enhanced computed tomography (CT), play a crucial role in confirming the diagnosis and evaluating complications of pancreatitis. While the pancreatic parenchyma may appear normal in the early stages, indirect findings such as peripancreatic fat inflammation, mesenteric edema, and fat stranding may be detected (3). These findings, when evaluated together with systemic inflammatory response, may directly influence clinical management.

In this report, we present an atypical case of acute pancreatitis characterized by early regression of pancreatic enzymes but progression of inflammatory parameters, supported by imaging findings of mesenteric inflammation.

Case

A 57 year old female presented to the emergency department with epigastric pain, nausea, and vomiting that started four days prior, accompanied by fever, chills, and shivering.

The pain was reported to radiate to the back, particularly while lying down. Due to nausea and vomiting over the past three days, impaired oral intake resulted in approximately 6 kg weight loss and significant fatigue.

Her medical history included hypertension, hypothyroidism, depression, and hyperlipidemia. She was using levothyroxine, bisoprolol, an angiotensin receptor blocker, sertraline, and atorvastatin. She had a history of total abdominal hysterectomy and bilateral salpingo-oophorectomy performed 14 years earlier.

Physical examination revealed tenderness in the epigastric and right upper quadrant regions; no guarding or rebound tenderness was observed. Initial laboratory results showed C-reactive protein (CRP) 34 mg/L, amylase 149 U/L, and lipase 177 U/L. No other significant abnormalities were detected in biochemical parameters.

Figure 1 Contrast-enhanced abdominal CT demonstrated mild edema in the mesenteric planes of the upper and middle abdomen, suggestive of an infectious process. Additionally, mesenteric fat stranding was observed around the inferior pancreas and at the pancreatic head-body junction.

Figure 2 Inflammatory fat stranding and increased density were observed around the second portion of the duodenum.

No mass, abscess, pseudocyst, or significant necrosis was detected.

The patient was admitted to the general surgery ward. On the third day of hospitalization, CRP increased to 128 mg/L, while amylase decreased to 75 U/L and lipase to 47 U/L. Abdominal ultrasonography revealed increased echogenicity in the pancreatic head consistent with inflammation. Considering the clinical picture, systemic inflammatory response, and CT findings suggesting a possible infectious process, empirical antibiotic therapy was initiated. During follow-up, CRP decreased to 27 mg/L and clinical improvement was observed. The patient was discharged.

Follow-up ultrasonography demonstrated mild intrahepatic biliary duct dilatation and biliary sludge in the gallbladder, while the pancreas appeared normal in size and echogenicity.

Discussion

In this case, limited elevation and rapid decline of pancreatic enzymes, along with progression of inflammatory parameters, resulted in biochemical-inflammatory dissociation. The literature indicates that pancreatic enzyme levels do not always correlate with clinical severity and may decrease in late-presenting cases (2). Therefore, imaging findings should play a central role in the diagnostic process when clinical suspicion persists.

Mesenteric edema and peripancreatic fat stranding observed on contrast-enhanced CT are among the early signs of inflammation (3). In this case, mesenteric inflammatory changes around the inferior pancreas and second portion of the duodenum (Figure 1, Figure 2) supported the diagnosis of acute pancreatitis when evaluated in conjunction with clinical findings.

Routine prophylactic antibiotic use is not recommended in acute pancreatitis. The American College of Gastroenterology (ACG) guidelines recommend antibiotic therapy only in cases of infected pancreatic necrosis or suspected extrapancreatic infection (4). Similarly, IAP/APA guidelines support antibiotic use when infection is suspected (5). In this case, the presence of fever, significant CRP elevation, and CT findings suggesting a possible infectious process clinically justified empirical antibiotic therapy. The subsequent decrease in CRP and clinical improvement support the likelihood of concomitant infection.

Biliary sludge detected on follow-up ultrasonography suggests microlithiasis as a possible etiology. The literature indicates that biliary sludge may play a role in the development of pancreatitis (6). Therefore, etiological evaluation and close follow-up are important in terms of recurrence.

Conclusion

Early regression of pancreatic enzymes does not exclude the diagnosis of acute pancreatitis. Progression of inflammatory parameters and indirect radiological findings such as mesenteric inflammation should be evaluated together with clinical findings. In selected cases with suspected infection, guideline-based antibiotic therapy may be appropriate and contribute to clinical improvement. Although biochemical markers assist in diagnosis, definitive diagnosis relies on clinical presentation and symptomatology. Dissociation between biochemical and inflammatory parameters requires careful diagnostic consideration.

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Keywords
Acute pancreatitis, Early enzyme regression, Inflammatory progression, Mesenteric inflammation, Biochemical dissociation, Biliary sludge

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Ruptured Renal Angiomyolipoma Detected After a Motor Vehicle Accident: A Case-Based Chapter

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Abstract

Renal angiomyolipoma (AML) is a benign mesenchymal renal tumor composed of variable proportions of mature adipose tissue, dysmorphic blood vessels, and smooth muscle. Although many AMLs remain asymptomatic and are detected incidentally, larger or symptomatic lesions may rupture and cause clinically significant retroperitoneal hemorrhage. Trauma may precipitate rupture by disrupting the fragile vascular component of the lesion. This chapter presents the emergency department evaluation and multidisciplinary management of a 61-year-old woman in whom a ruptured right renal AML was detected during whole-body trauma imaging after a motor vehicle accident. The patient was initially conscious and hemodynamically stable, but contrast-enhanced computed tomography demonstrated a 7 x 6 cm heterogeneous right renal mass containing fat-density components, perirenal hematoma, and findings suspicious for active extravasation. The case emphasizes that clinically stable trauma patients may harbor potentially fatal retroperitoneal bleeding, and that serial reassessment, hemodynamic monitoring, hemoglobin surveillance, and early urology consultation are essential. The patient was managed conservatively because there was no persistent hemodynamic instability or significant serial hemoglobin decline. The case also illustrates the need to interpret post-traumatic metabolic acidosis and stress hyperglycemia in context; diabetic ketoacidosis was considered in the differential diagnosis but was not confirmed in the absence of ketone and anion-gap data.

Keywords: renal angiomyolipoma; blunt abdominal trauma; retroperitoneal hemorrhage; Wunderlich syndrome; emergency medicine; multitrauma

1. Introduction

Renal angiomyolipomas are benign mesenchymal tumors arising from perivascular epithelioid cells and classically contain three tissue components: blood vessels, smooth muscle, and fat. Most are sporadic and asymptomatic; however, their abnormal vascular architecture makes them vulnerable to bleeding. When hemorrhage occurs without an external cause, the clinical entity is often described as Wunderlich syndrome, referring to spontaneous non-traumatic renal or perirenal hemorrhage. In the trauma setting, the same fragile vascular

substrate may be disrupted by blunt force, producing retroperitoneal hemorrhage that may initially remain clinically silent.

The traditional 4 cm size threshold has historically been used to identify AMLs at higher risk of hemorrhage, but contemporary evidence emphasizes that size alone is an imperfect predictor. Symptoms, growth rate, intralesional aneurysms, vascularity, pregnancy status, access to emergency care, and patient-specific factors should also inform management decisions. In acutely bleeding AML, transarterial embolization is generally favored when intervention is required because it is minimally invasive and preserves renal parenchyma. Nevertheless, selected hemodynamically stable patients without progressive anemia or ongoing clinical deterioration may be managed conservatively under close monitoring.

This chapter presents a trauma-associated ruptured renal AML detected during emergency department imaging after a motor vehicle accident. The case highlights the diagnostic value of comprehensive trauma imaging and the importance of integrating radiologic findings, serial clinical assessment, and metabolic evaluation before determining the definitive management strategy.

2. Case Presentation

A 61-year-old woman was brought to the emergency department after being involved in a motor vehicle accident as a pedestrian. On arrival to the trauma bay, she was conscious and hemodynamically stable. The primary survey demonstrated preserved airway, breathing, and circulation. Physical examination revealed marked maxillofacial trauma and an obvious deformity of the right wrist.

Given the high-energy mechanism and multisystem physical findings, the patient underwent a comprehensive trauma imaging protocol. Contrast-enhanced computed tomography revealed fractures involving the mandible, bilateral temporomandibular joints, and the L1 vertebral body, together with a right distal radius fracture. More importantly, abdominal CT demonstrated a 7 x 6 cm heterogeneous mass in the middle zone of the right kidney with macroscopic fat-density components. Perirenal hemorrhagic changes and imaging findings suspicious for active contrast extravasation were present, supporting the diagnosis of a ruptured renal AML.

Laboratory evaluation performed in parallel with imaging showed a significant metabolic disturbance. Blood gas analysis and biochemical parameters demonstrated metabolic acidosis with a pH of 7.221 and bicarbonate level of 13.7 mEq/L, accompanied by hyperglycemia with a glucose level of 239 mg/dL. These findings were interpreted in the context of trauma-related stress, possible hypoperfusion, and volume loss. Diabetic ketoacidosis was also considered in the differential diagnosis because of the accompanying hyperglycemia; however, it was not definitively established because serum or urine ketone levels and anion-gap data were not available.

3. Emergency Department Management and Clinical Course

A multidisciplinary management strategy was initiated in the emergency department. Orthopedic consultation was obtained for the extremity injuries, and the right distal radius fracture was stabilized with a splint. The vertebral and maxillofacial injuries were evaluated according to the institutional trauma pathway.

The metabolic derangement was managed with intravenous fluid resuscitation and close biochemical monitoring. Because hyperglycemia was present, diabetic ketoacidosis remained within the clinical differential; however, treatment decisions were guided by serial reassessment and the available laboratory profile rather than by labeling the disorder as confirmed DKA.

Urology was consulted urgently because of the radiologic diagnosis of ruptured AML and the suspected hemorrhagic risk. Despite the lesion size and CT findings concerning for active extravasation, the patient remained hemodynamically stable, and serial hemoglobin measurements did not demonstrate a clinically significant decline. Therefore, immediate surgery or endovascular embolization was deferred. The patient was admitted to the intensive care unit for strict immobilization, close hemodynamic observation, serial abdominal examinations, and repeated laboratory monitoring.

4. Discussion

This case illustrates several clinically important principles in the emergency management of multitrauma patients. First, retroperitoneal bleeding may be occult during the initial assessment, especially when the patient is normotensive and has distracting injuries such as maxillofacial or extremity trauma. The retroperitoneal space can accommodate a substantial volume of blood before overt hemodynamic deterioration occurs. Therefore, mechanism-based imaging remains essential in high-energy trauma, even when the initial examination appears reassuring.

Second, AML rupture is a distinct radiologic and clinical entity. The presence of macroscopic fat within a renal mass strongly supports AML, although fat-poor AML and renal cell carcinoma can create diagnostic uncertainty in selected cases. In this patient, the combination of fat-density components, heterogeneous intralesional structure, perirenal hemorrhage, and suspected extravasation favored ruptured AML.

Third, the management of ruptured AML must be individualized. Hemodynamic instability, ongoing transfusion requirement, progressive hemoglobin decline, expanding hematoma, or definite active arterial bleeding typically favors urgent intervention, most often selective arterial embolization when available. Conversely, conservative management may be appropriate when the patient remains stable, hemoglobin levels are not falling, and bleeding appears contained. This case demonstrates that even a 7 x 6 cm AML with suspected rupture can be observed safely in a carefully monitored setting when the clinical trajectory is favorable.

Finally, the metabolic abnormalities in this patient required careful interpretation. Trauma, pain, catecholamine surge, tissue hypoperfusion, and occult hemorrhage can all contribute to metabolic acidosis and hyperglycemia. Although diabetic ketoacidosis should be considered when metabolic acidosis and hyperglycemia coexist, it should not be definitively diagnosed without supportive ketone and anion-gap data. In emergency medicine, this distinction is clinically relevant because misclassification may lead to inappropriate prioritization or overtreatment. A balanced approach includes simultaneous resuscitation, repeated blood gas and electrolyte assessment, glucose monitoring, and targeted evaluation for ketoacidosis when clinically indicated.

5. Conclusion

Ruptured renal angiomyolipoma is an uncommon but potentially life-threatening finding in trauma patients. Hemodynamic stability at presentation does not exclude clinically important retroperitoneal hemorrhage. Comprehensive trauma imaging, early multidisciplinary consultation, serial hemoglobin monitoring, and repeated clinical reassessment are essential for safe decision-making. In carefully selected stable patients, conservative management with intensive monitoring may be appropriate, whereas unstable patients or those with ongoing bleeding should be considered for selective arterial embolization or surgery. This case also underlines the importance of evaluating metabolic acidosis and hyperglycemia in the broader trauma context rather than prematurely assigning a single diagnosis.

Figures

Figure 1. Axial contrast-enhanced abdominal computed tomography images showing a heterogeneous right renal mass with macroscopic fat-density components, perirenal hemorrhagic changes, and findings suspicious for rupture of renal angiomyolipoma. (A-B) Consecutive axial slices demonstrating the renal lesion and surrounding retroperitoneal hematoma.

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3817

A rare cause of resistant headache in emergency room

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Keywords: pituitary adenoma, thunderclap headache, emergency department, sellar mass, transsphenoidal surgery

Introduction and aim

Headache account for approximately 2–3% of visits to the emergency department (ED).[1,2,3]While most cases are benign primary headaches, secondary causes, some of which are life-threatening, must not be overlooked. Pituitary adenomas account for 10–15% of intracranial tumors[4] and can lead to severe headaches by stretching the dural structures around the sella turcica.[5,6] We present a case in which a normal neurological examination was nearly misleading, but clinical red flags led to prompt imaging and a timely diagnosis.

Materials and methods

A 56-year-old male presented to the ED at night with a sudden-onset headache that progressively worsened, accompanied by nausea. He described it as the worst headache of his life and stated that he had experienced no similar episodes previously, had suffered no recent trauma and had no infectious prodrome.

Vital signs were stable. Neurological examination, including visual acuity and visual fields, revealed no focal deficits. However, the thunderclap onset and age over 50 were flagged as red flags warranting further investigation. Non-contrast cranial computed tomography (CT) and CT angiography were initially performed to rule out hemorrhage. Contrast-enhanced pituitary magnetic resonance imaging (MRI) with diffusion sequences were then requested to characterize the lesion.

Results and conclusion

CT revealed a sellar mass measuring approximately 20×28 mm expanding the sellar cavity and extending into the right sphenoid sinus (Figure 1). CT angiography excluded vascular pathology. The MRI scan revealed a 33×26×22 mm lesion transgressing the sella, invading both cavernous sinuses predominantly on the right(Figure 2,3,4). There was minimal central enhancement and narrowing of the prepontine cistern, which is consistent with a pituitary adenoma. As part of the endocrinological assessment, a baseline anterior pituitary hormone

panel was obtained and the preoperative prolactin level was measured at 54.50 ng/mL, which is consistent with mild to moderate hyperprolactinemia.

The treatment of pituitary adenomas is guided by tumor size, growth pattern and hormonal activity.[4] Hormonally active tumors, such as prolactinomas, generally respond to dopamine agonist therapy and surgery is not considered the primary approach. In contrast, macroadenomas that exert a mass effect on the optic chiasm or that invade the cavernous sinus, as well as tumors that secrete growth hormone or adrenocorticotropic hormone (ACTH), are primarily managed surgically. In this case, the size of the lesion and its anatomical location left little room for a conservative approach, and early surgery was justified by the need to prevent progressive visual loss and hypopituitarism.

The patient underwent a joint evaluation by the neurosurgery, neurology, endocrinology and ophthalmology departments on the same day, was admitted to the neurosurgery ward and underwent transsphenoidal hypophysectomy without complications. The final histopathological examination of the surgical specimen confirmed the diagnosis. Immunohistochemical staining was positive for both synaptophysin and prolactin, definitively establishing a diagnosis of prolactinoma.

Pituitary adenomas can closely mimic primary headache syndromes. The pain arises from the trigeminal afferents within the diaphragma sellae and the surrounding dura, and it can present as frontal or retro-orbital pain that is indistinguishable from migraine.[5,6] A normal neurological examination is not sufficient grounds for excluding significant intracranial pathology.[1,2,3] Advanced imaging, including MRI, is essential in patients with sudden, severe headaches and red flags. Although CT is useful for detecting mass effect and excluding hemorrhage, MRI remains the gold standard for characterizing sellar lesions.

In this case, the mildly elevated preoperative prolactin level, together with positive synaptophysin and prolactin immunoreactivity in the pathological examination, provided biochemical and histological confirmation of a prolactinoma. This highlights that even hormonally active adenomas may require surgical treatment if mass effect is present. This case demonstrates that, when patients present to the ED with a severe headache for the first time or a thunderclap headache, recognizing clinical red flags should prompt advanced neuroimaging without hesitation. Timely multidisciplinary management can directly translate into a favorable outcome.

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Figure 1. Axial Cranial CT

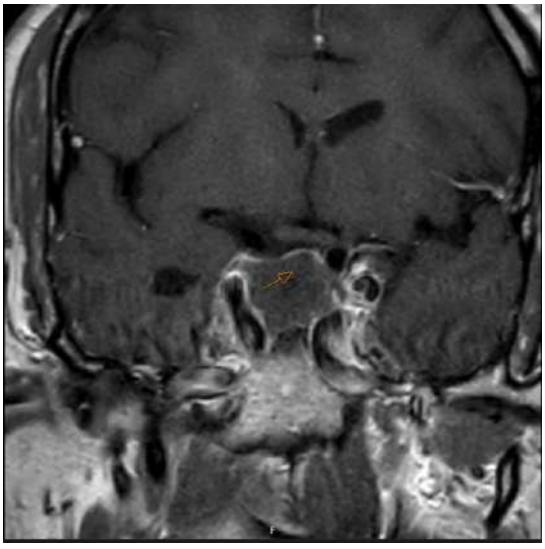

Figure 2. T1 weighted coronal MRI

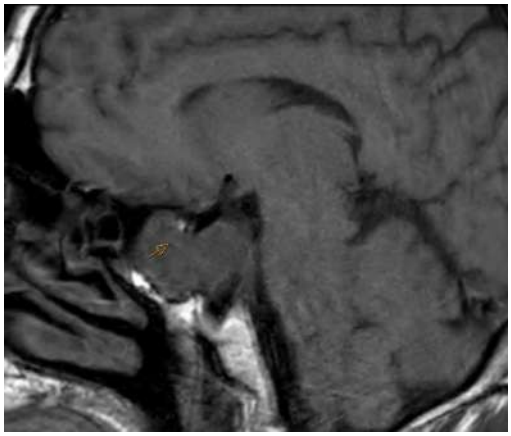

Figure 3. T1 weighted sagittal MRI

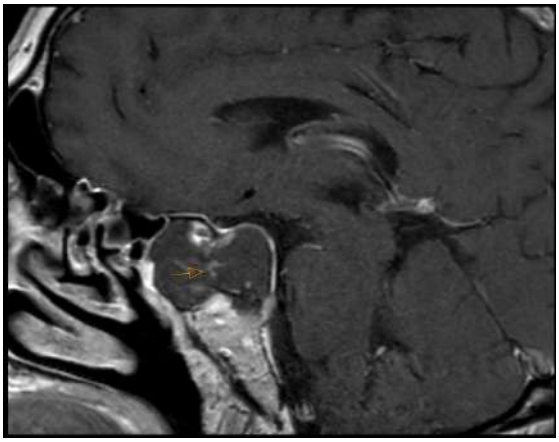

Figure 4. T2 weighted sagittal MRI

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Association Between CRP/Albumin Ratio and Hospitalization in Patients with Cellulitis Presenting to the Emergency Department

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Abstract

Introduction:

Cellulitis is an acute bacterial infection of the dermis and subcutaneous tissue and is commonly encountered in the emergency department. It most frequently affects the lower extremities and presents with erythema, increased temperature, edema, and tenderness. The most common causative agents are beta-hemolytic streptococci and *Staphylococcus aureus*. Although hospitalization rates have increased in recent years, the need for admission is not always clearly defined. Clinical decisions are typically based on a combination of clinical findings, comorbidities, and laboratory data. However, the role of routine laboratory parameters in predicting hospitalization remains unclear.

Objective:

To investigate the association between routine laboratory parameters, particularly the C-reactive protein/albumin ratio (CAR), and hospitalization in patients with cellulitis.

Materials and Methods:

This retrospective observational study included patients diagnosed with cellulitis in the emergency department between November 1, 2025, and February 1, 2026. Demographic data and laboratory parameters, including white blood cell (WBC), neutrophil (NEU), albumin, CRP, and CAR, were recorded. Patients were divided into hospitalized and non-hospitalized

groups. Statistical analyses were performed using IBM SPSS 27, and the diagnostic performance of CAR was evaluated using ROC curve analysis.

Results:

A total of 43 patients were included, of whom 20.9% were hospitalized. WBC and neutrophil levels were significantly higher in hospitalized patients ($p<0.001$). While CRP and albumin alone were not significantly associated with hospitalization ($p>0.05$), CAR showed a significant association ($p=0.045$). The area under the ROC curve for CAR was 0.719, indicating moderate discriminative ability, with an optimal cut-off value of approximately 2.13.

Conclusion:

CAR may be a practical and easily applicable marker for predicting hospitalization in cellulitis patients and may provide greater clinical value than individual parameters.

Keywords:

Cellulitis, Emergency Department, CRP/Albumin Ratio; Hospitalization

Introduction

Cellulitis is an acute bacterial infection of the dermis and subcutaneous soft tissues and is among the most frequently encountered skin and soft tissue infections in the emergency department [1,2]. Clinically, it is most commonly characterized by erythema, increased local temperature, edema, and tenderness, with lower extremity involvement being particularly prominent [2,3]. The most common causative agents are beta-hemolytic streptococci and *Staphylococcus aureus*; however, patient characteristics, accompanying comorbidities, and local resistance patterns may directly influence treatment strategies [1,2].

In recent years, skin and soft tissue infections have imposed a considerable burden on both outpatient clinics and emergency departments. As emphasized in the IDSA guideline, emergency department visits and hospital admissions related to these infections have increased significantly over time [1]. Cellulitis also represents an important cause of hospitalization associated with a substantial economic burden on healthcare systems [4].

Nevertheless, determining which patients can be safely managed as outpatients and which require hospitalization remains unclear in daily clinical practice [3,4].

Current guidelines recommend considering hospitalization in cases of systemic signs of infection, hemodynamic instability, altered mental status, suspicion of deep or necrotizing infection, immunosuppression, and failure of outpatient therapy [1,5]. However, in real-world clinical practice, hospitalization decisions are made not only according to physical examination findings but also based on the extent of infection, accompanying comorbidities, and laboratory findings [3,6]. In particular, whether blood parameters obtained at initial presentation in the emergency department can help predict disease severity and the need for hospitalization is of clinical importance.

Several clinical and biochemical markers associated with poor clinical outcomes or outpatient treatment failure in cellulitis patients have been described in the literature. The presence of fever, chronic edema/lymphedema, chronic ulceration, and a history of cellulitis in the same region have been reported to be associated with treatment failure [6]. Similarly, elevated lactate levels, objective fever, and hand-localized infections have been shown to predict the need for hospitalization in patients managed in observation units [7]. In addition, systems such as the Dundee severity classification have been proposed as practical tools for predicting mortality, length of hospital stay, and readmission risk in cellulitis cases [8]. However, despite these classifications, the role of routine hemogram and biochemical parameters in hospitalization decisions has not yet been fully clarified.

Therefore, in this study, we aimed to investigate the relationship between certain blood parameters measured at admission and hospitalization among patients diagnosed with cellulitis who presented to Malatya Training and Research Hospital between November 1, 2025, and February 1, 2026. We believe that demonstrating the value of rapid, accessible, and low-cost laboratory markers in predicting hospitalization needs may contribute to the clinical decision-making process in the emergency department.

Materials and Methods

This study was designed as a retrospective observational study evaluating patients diagnosed with cellulitis who presented to the Emergency Department of Malatya Training and Research Hospital between November 1, 2025, and February 1, 2026.

Patients clinically diagnosed with cellulitis in the emergency department and whose demographic and laboratory data were fully accessible through the hospital information system were included in the study. Patients with missing data were excluded.

Age (years) and sex information were recorded for all patients. Laboratory parameters measured at admission included white blood cell count (WBC, $\times 10^9$/L), neutrophil count (NEU, $\times 10^9$/L), albumin (g/dL), and C-reactive protein (CRP, mg/L). Additionally, the CRP/albumin ratio (CAR) was calculated and included in the analyses.

The primary endpoint was hospitalization status, and patients were divided into two groups as “hospitalized” and “non-hospitalized.”

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics 27.

The normality of continuous variables was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Variables with normal distribution were expressed as mean ± standard deviation, whereas variables without normal distribution were expressed as median (IQR: 25th–75th percentile).

For intergroup comparisons, the independent samples t-test was used for normally distributed variables, while the Mann–Whitney U test was used for non-normally distributed variables. Categorical variables were presented as number (n) and percentage (%) and compared using appropriate statistical tests.

Correlation analyses were performed to evaluate the relationship between hospitalization and laboratory parameters.

Receiver Operating Characteristic (ROC) curve analysis was conducted to evaluate the diagnostic performance of the CRP/albumin ratio in predicting hospitalization, and the area under the curve (AUC) was calculated.

A p-value <0.05 was considered statistically significant for all analyses.

Results

A total of 43 patients were included in the study, and complete data from all patients were analyzed. The mean age of the patients was 69.44±12.56 years, while the median age was 71 (IQR: 64–77). Regarding sex distribution, 60.5% of the patients were male (n=26) and 39.5% were female (n=17).

Evaluation of hospitalization status revealed that 20.9% of the patients (n=9) were hospitalized, whereas 79.1% (n=34) were managed as outpatients.

For the parametric variable albumin, the mean ± standard deviation was 3.07±0.76.

The median and IQR values of the non-parametric variables were as follows: age 71 (64–77), WBC 7.2 (5.1–9.2), NEU 5.1 (3.5–7.4), CRP 8.3 (4.9–11.2), and CRP/albumin 2.9 (1.4–4.0). Detailed results are presented in Table 1.

Table 1. Mean, SD, Median, and IQR Values of the Study Variables

					IQR	
Variables	n	Mean	SD	Median	0,25	0,75
Age	43			71	64	77
Wbc				7.2	5.1	9.2
Neu				5.1	3.5	7.4
Crp				8.3	4.9	11.2
Crp/albumin				2.9	1.4	4.0
Albumin		3.07	0.76			

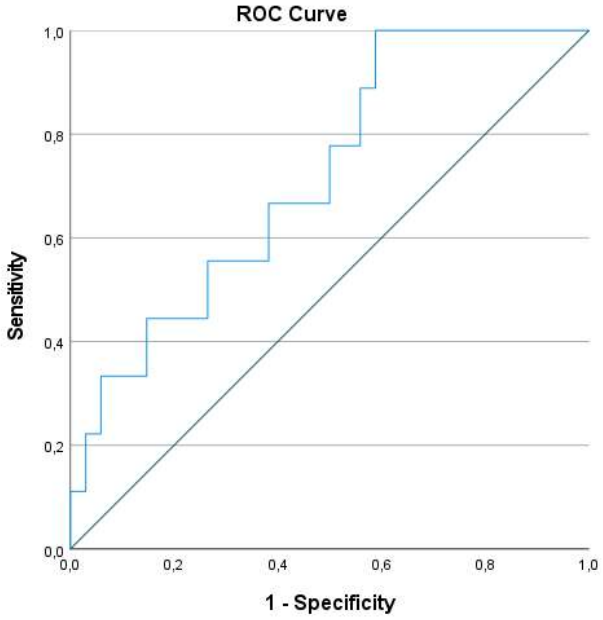
In subsequent analyses, patients were compared according to hospitalization status. Independent samples t-test analysis demonstrated no statistically significant difference in albumin levels between hospitalized and non-hospitalized groups (p=0.078).

In the Mann–Whitney U analysis performed for non-normally distributed variables, white blood cell count (WBC, $\times 10^9$/L) and neutrophil count (NEU, $\times 10^9$/L) were found to be significantly higher in the hospitalized group ($p<0.001$ and $p<0.001$, respectively). The CRP/albumin ratio (CAR) was also higher in the hospitalized group and was statistically significant (p=0.045). In contrast, no significant differences were observed between the groups in terms of CRP (mg/L) and age (years) (p=0.135 and p=0.940, respectively).

The diagnostic performance of the CRP/albumin ratio in predicting hospitalization was evaluated using ROC curve analysis. The area under the curve (AUC) was calculated as 0.719 (95% CI: 0.544–0.894; p=0.045).

When determining the cut-off point, the value with the highest Youden index was selected, considering that sensitivity and specificity could not simultaneously equal 1. Accordingly, the optimal threshold value was determined to be approximately 2.13, with a sensitivity of 88.9% and specificity of 41.2%. The ROC curve is presented in Figure 1.

Figure 1. ROC Curve Analysis of CAR Value in Relation to Hospitalization



Discussion

In our study, a considerable proportion of patients presenting to the emergency department with cellulitis were in the advanced age group, and approximately one-fifth required hospitalization. Evaluation of laboratory parameters demonstrated that white blood cell count (WBC) and neutrophil count (NEU) were significantly associated with hospitalization, whereas CRP and albumin levels alone did not show a significant association. Notably, the CRP/albumin ratio (CAR), which combines these two parameters, was significantly associated with hospitalization and demonstrated moderate diagnostic performance.

Although studies investigating predictors of hospitalization in skin and soft tissue infections are limited, inflammatory markers reflecting infection severity have been reported to be associated with clinical outcomes [1,2]. In particular, leukocytosis and neutrophilia in acute infections have been shown to be associated with more severe clinical presentations and increased hospitalization requirements [6]. However, CRP alone may be insufficient in predicting clinical outcomes, and combined markers that better reflect systemic inflammation may provide greater clinical value [9]. Indeed, recent studies have demonstrated that the CRP/albumin ratio is associated with mortality and poor clinical outcomes in sepsis and various infectious conditions [10].

Consistent with these findings, our study demonstrated that WBC and neutrophil values were strongly associated with hospitalization requirements, whereas CRP and albumin levels alone were not statistically significant. In contrast, the significance of the CAR parameter suggests that evaluating inflammatory response together with both positive and negative acute-phase reactants may provide a more meaningful assessment.

Based on these findings, we believe that easily accessible and rapidly obtainable hemogram parameters, along with simple and calculable ratios such as CAR, may assist in predicting hospitalization needs in patients with cellulitis in the emergency department. Nevertheless, these parameters should be used in conjunction with clinical evaluation rather than as standalone decision-making tools.

Conclusion

In this study, WBC, neutrophil count, and CRP/albumin ratio were found to be associated with hospitalization in patients with cellulitis. While CRP and albumin alone were not significant, their combined parameter, CAR, was identified as a more meaningful marker. These easily obtainable parameters in the emergency department may support hospitalization decisions in addition to clinical evaluation.

Limitations

This study was conducted as a single-center retrospective study, and the limited number of patients restricts the generalizability of the findings. Additionally, the absence of clinical severity scores and comorbid diseases in the analyses may have affected the interpretation of the results.

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Clinical Characteristics of Suicide-Related Drug and Toxic Substance Ingestions Presenting to the Emergency Department in 2024: A Retrospective Study

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Introduction: Acute poisonings are among the leading causes of emergency department admissions worldwide and are associated with significant morbidity and mortality. In particular, suicide-related drug and toxic substance ingestions are considered medical emergencies requiring rapid clinical evaluation and appropriate treatment. Determining the demographic and clinical characteristics of these cases is important for identifying risk factors and improving clinical management.

Materials and Methods: In this retrospective study, adult patients presenting to the emergency department of Malatya Training and Research Hospital between January 1 and December 31, 2024, due to suicide-related drug or toxic substance ingestion were evaluated. Patients were selected from cases recorded with the ICD-10 diagnosis code X44 in the hospital information system. Demographic characteristics, types of toxic agents ingested, treatment methods applied, and clinical outcomes were analyzed. Statistical analyses were performed using IBM SPSS Statistics version 27.0.

Results: A total of 90 patients who met the inclusion criteria were evaluated. Of the patients, 85.6% were female. The most frequent admissions occurred in winter (35.6%) and summer (30%). Psychiatric drugs (35.6%) were the most commonly misused agents, followed by analgesics (21.1%) and multiple drug ingestions (16.7%). Supportive treatment was administered to 54.4% of the patients, while activated charcoal, gastric lavage, and antidote

therapies were used in some cases. A total of 76.7% of patients were followed in inpatient wards and 23.3% in intensive care units, and all patients were discharged with full recovery.

Conclusion: Suicide-related toxic substance ingestions are common clinical conditions encountered in the emergency department. Early evaluation, appropriate supportive treatment, and close clinical monitoring play an important role in achieving favorable clinical outcomes.

Keywords: acute intoxication, suicide attempt, emergency department, drug poisoning

INTRODUCTION

Acute poisonings constitute a significant proportion of emergency department admissions worldwide and are recognized as an important public health problem (1). In particular, the intentional ingestion of drugs and toxic substances represents a medical emergency requiring rapid clinical assessment and appropriate management (2). The clinical presentation may vary depending on the type and dose of the ingested substance, as well as the patient's comorbid conditions, and may lead to serious complications in some cases (2,3). Recent epidemiological studies have demonstrated that intentional poisoning attempts are more frequently observed, especially among young adult women (4,5). Furthermore, antidepressants, antipsychotics, and analgesics have been reported as the most commonly implicated agents in a substantial proportion of these cases (4,6). Therefore, evaluating the demographic and clinical characteristics of patients presenting to the emergency department with intentional toxic substance ingestion is crucial for identifying risk factors and improving clinical management.

MATERIALS AND METHODS

Inclusion Criteria: Patients in the adult age group (≥18 years) who presented to the emergency department due to intentional ingestion of drugs, corrosive substances, or other toxic agents and were subsequently hospitalized; patients whose data were retrospectively

accessible through the hospital information system; and patients admitted between January 1, 2024, and December 31, 2024.

Exclusion Criteria: Patients under 18 years of age; patients with incomplete data in the hospital information system; and patients who were transferred from the emergency department to other hospitals or who left the hospital against medical advice without accepting hospitalization.

Patient Selection

A total of 96 patients recorded in the Malatya Training and Research Hospital Akgün information system between January 1, 2023, and December 31, 2023, with the ICD-10 diagnosis code X44 (accidental poisoning by and exposure to drugs, medicaments, and biological substances, other) were identified. The epicrisis reports available in the Akgün information system were reviewed in detail. Six patients who did not meet the inclusion criteria were excluded. The study was conducted with the data of 90 patients who met the eligibility criteria.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, New York, USA). Descriptive statistical methods were expressed as mean, median, standard deviation, percentage, interquartile range [25–75% (IQR)], and frequency.

RESULTS

The study was conducted with 90 patients who met the inclusion criteria. Of these, 77 patients were female (85.6%).

A total of 32 patients (35.6%) presented to the emergency department during the winter months, 27 (30%) during the summer, 18 (20%) during the autumn, and 13 (14.4%) during the spring.

Regarding the type of exposure, 32 patients (35.6%) had ingested psychiatric medications (antidepressants, antipsychotics), 19 (21.1%) analgesics (NSAIDs and paracetamol), 15 (16.7%) multiple drugs (including analgesics, antibiotics, antihistamines, muscle relaxants, gastroprotective agents, and psychiatric medications), 8 (8.9%) corrosive substances, 7 (7.8%) antibiotics alone, 5 (5.6%) pesticides, and 2 (2.2%) alcohol.

In terms of management, 49 patients (54.4%) received general supportive care. Activated charcoal was administered to 14 patients (15.6%), while 22 patients (24.4%) received activated charcoal following gastric lavage. Three patients received specific antidote therapy, and two patients underwent gastric lavage alone.

A total of 69 patients (76.7%) were followed up in the internal medicine ward, whereas 21 patients (23.3%) were admitted to the intensive care unit. All patients were discharged with full recovery.

DISCUSSION

In this study, the demographic characteristics, distribution of causative agents, and treatment approaches of patients presenting to the emergency department in 2024 with intentional drug and toxic substance ingestion were evaluated. Our findings demonstrated that the majority of cases were female patients and that psychiatric medications constituted the most common group of agents. Consistent with the literature, intentional poisoning attempts have been reported to occur more frequently among young adult women, with medications being the most commonly used toxic agents (7,8). Furthermore, several epidemiological studies have shown that psychiatric drugs and analgesics represent a substantial proportion of poisoning cases presenting to the emergency department (7,9).

In our study, supportive care was sufficient in a considerable proportion of patients, while a smaller number required activated charcoal, gastric lavage, or antidote therapy. The literature

emphasizes that the cornerstone of acute poisoning management includes stabilization of vital signs, close clinical monitoring, and the application of decontamination methods when indicated (9,10). Particularly in patients presenting early, appropriate decontamination strategies may reduce the systemic effects of the toxic substance (10,11).

The fact that the majority of patients in our study were managed in general wards, with a smaller proportion requiring intensive care, highlights the importance of early assessment and appropriate treatment in determining clinical outcomes. However, the clinical course may be severe in certain poisoning cases, necessitating intensive care support (12,13). Therefore, identifying the epidemiological characteristics of intentional toxic substance ingestion, defining high-risk patient groups, and improving clinical management strategies may provide significant contributions to patient care in the emergency setting.

CONCLUSION

In 2024, female patients and psychiatric medications were predominant among cases of intentional toxic substance ingestion presenting to the emergency department. Early assessment, appropriate supportive care, and careful clinical monitoring play a crucial role in achieving favorable clinical outcomes in these patients.

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Role of Acute Phase Reactants and Demographic Data in Predicting Mortality and Prognosis in Patients with Meningitis

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Introduction

Meningitis, the incidence of which varies by region, still emerges as a significant health problem in low- and middle-income countries. One of the most critical parameters determining the prognosis is early diagnosis and the timely initiation of antimicrobial therapy. Laboratory parameters play a vital role in establishing an early diagnosis.

Objective

In our study, we aimed to determine the predictability of mortality and prognosis in patients with meningitis in the light of demographic characteristics and acute phase reactants.

MATERIALS AND METHODS

Study Design and Population

This study was conducted through a retrospective analysis of data from 41 patients who were hospitalized and treated with a preliminary diagnosis of meningitis at Malatya Training and Research Hospital between December 2025 and March 2026.

Data Collection and Parameters

Patient data were obtained by scanning the hospital automation system and patient files. The following data of all patients included in the study were recorded: age and sex, serum C-reactive protein (CRP) and albumin levels at admission, total length of hospital stay (days), and in-hospital mortality status.

The diagnosis of meningitis was established based on clinical findings (fever, nuchal rigidity, altered mental status), direct examination of cerebrospinal fluid (CSF), biochemical analyses, and culture results.

Statistical Analysis

Statistical analyses were performed using SPSS v26.0 software. The conformity of data to normal distribution was evaluated using the Shapiro-Wilk test. The Independent Samples t-test was used to compare continuous variables with normal distribution, while the Mann-Whitney U test was used for non-normally distributed data. To examine the correlation between variables, Pearson or Spearman correlation analysis was applied according to the distribution status of the data.

Findings

The mean age of the 41 patients included in the study was determined as 68.02±14.40 years. When the sex distribution of the patients was examined, 13 (31.7%) female patients were identified. When the laboratory data of the entire group were analyzed, the median CRP level was found to be 8.5 mg/L (IQR 25–75: 5.1–16.35). The albumin level was detected as 2.94±0.52g/dL. The median length of hospital stay was 7 (IQR 25–75: 5–8) days. During the study period, mortality was observed in 4 patients (9.7%).

- The mean rank of CRP in patients with mortality (mean rank: 37.75) was found to be significantly higher than that of discharged patients (mean rank: 19.19) (Mann-Whitney U = 7.000; p = 0.013).
- The length of hospital stay for cases resulting in mortality was statistically significantly longer compared to survivors (Mann-Whitney U = 0.500; p = 0.001).
- A positive, moderate, and statistically significant correlation was found between CRP levels and the length of hospital stay (r = 0.457; p = 0.003).
- A positive, moderate, and significant correlation was found between age and mortality (r = 0.317; p = 0.043).

Conclusion

This study indicates that advanced age and a high systemic inflammatory response are directly associated with mortality in patients diagnosed with meningitis. It demonstrated that high CRP levels serve as a statistically significant and strong biomarker in predicting both mortality risk and the length of hospital stay. The low albumin levels and high mean age in our patients hold an important place in determining the prognosis. In conclusion, elderly male patients with a high inflammatory load require closer monitoring.

Discussion

In our study, the mean age of our patients was determined as 68.02±14.40 years. In consistency with our study, there are studies in the literature showing that meningitis cases are seen more frequently at advanced ages (1). The higher frequency of meningitis in the elderly population can be explained by the immunity of the younger age group against relevant agents due to vaccines included in routine immunization programs, and the weakened general immunity in the advanced age group.

When the sex distribution of our patients was examined, a male predominance (68.3%) was observed. Male patient rates close to the 68.3% rate detected in our study are also available in similar retrospective studies (2). We think this situation may be related to males being exposed to pathogens causing meningitis more than females due to their social roles or outdoor activities.

When the laboratory data of all our patients were analyzed, the median CRP level was found to be 8.5 mg/L (IQR 25–75: 5.1–16.35). The increased CRP levels detected in our study parallel current studies (3). Elevated CRP can be explained by the pathogen causing meningitis also stimulating systemic inflammatory processes.

Albumin levels were found to be 2.94 ± 0.52 g/dL in our patients. Albumin, a negative acute phase reactant, was found to be low, in parallel with studies similar to ours (4). Increased vascular wall permeability during infection and decreased albumin production in the liver during infection explain the low course of albumin in meningitis.

The median length of hospital stay for the patients was 7 (IQR 25–75: 5–8) days. The length of hospital stay in our patients is shorter compared to the literature (5). This shorter length of stay is thought to stem from early diagnosis leading to prompt initiation of treatment, or a lower incidence of neurological complications.

In our study, 4 patients (9.7%) resulted in mortality, and this rate is in parallel with the literature (5). Since the presentation of meningitis creates a severe inflammatory response, it can cause mortality in some of the patients.

In patients with a mortal course, the CRP value was found to be significantly higher than that of discharged patients (Mann-Whitney U = 7.000; p = 0.013). CRP has also been found higher in meningitis patients in current studies (5). This situation can be explained by the systemic inflammatory response elicited by meningitis in the body and the subsequent increase in acute phase reactants.

The length of stay for cases resulting in mortality was statistically significantly longer compared to survivors (Mann-Whitney U = 0.500; p = 0.001). In current studies, the length of

hospital stay also shows a significant relationship with mortality (5). This can be explained by the lower response to treatment in patients with a longer length of hospital stay.

A positive, moderate, and statistically significant correlation was found between CRP levels and the length of stay in our patients ($r = 0.457$; $p = 0.003$). The significant relationship of the length of hospital stay with CRP levels shows similarity with other studies in the literature (1). This situation can be explained by CRP levels reflecting the systemic inflammatory status and high inflammation prolonging the hospital stay.

A positive, moderate, and significant correlation was found between the age of our patients and mortality ($r = 0.317$; $p = 0.043$). This situation has also been found consistent in conducted studies (6). The high mortality rate in advanced age stems from the weakening of the immune system with age.

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Flail chest

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Introduction and Purpose: Flail chest, also known as 'flail chest', refers to blunt chest trauma. This type of chest injury, characterized by fractures in two or more adjacent ribs, occurs particularly after high-energy trauma such as severe traffic accidents, falls, or collisions.

Materials and Methods: A 79-year-old female patient was brought to the emergency department by ambulance after a non-vehicular traffic accident. Her medical history included hypertension and rheumatoid arthritis. Blood pressure measured at presentation was 110/50, respiratory rate: 18, saturation: 95, conscious, general condition fair. Abdominal examination was unremarkable, nm: normal, ss: rales present in right lung sounds. Laboratory values for our patient were ast: 165, alt: 168, wbc: 9830, hgb: 12.5. The aim of this study is to emphasize that early and supportive treatment is extremely important in cases where flail chest is detected. Otherwise, more serious problems that cause life-threatening complications may arise, and if treatment is delayed, cases may result in death. Imaging performed on our patient (Figures 1-2) revealed multiple rib fractures and accompanying hemothorax in the right hemithorax.Following this, the patient was referred to thoracic surgery and intensive care, and admitted to intensive care for further examination and treatment.

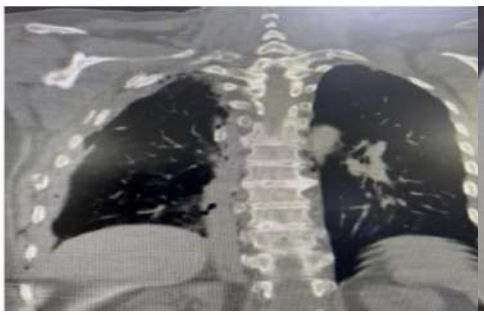

Figure 1

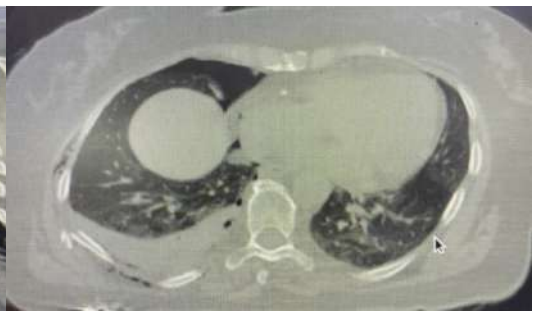

Figure 2

Results and Conclusion: As is well known, flail chest cases are easily recognizable by paradoxical chest movements. Approximately 5 to 15 percent of chest wall injuries develop into flail chest. This disease, characterized by the chest wall moving inward during inhalation and outward during exhalation, can be detected by symptoms such as respiratory distress. In some patients, this disease can be diagnosed by advanced imaging tests even without these

symptoms. Indeed, in our case, although paradoxical breathing had not yet developed, the diagnosis was made by imaging and clinical examination. Patients with flail chest are at high risk of developing life-threatening complications such as pneumothorax, hemothorax, and shock. Protecting the airway is crucial in the initial intervention. Chest stabilization, early diagnosis, and early intervention are of vital importance in these patients.

Keywords: flail chest, hemothorax, pneumothorax

4035 **MERS Triggered by Recurrent Seizures in an Epileptic Adult**

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Abstract

Mild Encephalitis/Encephalopathy with a Reversible Splenial Lesion (MERS) is a rare clinico-radiological syndrome typically reported in pediatric populations, characterized by transient lesions in the splenium of the corpus callosum. Adult cases are uncommon and are generally precipitated by infections, metabolic disturbances, or seizures. We report the case of a 28-year-old woman with a known history of epilepsy who presented to the emergency department with recurrent seizures and secondary head trauma. Brain imaging revealed diffusion-restricted lesions in the splenium, consistent with MERS. The patient was successfully managed with anti-seizure medications and supportive care, achieving rapid clinical stabilization. This case highlights the rare association of recurrent epileptic seizures with MERS in adults and underscores the importance of recognizing this reversible entity to prevent unnecessary interventions. In these patients, MRI remains the gold standard for diagnosis. With seizure control and supportive care, patients generally show rapid recovery.

Keywords: Epilepsy, generalized seizures, MERS

Introduction

Mild Encephalitis/Encephalopathy with a Reversible Splenial Lesion (MERS) is a transient condition initially described in children. Its hallmark radiological feature is a reversible lesion in the splenium of the corpus callosum on diffusion-weighted MRI (1). Clinical presentations vary, often including altered consciousness, seizures, or headaches. While infectious and metabolic etiologies are frequently implicated, seizure-induced MERS in adults remains exceedingly rare (2). Heightened awareness of this clinico-radiological syndrome is critical to avoid misdiagnosis and overly aggressive treatments.

Case Report

A 28-year-old woman with an established diagnosis of epilepsy presented following head trauma sustained during a cluster of seizures. She reported experiencing 7–8 generalized tonic-clonic seizures earlier that day, noting a gradual increase in seizure frequency over the preceding two months. Her sole daily medication was levetiracetam (1500 mg/day), with no other substance use or comorbidities reported. Upon initial examination, the patient was alert (GCS 15), fully oriented, and exhibited no focal neurological deficits. Initial management included intravenous fluid resuscitation and 3000 mg of IV levetiracetam. During clinical observation, she experienced a breakthrough seizure. This was promptly aborted with 10 mg of IV diazepam, followed by 10 mg of IV midazolam, which achieved complete seizure control.

Laboratory Findings: WBC 12,000 /µL, CRP 7 mg/L, pH 7.44, Lactate 0.8 mmol/L, Sodium 138 mmol/L. Systemic infection and metabolic derangement were ruled out. Diffusion-weighted MRI demonstrated a diffusion-restricted, hyperintense lesion in the splenium of the corpus callosum, consistent with MERS (Figure 1). A non-contrast CT showed a subtle hypodense area in the same region, suggestive of cytotoxic edema (Figure 2).

Figure 1. Diffusion-weighted MRI demonstrating hyperintense lesion in the splenium of the corpus callosum.

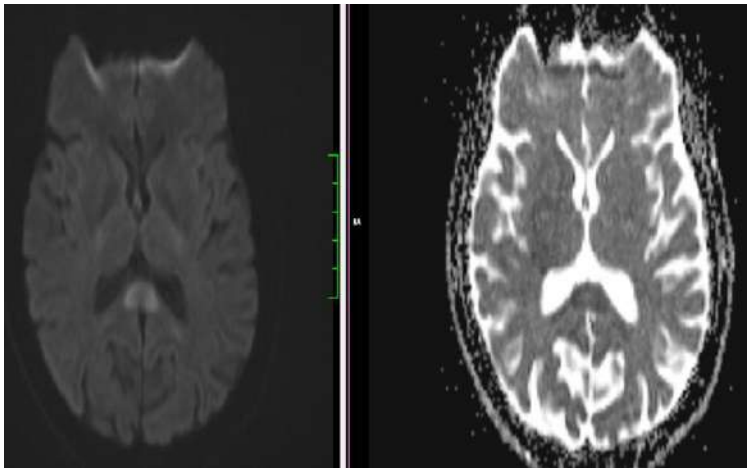
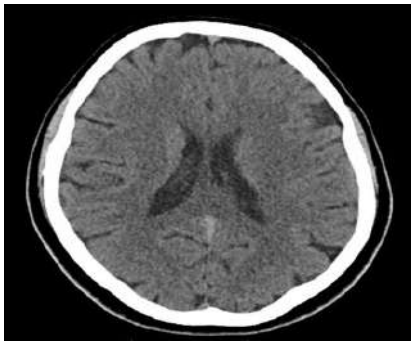


Figure 2. Non-contrast CT showing subtle hypodense area in the splenium.



Discussion

MERS is defined by its characteristic transient splenial lesions and typically follows a spontaneous resolution (3). The underlying pathophysiology is hypothesized to involve transient intramyelinic edema secondary to excitotoxicity, prolonged seizure activity, or metabolic stress. In adult populations, seizure-induced MERS is remarkably scarce (4). In our patient, the unusually high frequency of recurrent seizures likely acted as the primary trigger for the splenial lesion, especially after laboratory results safely excluded systemic infections or severe metabolic imbalances.

Furthermore, early identification via diffusion-weighted MRI is vital to differentiate this reversible condition from acute ischemia or demyelinating diseases. Prompt seizure control remains paramount, as it halts ongoing excitotoxicity and facilitates the rapid resolution of the splenial edema.

Conclusion

Recurrent seizures in epileptic patients can, albeit rarely, precipitate MERS in adulthood. Early recognition of this specific radiological pattern is essential for appropriate patient management and the avoidance of invasive diagnostic procedures. MRI remains the gold standard for diagnosis, and with optimal seizure control and supportive care, patients generally achieve a full and rapid recovery.

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4087

The Hidden Cause of Syncope: A Case of Subclavian Steal Syndrome in the Emergency Department

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Introduction

Subclavian steal syndrome is a vascular disorder characterized by a compensatory alteration in blood flow, in which blood is diverted from the cerebral and vertebrobasilar circulation to meet the metabolic demands of the upper extremity in the presence of subclavian artery obstruction (1). The subclavian artery is the main arterial supply of the upper limb and also gives rise to the vertebral arteries, which constitute an important component of the posterior cerebral circulation. Subclavian steal syndrome most commonly develops as a consequence of severe stenosis or complete occlusion of the subclavian artery proximal to the origin of the vertebral artery. This pathological process generates a significant intravascular pressure gradient. The pressure distal to the stenotic subclavian segment becomes lower than the pressure transmitted from the contralateral vertebral artery through the basilar system and the circle of Willis. As a result, blood flow is redirected in a retrograde manner through the ipsilateral vertebral artery to maintain upper extremity perfusion. This hemodynamic mechanism leads to diversion of blood away from the posterior cerebral circulation, which is referred to as the “steal” phenomenon (2,3). When blood flow is preferentially directed from the posterior circulation toward the upper extremity, particularly during arm exertion, vertebrobasilar perfusion may be compromised and clinical symptoms may develop (4,5). It is important to distinguish subclavian steal phenomenon from subclavian steal syndrome. The demonstration of retrograde vertebral artery flow on Doppler ultrasonography alone is defined as subclavian steal phenomenon, whereas the coexistence of neurological symptoms or upper extremity ischemic manifestations indicates subclavian steal syndrome (5,6). Atherosclerosis is the most common cause of subclavian artery stenosis. Less frequently, Takayasu arteritis, radiation-induced vasculopathy, trauma, congenital vascular anomalies, and structural changes following surgical procedures may also contribute to its etiology (7). The condition is often asymptomatic, and even advanced stenosis may remain clinically silent because of well-developed collateral circulation. In symptomatic patients, manifestations of vertebrobasilar insufficiency may include dizziness, syncope, vertigo, ataxia, diplopia, visual disturbance, tinnitus, and, rarely, transient ischemic attack. Upper extremity findings may include arm claudication, exertional fatigue, paresthesia, coldness, and reduced pulse amplitude (4,8). In patients who have undergone coronary artery bypass grafting using the left internal mammary

artery, left subclavian artery stenosis may compromise coronary perfusion by reversing flow away from the coronary circulation. This condition may result in myocardial ischemia and is referred to as coronary-subclavian steal syndrome (9,10). In clinical evaluation, a systolic blood pressure difference between the two arms is an important diagnostic clue. An inter-arm systolic pressure difference of 15–20 mmHg or greater is generally considered suggestive of subclavian artery stenosis, and larger differences are associated with a higher likelihood of symptomatic disease (7,11). Physical examination findings such as supraclavicular bruit, unilateral weakening of the radial pulse, upper extremity coldness, and paresthesia may further support clinical suspicion. Therefore, subclavian steal syndrome should be considered in the differential diagnosis of patients presenting with posterior circulation symptoms, unexplained dizziness or syncope, visual loss, unexplained transient ischemic attacks, exertional upper extremity symptoms, a marked inter-arm blood pressure difference, or asymmetry in upper extremity pulse amplitude. Early recognition is clinically important to guide timely and appropriate revascularization decisions (12).

Case

A 51-year-old woman presented to the emergency department with loss of consciousness lasting approximately 10 minutes, which occurred after complaints of headache, chest tightness, nausea, and left arm pain. She reported recurrent episodes of dizziness and syncope over the previous six years. Her medical history included diabetes mellitus and hypertension. She was receiving oral antidiabetic, antihypertensive, and antiplatelet therapy. She was also an active smoker with an estimated smoking history of 15 pack-years. On initial examination, the patient was conscious, cooperative, and oriented. Her vital signs were as follows: body temperature 36.1 °C, heart rate 100 beats/min, blood pressure 180/90 mmHg, and oxygen saturation 97% on room air. No acute neurological deficit was detected. However, vascular examination revealed a diminished left radial pulse. Repeated blood pressure measurements demonstrated a marked inter-arm difference, with 97/65 mmHg in the left arm and 160/79 mmHg in the right arm. Electrocardiography and echocardiography were within normal limits. Routine laboratory tests revealed no emergency pathology. Cranial computed tomography showed no acute neurological abnormality, diffusion-weighted magnetic resonance imaging demonstrated no diffusion restriction, and cranial CT angiography revealed no large-vessel occlusion. Overall, no acute neurovascular pathology was identified. To exclude aortic dissection in the differential diagnosis, thoracoabdominal CT angiography was performed. Imaging revealed thrombus formation and stenosis involving an approximately 2–3 cm segment at the origin of the left subclavian artery (Figure 1, 2). The patient subsequently underwent stent placement by interventional radiology and was discharged after an uneventful follow-up period with clinical recovery.

Figure 1: The stenotic segment of the left subclavian artery is demonstrated by the arrow on the coronal section.

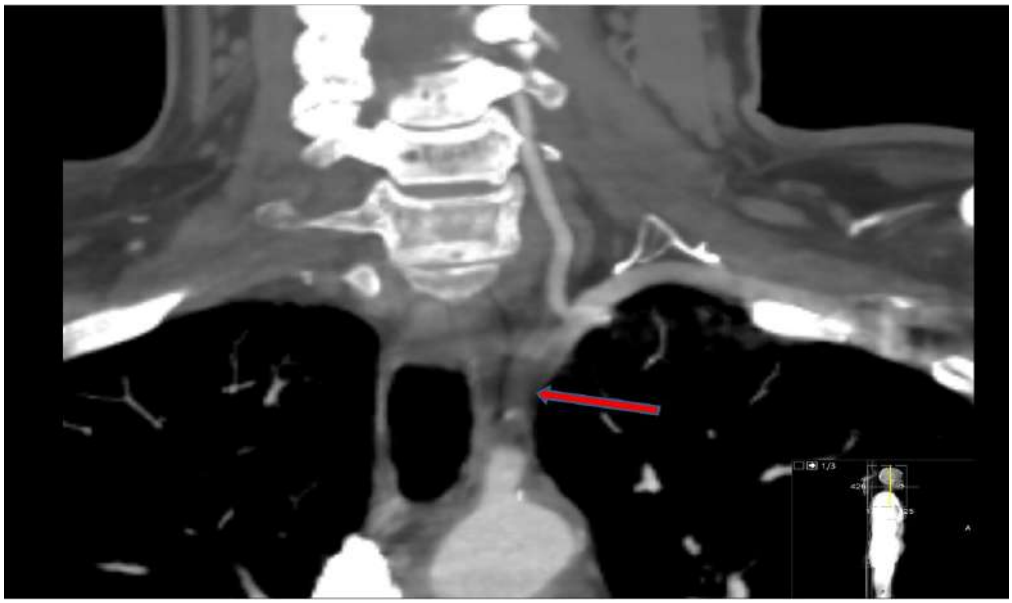
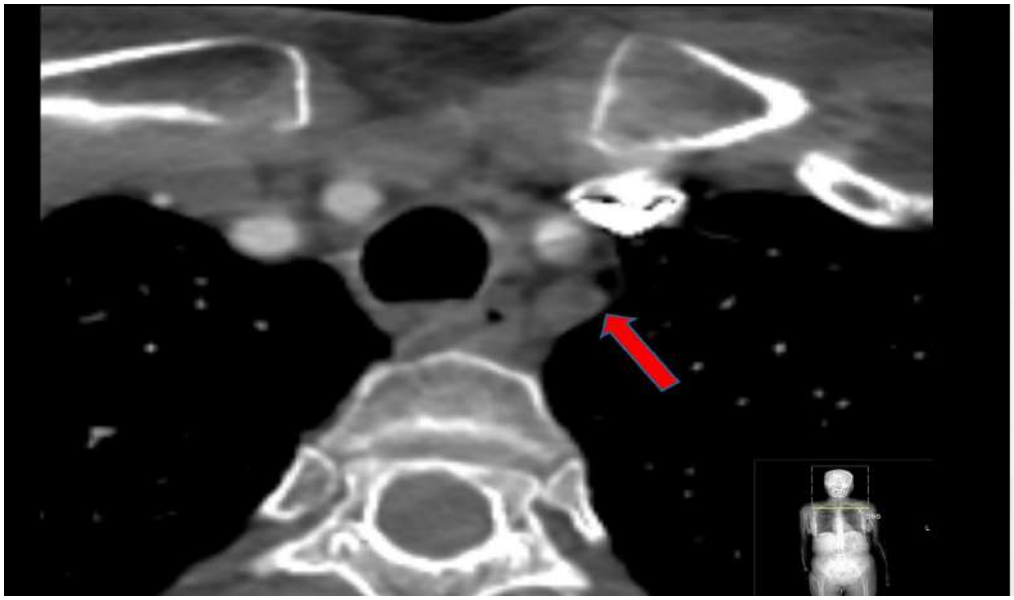


Figure 2: The stenotic segment of the left subclavian artery is demonstrated by the arrow on the axial section.



Discussion

Subclavian steal syndrome is a rare but clinically relevant vascular condition that results from reversal of vertebral artery flow secondary to hemodynamically significant stenosis or occlusion of the proximal subclavian or brachiocephalic artery (12,13). In the present case, the patient had experienced recurrent dizziness and syncopal episodes for six years. Although acute neuroimaging did not demonstrate any ischemic pathology, the chronic and recurrent nature of her symptoms warranted investigation for an underlying hemodynamic disorder. A notable aspect of this case was the nonspecific nature of the patient's initial presentation to the emergency department. Symptoms such as chest tightness, nausea, headache, left arm pain, and syncope commonly raise concern for more frequent and potentially life-threatening conditions, including acute coronary syndrome, pulmonary embolism, aortic dissection, cardiac arrhythmias, hypoglycemia, epileptic seizure, and acute cerebrovascular events. Accordingly, electrocardiography, echocardiography, routine laboratory testing, cranial computed tomography, diffusion-weighted magnetic resonance imaging, and cranial CT angiography were performed, and acute cardiac as well as central neurovascular pathologies were excluded. However, the presence of a marked inter-arm blood pressure difference together with reduced left radial pulse amplitude shifted the diagnostic focus toward aortic and peripheral vascular etiologies. Previous literature emphasizes that bilateral blood pressure measurement and comparison of radial pulses are simple but clinically valuable components of the physical examination in the assessment of subclavian artery stenosis (7,11). In subclavian artery stenosis, an inter-arm systolic blood pressure difference represents an important warning sign. Several studies have suggested that a systolic pressure difference of 15–20 mmHg or greater between the arms may be clinically meaningful for subclavian artery stenosis, and that the likelihood of stenosis and symptomatic disease increases as this difference becomes more pronounced (7,14). In this patient, blood pressure was 160/79 mmHg in the right arm and 97/65 mmHg in the left arm. This finding was critical not only for blood pressure management but also for identifying the affected side and estimating the hemodynamic significance of the lesion. In particular, when antihypertensive therapy is adjusted on the basis of measurements obtained from the stenotic arm, falsely low readings may mislead the clinician. Therefore, in patients with suspected subclavian artery stenosis, blood pressure should be measured in both arms. The detection of thrombus and stenosis involving an approximately 2–3 cm segment at the ostium of the left subclavian artery in our patient provided an anatomical correlate for her clinical presentation. Subclavian steal syndrome is reported more commonly on the left side. This predominance may be related to the anatomical angle at which the left subclavian artery arises from the aortic arch, which may predispose to flow turbulence and atherosclerotic plaque formation (6). In our patient, the presence of diabetes mellitus, hypertension, and active smoking suggested that the stenosis most likely developed on an atherosclerotic background. Current literature indicates that

subclavian artery stenosis should not be interpreted merely as a localized vascular lesion, but rather as a manifestation of systemic atherosclerotic disease (7,14). Therefore, patients diagnosed with this condition should undergo comprehensive cardiovascular risk assessment, including evaluation for coronary artery disease, carotid artery disease, peripheral arterial disease, and cerebrovascular disease. Syncope is not among the most common presenting features of subclavian steal syndrome; however, it may occur when vertebrobasilar perfusion is transiently reduced. Upper extremity exertion, postural changes, fluctuations in systemic blood pressure, or hypotensive episodes related to antihypertensive therapy may further accentuate symptoms (8,13,15). In this case, the approximately 10-minute episode of loss of consciousness, followed by the absence of an acute neurological deficit, required consideration of epileptic seizure, cardiac syncope, and neurovascular hypoperfusion in the differential diagnosis. The lack of an acute ischemic lesion, exclusion of central large-vessel occlusion, and coexistence of a weak left radial pulse with a substantial inter-arm pressure difference supported a vascular and hemodynamic mechanism. Color Doppler ultrasonography is often considered the first-line diagnostic modality. It allows noninvasive evaluation of vertebral artery flow direction, subclavian artery flow velocities, and the hemodynamic impact of the stenosis. Findings such as complete retrograde vertebral flow, partial steal pattern, or systolic notching may provide information regarding the severity of stenosis (5,6). In the present case, thoracoabdominal CT angiography was performed to exclude urgent vascular conditions such as aortic dissection and simultaneously enabled visualization of the ostial lesion in the left subclavian artery. Computed tomography angiography and magnetic resonance angiography provide detailed anatomical information regarding lesion location, length, degree of calcification, and associated aortic arch pathology, and therefore play an important role in treatment planning (12,16). The management in this case, including antiplatelet therapy, avoidance of hypotension, and planning of follow-up for revascularization, is consistent with the literature. In patients with suspected vertebrobasilar hypoperfusion, overly aggressive or misdirected antihypertensive treatment may worsen symptoms. For this reason, blood pressure management should take into account the arm from which the measurement is obtained, the magnitude of the inter-arm pressure difference, and the patient's neurological symptoms. This case is clinically important because it demonstrates that, in patients presenting to the emergency department with unexplained syncope and dizziness, focusing exclusively on cardiac and neurological etiologies may be insufficient. A simple bilateral blood pressure measurement and careful peripheral pulse examination may substantially alter the diagnostic pathway (4).

Conclusion

Subclavian steal syndrome is an uncommon but clinically important cause of recurrent syncope and vertebrobasilar hypoperfusion. In patients with unexplained syncope, normal neuroimaging should not preclude further vascular evaluation when inter-arm blood pressure difference or pulse asymmetry is present. Bilateral blood pressure measurement and careful peripheral pulse examination are simple but critical diagnostic steps that may enable early diagnosis and guide timely revascularization.

Keywords: Inter-arm blood pressure difference, Subclavian steal syndrome, Syncope.

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4117

The Prognostic Value of Biochemical Markers in Predicting Length of Hospital Stay in Electrical Injuries

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ABSTRACT

Objective
This study aimed to evaluate the demographic characteristics and laboratory parameters of adult patients presenting to the emergency department due to electrical shock and to investigate the relationship between these parameters and hospitalization status and length of hospital stay.

Materials and Methods
A total of 25 patients aged 18 years and older who presented to the emergency department with a diagnosis of electrical shock were included in this retrospective study. Patients' demographic characteristics, clinical outcomes, and laboratory data were obtained retrospectively from the hospital information system. Parametric data were expressed as mean ± standard deviation, and non-parametric data as median (IQR). Independent samples t-test and Mann–Whitney U test were used for comparisons between qualitative and continuous variables. Relationships between variables were evaluated using Spearman correlation analysis. Statistical significance was accepted as $p<0.05$.

Results
Sixty percent of the patients were male, and the mean age was 33±10.95 years. Eighteen patients were discharged after outpatient treatment, while 7 patients were hospitalized. Strong and significant positive correlations were found between CK, CK-MB, and AST levels and length of hospital stay (respectively $r=0.728–0.792$; $p<0.001$). In addition, significant positive relationships were found between creatinine and urea levels and length of hospital stay. Strong correlations were also detected among creatinine, CK, CK-MB, and AST. Significant differences were observed between gender and creatinine, hemoglobin, and platelet values.

Conclusion
In patients with electrical shock, especially CK, CK-MB, AST, urea, and creatinine levels show significant associations with length of hospital stay. These biochemical markers may be useful in predicting disease severity and clinical course. Early evaluation in the emergency department may contribute to the identification of high-risk patients.

KEYWORDS
Electrical shock, rhabdomyolysis, creatine kinase, acute kidney injury, length of hospital stay.

Introduction

Electrical shock is an important cause of trauma resulting from exposure to energy ranging from low voltage to high voltage and can affect multiple organ systems[1]. Clinical findings may range across a broad spectrum from mild skin lesions to severe cardiac arrhythmias, muscle necrosis, rhabdomyolysis, and acute kidney injury[2]. This heterogeneous clinical presentation complicates the evaluation and management of patients in the emergency department.

In electrical injuries, the degree of tissue damage is directly related to the intensity and duration of the current exposure and the pathway it follows through the body. Especially damage occurring in muscle and myocardial tissue may lead to increases in biochemical markers such as serum creatine kinase (CK), CK-MB, and aspartate aminotransferase (AST). In addition, kidney injury that may develop secondary to rhabdomyolysis gains clinical importance with elevations in urea and creatinine levels[3].

In patients presenting to the emergency department with electrical shock, early evaluation of laboratory parameters is critically important in determining disease severity, predicting complications, and identifying the need for hospitalization[1]. However, studies clearly demonstrating the relationship between clinical course and laboratory parameters in this patient group are limited.

In this study, it was aimed to evaluate the demographic characteristics and laboratory parameters of adult patients presenting with a diagnosis of electrical shock and to investigate the relationships of these parameters with hospitalization status and length of hospital stay.

Materials and Methods

This study was conducted by retrospectively examining the data of adult patients presenting to the emergency department with a diagnosis of electrical shock. A total of 25 patients who presented within the specified period and whose complete medical records were available were included in the study.

Patients aged 18 years and older diagnosed with electrical shock were included in the study, while patients with incomplete data were excluded. Patients' demographic characteristics (age, gender), clinical outcomes (outpatient treatment or hospitalization status), and laboratory parameters were retrospectively obtained through the hospital information management system.

The evaluated laboratory parameters included parametric data such as hemoglobin, albumin, lymphocyte, platelet, urea, and creatinine, as well as non-parametric data such as C-reactive protein (CRP), neutrophil, amylase, creatine kinase (CK), CK-MB, aspartate aminotransferase (AST), and monocyte. Parametric data were expressed as mean ± standard deviation (SD), while non-parametric data were expressed as median and interquartile range (IQR; 25%–75%).

Statistical analyses were performed using an appropriate software program. Relationships between qualitative variables and parametric data were evaluated using the independent samples t-test. Mann–Whitney U test was used for comparisons between qualitative variables and non-parametric data. Relationships between quantitative variables were examined using Spearman correlation analysis. Statistical significance level was accepted as $p<0.05$.

Statistical Analysis

The distribution of continuous variables was evaluated using visual (histogram and probability plots) and analytical methods. Data conforming to normal distribution were expressed as mean ± standard deviation (SD), while data not conforming to normal distribution were expressed as median and interquartile range (IQR; 25%–75%). Categorical variables were presented as number (n) and percentage (%).

Independent samples t-test was used for comparisons between qualitative variables and parametric data. Mann–Whitney U test was preferred for comparisons between qualitative variables and continuous variables that did not show normal distribution. Spearman correlation analysis was used to evaluate relationships between continuous variables.

Statistical significance level was accepted as $p<0.05$.

RESULTS

In our study, we reviewed the data of 25 adult patients diagnosed with electrical shock. Fifteen (60%) of the patients were male and 10 (40%) were female. All patients were in the adult age group and the mean age was 33 years. Eighteen patients were discharged after outpatient treatment, while 7 patients were hospitalized for treatment.

The mean and SD values of our parametric data were respectively; age:33±10.951, albumin:4.33±0.410, lymphocyte:3.09±0.917, creatinine:0.91±0.286, urea:30.93±9.713, hemoglobin:14.44±2.157, platelet:285.16±74.856 (Table 1).

Table 1. Mean and SD values of our parametric data

Variables	n	Mean	SD
Age (year)	25	33	10.951
Albumin(g/L)		4.33	0.41
Lym (10^3/uL)		3.09	0.917
Creatinine (mg/dL)		0.91	0.286
Urea (mg/dL)		30.93	9.713
Hgb (mmol/L)		14.44	2.157
Plt (10^3uL)		285.16	74.856

The median and IQR values of our non-parametric data were as follows: CRP: 1 (0.15–1.70), neutrophils: 5.52 (4.07–7.87), amylase: 59 (46.5–81), CK: 141 (82.5–220), CK-MB: 14 (9.5–39), AST: 24 (18–49.5), and monocytes: 0.69 (0.58–0.90) (Table 2).

Table 2. Median and IQR values of our non-parametric data.

			IQR	
Variables	n	Median	25%	75%
Crp (g/L)	25	1	0.15	1.7
Neu (10^3/uL)		5.52	4.07	7.87
Amylase (U/L)		59	46.5	81
Ck (U/L)		141	82.5	220
Ck-mb (U/L)		14	9.5	39
Ast (U/L)		24	18	49.5
Mono (10^3/uL)		0.69	0.58	0.9

We compared the relationship between our qualitative variable and parametric variables using the t test. Gender, which was our qualitative variable, showed a statistically significant relationship with creatinine level, and the creatinine level was higher in males ($p=0.045$). When we examined the relationship between gender and platelet count, we found the platelet value to be higher in females, and this relationship was statistically significant ($p=0.008$). When we examined the relationship between gender and hemoglobin, we found the hemoglobin value to be higher in males ($p<0.001$).

We evaluated the relationship between gender, our qualitative variable, and our non-parametric variables using the Mann–Whitney U test. We did not find a statistically significant relationship between gender and our non-parametric variables.

We evaluated the relationships among our quantitative variables using Spearman correlation analysis. We found a moderate positive correlation between length of hospitalization and urea level ($r=0.453$, $p=0.023$). In addition, we detected strong and statistically significant positive correlations between length of hospitalization and creatinine ($r=0.738$, $p<0.001$), CK ($r=0.728$, $p<0.001$), CK-MB ($r=0.792$, $p<0.001$), and AST ($r=0.792$, $p<0.001$) levels. We identified significant positive correlations between urea and creatinine ($r=0.545$, $p=0.005$), CK ($r=0.524$, $p=0.007$), and AST ($r=0.512$, $p=0.009$). We also found significant relationships among muscle and liver enzymes. Creatinine showed significant positive correlations with CK ($r=0.787$, $p<0.001$), CK-MB ($r=0.515$, $p=0.008$), and AST ($r=0.617$, $p=0.001$). Similarly, we found strong positive correlations between CK and CK-MB ($r=0.526$, $p=0.007$) and AST ($r=0.765$, $p<0.001$). We also observed a significant positive correlation between CK MB and AST ($r=0.677$, $p<0.001$). All details are presented in Table 3.

Table 3. Correlation analysis findings

Variable 1	Variable 2	r (Spearman)	p value
Lenght of Stay	Üre	0.453	0.023
Lenght of Stay	Kreatinin	0.738	<0.001
Lenght of Stay	CK	0.728	<0.001
Lenght of Stay	CK MB	0.792	<0.001
Lenght of Stay	AST	0.792	<0.001
Urea	Kreatinin	0.545	0.005
Urea	CK	0.524	0.007
Urea	AST	0.512	0.009
Creatinine	CK	0.787	<0.001
Creatinine	CK MB	0.515	0.008
Creatinine	AST	0.617	0.001
CK	CK MB	0.526	0.007
CK	AST	0.765	<0.001
CK MB	AST	0.677	<0.001

Discussion

Electrical injury is an important cause of trauma resulting from exposure to energy ranging from low voltage to high voltage and can affect multiple organ systems [1]. In this study, demographic characteristics and laboratory parameters of adult patients presenting to the emergency department due to electrical injury were evaluated, and the relationship between these parameters and length of hospital stay was investigated. The findings obtained demonstrated that biochemical markers, particularly those reflecting muscle damage and renal function, were significantly associated with the clinical course.

In our study, the vast majority of patients were male. This may be explained by the fact that electrical injuries are more frequently encountered in the male population actively involved in working life. Similarly, there are studies in the literature reporting a higher incidence of electrical injury cases among males [3].

One of the most important components of tissue damage developing after electrical injury is muscle destruction. The resulting rhabdomyolysis manifests itself with increased creatine kinase (CK) and CK MB levels [4]. In our study, strong and significant positive correlations were detected between CK, CK MB, and AST levels and length of hospital stay. This finding indicates that as the degree of muscle and cellular damage increases, the clinical course becomes more severe and the duration of hospitalization is prolonged. In particular,

concurrent increases in markers such as CK and AST may be considered an important indicator of widespread tissue damage.

The positive correlation detected between urea and creatinine levels, which reflect renal function, and length of hospital stay demonstrates the clinical importance of renal involvement in electrical injuries. Similar studies are available in the literature [5]. Myoglobin release secondary to rhabdomyolysis may lead to acute tubular damage and impaired renal function. Therefore, close monitoring of especially elevated creatinine levels is important for the early detection of complications.

In our study, the presence of significant positive correlations between urea and creatinine, CK, and AST supports the existence of a pathophysiological relationship among muscle damage, liver involvement, and renal function. Similar studies are available in the literature [6]. Likewise, the strong relationships among CK, CK MB, and AST are important findings reflecting the multisystem effects of electrical injury. The combined evaluation of these parameters may contribute to a more accurate prediction of disease severity.

The absence of a significant relationship between non-parametric variables and gender suggests that these parameters may reflect the severity of electrical injury independently of gender. This finding supports that these biomarkers may serve as more objective indicators in clinical use.

In conclusion, the significant relationship between CK, CK MB, AST, urea, and creatinine levels and length of hospital stay in patients with electrical injury suggests that these parameters may be useful in predicting disease severity. Early evaluation of these biochemical markers in the emergency department may provide clinical benefit in identifying high-risk patients and establishing an appropriate treatment plan. Future studies with larger sample sizes and prospective designs are needed to support these findings.

Limitations

This study has several important limitations. First, due to the retrospective design of the study, the data were limited to patient records, and there is a possibility of incomplete or inaccurate records. This may affect the accuracy and interpretation of the findings obtained.

Second, the study was conducted at a single center and the number of patients was relatively low (n=25), which limits the generalizability of the results. Data obtained from larger sample-sized and multicenter studies would contribute to supporting the findings more strongly.

Clinically important variables such as the severity of electrical injury, voltage level of exposure, the pathway of the current through the body, and duration of exposure could not be evaluated in the study. The lack of these factors limited a more detailed analysis of the relationship between laboratory parameters and clinical course.

In addition, long-term clinical outcomes and complications of the patients (such as late renal injury or cardiac involvement) could not be evaluated. Therefore, the findings obtained reflect only short-term outcomes limited to length of hospital stay.

Finally, the biochemical parameters evaluated in the study were based on single-time measurements, which did not allow monitoring of dynamic changes and treatment response. Future prospective studies including serial measurements will more clearly reveal the clinical utility of these parameters.

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Association of the BUN/Creatinine Ratio with 30-Day Mortality and Its Diagnostic Performance in Non-Traumatic Subarachnoid Hemorrhage

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Abstract

Objective:

Subarachnoid hemorrhage (SAH) is a severe neurological emergency with high morbidity and mortality, where early risk stratification is essential. Easily accessible biochemical markers are increasingly used alongside clinical and radiological scores to predict prognosis. This study aimed to assess the association between the blood urea nitrogen (BUN)/creatinine ratio

and 30-day mortality, and to evaluate its predictive performance in patients with non-traumatic SAH presenting to the emergency department.

Methods:

This retrospective observational study included patients with imaging-confirmed non-traumatic SAH who presented to the emergency department over the past two years. Demographic and laboratory data were obtained from the hospital information system. Evaluated parameters included WBC, hemoglobin, platelet count, BUN, creatinine, glucose, albumin, and sodium. The BUN/creatinine ratio was calculated, and serum osmolarity was derived using a standard formula. Patients were grouped according to 30-day mortality. Group comparisons were performed using the Mann-Whitney U test due to non-normal distribution. The predictive performance of the BUN/creatinine ratio was evaluated by ROC analysis, and the optimal cut-off value was determined using the Youden index.

Results:

A total of 40 patients were included, comprising 24 (60%) males and 16 (40%) females. The 30-day mortality rate was 12.5%. The BUN/creatinine ratio was significantly associated with mortality ($p=0.043$), with higher values in non-survivors. No significant associations were observed between mortality and other laboratory parameters, including WBC, Hb, PLT, BUN, creatinine, glucose, albumin, sodium, and serum osmolarity (all $p>0.05$). ROC analysis showed that the BUN/creatinine ratio had significant discriminative ability for predicting mortality (AUC=0.783, 95% CI: 0.649-0.917, $p=0.043$). The optimal cut-off value was 53.1, with a sensitivity of 60% and specificity of 80%.

Conclusion:

The BUN/creatinine ratio was significantly associated with 30-day mortality in non-traumatic SAH and showed acceptable discriminative performance. As a rapid, inexpensive, and readily available parameter in the emergency department, it may be useful for early risk stratification and clinical decision-making. Larger, multicenter prospective studies are needed to confirm these findings.

Keywords:
Subarachnoid hemorrhage, BUN/creatinine ratio, mortality, prognosis, biomarker, emergency department, ROC analysis

Introduction

Subarachnoid hemorrhage (SAH), although less common among all stroke subtypes, is a serious neurological emergency associated with high mortality and morbidity. The vast majority of SAH cases develop secondary to rupture of an intracranial aneurysm, and a significant proportion of patients may die within the first hours or days. Among survivors, severe neurological sequelae are frequently observed. Therefore, the identification of clinical and laboratory markers that can predict prognosis in the early period is of great importance for patient management and the planning of treatment strategies [1,2].

Various clinical and radiological scoring systems, such as the Hunt–Hess score, the World Federation of Neurological Surgeons (WFNS) score, and the Fisher grading scale, are used to predict mortality and clinical outcomes after SAH. However, since these scores are largely based on clinical assessment, interest in biochemical markers that can be obtained rapidly and objectively in the emergency department has been increasing [3,4].

Blood urea nitrogen (BUN) and creatinine levels are fundamental indicators of renal function; however, the ratio of these two parameters, the BUN/creatinine ratio, may provide additional information regarding hemodynamic status, intravascular volume status, and catabolic stress. Recent studies have demonstrated that the BUN/creatinine ratio may be associated with mortality in patients with cardiovascular diseases, heart failure, gastrointestinal bleeding, and those admitted to intensive care units [5,6]. Therefore, as an easily accessible and cost-effective biochemical marker, it has been gaining increasing attention in clinical practice.

The identification of biochemical markers that can predict early prognosis in patients with subarachnoid hemorrhage may facilitate clinical decision-making. In this study, we aimed to evaluate the association between the BUN/creatinine ratio and 30-day mortality in patients presenting to the emergency department with non-traumatic subarachnoid hemorrhage, and to investigate its diagnostic performance in predicting mortality.

Materials and Methods

This study is a retrospective observational study conducted to evaluate the clinical and laboratory data of patients presenting to our emergency department with non-traumatic subarachnoid hemorrhage (SAH). Patients who presented to our emergency department within the last two years and whose diagnosis of subarachnoid hemorrhage was confirmed by a radiologist using computed tomography (CT) or magnetic resonance imaging (MRI) were included in the study.

The inclusion criteria were defined as being 18 years of age or older, receiving a diagnosis of non-traumatic subarachnoid hemorrhage in the emergency department, and having complete laboratory parameters available in the hospital information system at the time of admission.

The exclusion criteria were defined as subarachnoid hemorrhages secondary to trauma, patients younger than 18 years of age, patients with incomplete laboratory data, and cases for whom mortality data could not be obtained from hospital records. A total of 78 patients were initially evaluated. Of these, 40 patients who met the inclusion and exclusion criteria were ultimately included in the study.

The demographic characteristics of the patients (age, sex) and laboratory parameters obtained at admission were retrospectively reviewed from the hospital information management system. The laboratory parameters analyzed included white blood cell count (WBC), hemoglobin (Hb), platelet count (PLT), blood urea nitrogen (BUN), creatinine, glucose, albumin, and sodium levels. In addition, the BUN/creatinine ratio was calculated from these parameters. Serum osmolarity of the patients was calculated using the following formula:

$$\text{Serum osmolarity} = 2 \times Na + (\text{Glucose} / 18) + (\text{BUN} / 2.8)$$

The 30-day mortality status of the patients was assessed using hospital records, and the patients were divided into two groups as those who developed mortality and those who did not.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics 27.0 (IBM Corp., Armonk, NY, USA). The distribution of variables was assessed using the Shapiro–Wilk test. Variables with normal distribution were expressed as mean ± standard deviation, whereas non-normally distributed variables were expressed as median and interquartile range (IQR). Comparisons between patients with and without mortality were performed using the Mann-Whitney U test. The diagnostic performance of the BUN/creatinine ratio in predicting mortality was evaluated using Receiver Operating Characteristic (ROC) curve analysis, and the optimal cut-off value was determined using the Youden index. A p-value of <0.05 was considered statistically significant.

Results

We evaluated patients with non-traumatic subarachnoid hemorrhage (SAH) who presented to our emergency department over the last two years. A total of 40 patients were included in the study. Of these, 24 (60%) were male and 16 (40%) were female. When we examined the 30-day mortality of the patients, we found that mortality occurred in 5 patients in total.

To assess the distribution of laboratory parameters, we performed a normality analysis. The results showed that hemoglobin (Hb, g/dL), platelet count (PLT, $\times 10^3/\mu L$), and white blood cell count (WBC, $\times 10^3/\mu L$) were normally distributed ($p>0.05$). In contrast, blood urea nitrogen (BUN, mg/dL), creatinine (Cr, mg/dL), glucose (mg/dL), albumin (g/dL), sodium (Na^+, mmol/L), the BUN/creatinine ratio, and calculated serum osmolarity (mOsm/kg) were not normally distributed ($p<0.05$).

Variables with normal distribution were expressed as mean ± standard deviation, whereas non-normally distributed variables were expressed as median and interquartile range (IQR). Descriptive statistics of the laboratory parameters are presented in Table 1.

Table 1. Descriptive statistics and distribution characteristics of laboratory parameters

Variables	n	p value	SD	mean	median	IQR 25%	IQR 75%
WBC (×10³/µL)		0,012			11,2	8,2	14,9
Hb (g/dL)		0,134	±0,2	13,3			
PLT (×10³/µL)		0,993	±14,8	248,6			
BUN (mg/dL)		<0,001			42,1	30,3	50,3
Creatinine (mg/dL)		<0,001			0,8	0,7	1
Glucose (mg/dL)	40	0,001			159	133	199
Albumin (g/dL)		<0,001			4	3,6	4,1
Na^+ (mmol/L)		<0,001			139	136	141
BUN/Creatinine ratio		0,001			44,7	35,8	53,9
Serum Osmolarity (mOsm/kg)		0,001			300	294	307

To evaluate the association between laboratory parameters and mortality, we used the Mann–Whitney U test. The analysis revealed a statistically significant association between the BUN/creatinine ratio and 30-day mortality (p=0.043). We observed that the BUN/creatinine ratio was higher in patients who developed mortality.

In contrast, no statistically significant association was found between mortality and WBC (p=0.854), Hb (p=0.336), PLT (p=0.244), BUN (p=0.261), creatinine (p=0.919), glucose (p=0.297), albumin (p=0.332), sodium (p=0.485), serum osmolarity (p=0.115), and the OSMALB index (p=0.198).

To assess the performance of the BUN/creatinine ratio in predicting mortality, we performed Receiver Operating Characteristic (ROC) curve analysis. The results demonstrated that the BUN/creatinine ratio had a statistically significant discriminative ability for predicting mortality (AUC = 0.783, 95% CI: 0.649–0.917, p = 0.043).

According to the Youden index, the optimal cut-off value was calculated as 53.1. At this threshold, the sensitivity was 60% and the specificity was 80%. The ROC curve is presented in Figure 1.

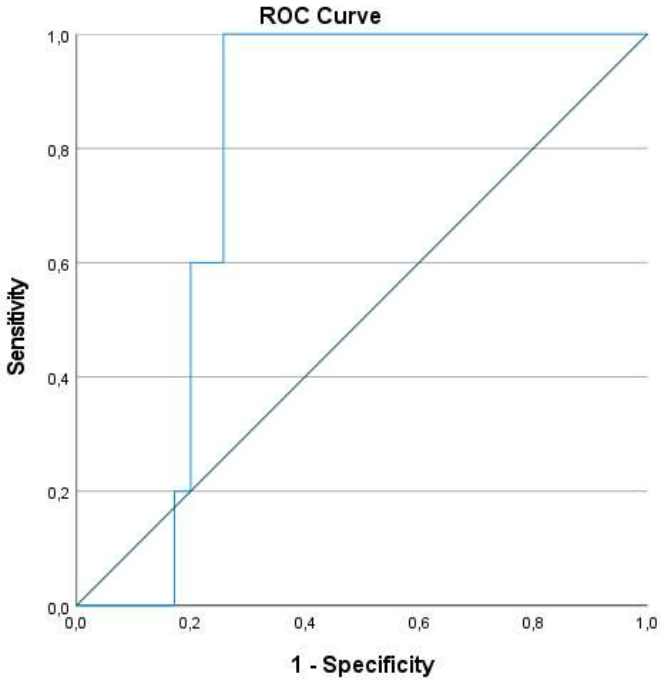

Figure 1. Receiver operating characteristic (ROC) curve of the BUN/creatinine ratio for mortality

Discussion

In our study evaluating patients with non-traumatic subarachnoid hemorrhage (SAH), we found that 60% of the cases were male and the 30-day mortality rate was 12.5%. Although subarachnoid hemorrhage accounts for approximately 5-10% of all strokes, it is an important neurological emergency associated with high mortality and morbidity [1]. In the literature, aneurysmal SAH has generally been reported to be more common in females, and many series have shown a female-to-male ratio ranging from approximately 1.2-1.6:1 [2,3]. In contrast, the predominance of male patients in our series may be related to the limited sample size, the characteristics of the patient population presenting to our center, and the demographic features of the study group.

In our study, when we evaluated the relationship between laboratory parameters and mortality, we found that the BUN/creatinine ratio was significantly associated with 30-day mortality. The BUN/creatinine ratio was higher in patients who developed mortality. In contrast, we did not find a statistically significant association between mortality and WBC, Hb, PLT, BUN, creatinine, glucose, albumin, sodium, serum osmolarity, or the OSMALB index. Additionally, ROC analysis demonstrated that the BUN/creatinine ratio had a statistically significant and acceptable discriminative performance in predicting mortality (AUC=0.783).

Beyond reflecting renal function, the BUN/creatinine ratio is a biochemical indicator that can provide information about hemodynamic status, intravascular volume status, and catabolic stress. Recent studies have shown that BUN or the BUN/creatinine ratio may be associated with mortality in critically ill patients and those with cardiovascular diseases [5-7]. In particular, elevated BUN levels have been reported to be associated with poor prognosis in intensive care and emergency patient populations [6,8,9].

In our study, the finding of a higher BUN/creatinine ratio in patients who developed mortality may be related to hypovolemia, dehydration, catabolic stress response, and alterations in renal perfusion that can occur after subarachnoid hemorrhage. This finding suggests that hemodynamic status and intravascular volume balance may play an important role in the clinical course of patients with SAH. It is well known that intravascular volume depletion and dehydration may adversely affect cerebral perfusion in acute neurological conditions and contribute to secondary brain injury. Therefore, maintaining adequate intravascular volume and preventing dehydration may be clinically important in the management of patients with SAH.

The fact that the BUN/creatinine ratio is a laboratory parameter that can be easily and rapidly obtained in the emergency department suggests that it may serve as a practical biomarker for evaluating hemodynamic status in the early period in patients with SAH. However, due to the limited sample size, this association needs to be confirmed in larger, multicenter studies.

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The Great Mimicker's Silent Strike: Aortic Dissection Masquerading As Acute Ischemic Stroke

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Introduction

Aortic dissection (AD) is defined as a separation of the layers composing the aortic wall. Classically, the dissection plane is described between the intima and the muscular layer; however, other authors describe it as exclusively involving the media or even a separation of the media from the adventitia (1). AD is the most prevalent aortic pathology, with an incidence of 2.6–3.5 cases per 100,000 inhabitants per year. Morbidity and mortality rates have been estimated to be as high as 90% at 3 months without proper and timely management, with an increase rate of 1% per initial hour if untreated within the first 24 hours (2). The typical clinical presentation is described as intense thoracic pain in the anterior chest or interscapular dorsal region, with migratory and tearing characteristics often associated with dysautonomic phenomena such as diaphoresis. Despite these typical symptoms, it has been reported that up to 38% of cases go unnoticed in an initial evaluation, either due to additional symptomatology or the absence of classic symptoms, and in 28% of cases the diagnosis is made post-mortem, highlighting the severity and importance of timely diagnosis (3, 4).

Given the nature of the affected structure and the wide range of organs that can be affected, signs and symptoms of AD can be quite diverse. It is estimated that up to 30% present with associated neurological manifestations, the most frequent being syncope and symptoms related to ischemic phenomena (4). Aortic dissection is often referred to as the 'great mimicker,' as its symptoms frequently mimic those of other common acute conditions, including acute coronary syndrome, pulmonary embolism, and ischemic stroke (5).

Case Presentation

A 62-year-old male patient presented to the emergency department at the 45th minute of symptom onset with acute-onset right-sided weakness and speech impairment. Significantly, the patient did not report any thoracic, back, or abdominal pain. His medical history was significant for a 30 pack-year smoking history and a 10-year history of poorly controlled hypertension. Initial physical examination revealed a blood pressure of 160/90 mmHg and a Glasgow Coma Scale (GCS) score of 14. Other vital signs remained stable.

Neurological assessment demonstrated 3/5 hemiparesis on the right side and aphasia. A notable finding upon examination was a strong pulse in the right radial artery, whereas the left radial pulse was markedly diminished.

In accordance with the acute stroke protocol, an initial non-contrast brain CT was performed, which revealed no evidence of acute hemorrhage or early ischemic changes. However, due to the significant pulse discrepancy identified during the physical examination, further investigations were conducted. CT Angiography of the thorax and abdomen revealed a dissection flap originating from the ascending aorta (Figure 1) and extending into the aortic arch and the left carotid artery (Figure-2), causing significant luminal narrowing in the left carotid artery and minimal pericardial effusion.

Figure 1: The dissection flap in the ascending aorta is indicated by a red arrow

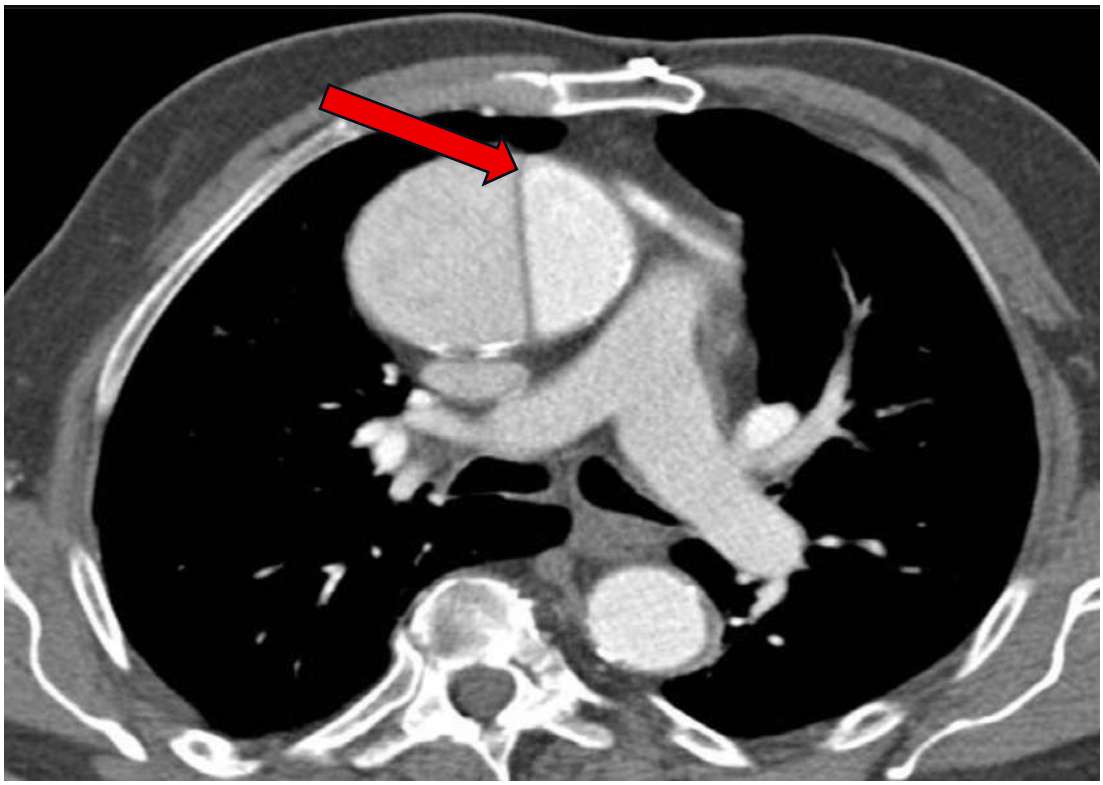
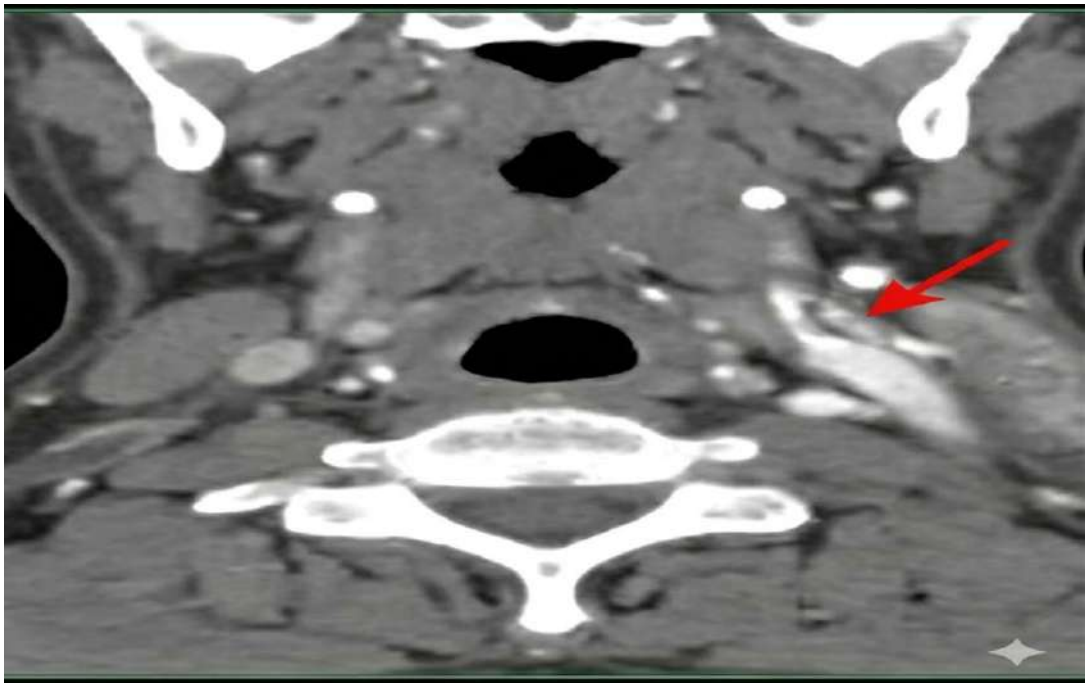


Figure-2: The dissection flap in the left carotid artery is indicated by a red arrow



Simultaneously, Diffusion-Weighted MRI (DWI) showed extensive diffusion restriction in the left hemisphere (frontal operculum, insular cortex, and parietal lobe), consistent with acute ischemic infarction. Following the diagnosis of cerebral hypoperfusion secondary to Stanford Type A aortic dissection (presenting as a stroke mimic), the planned thrombolytic therapy was immediately canceled. IV beta-blocker therapy was initiated, and the patient was urgently transferred to the cardiovascular surgery unit for emergency surgical intervention.

Discussion

AD is a critical medical emergency that demands timely and meticulous evaluation, diagnosis, and treatment to mitigate associated morbidity and mortality. Neurological symptoms associated with AD are often dramatic, potentially overshadowing other clinical features (6).

For both Stanford type A and B dissection, show a notable occurrence of alteration of consciousness, possibly due to ischemic events resulting from compromised cerebral circulation, both anteriorly via the internal carotid artery and posteriorly through the vertebral arteries. These ischemic events may be a consequence of either malperfusion syndrome or thrombotic events derived from the underlying pathology, commonly involving aortic arch or ascending aorta. In cases of alteration of consciousness or transient global amnesia, patients may be unable to report chest pain, thereby complicating the diagnostic process (2, 6, 7). Furthermore, 'painless' aortic dissection—as observed in our case—presents an even greater diagnostic challenge. While the absence of pain is sometimes attributed to a patient's inability to communicate due to neurological deficits, it is well-documented that a significant portion of AD patients truly do not experience the classic tearing sensation. This lack of hallmark

symptoms often leads clinicians toward a primary diagnosis of isolated ischemic stroke, potentially resulting in the administration of contraindicated thrombolytic therapies.

In the context of Stanford type A dissections, a distinctive pattern of symptomatology emerges. Motor syndrome takes precedence, followed by altered consciousness and subsequently, language disturbances. This pattern reflects a predominant impairment of anterior circulation, as critical neural areas such as Broca's area and the primary motor cortex rely on the middle cerebral artery, a terminal branch of the internal carotid artery. These results prompt consideration of arterial dissection as a potential etiology for a cerebrovascular event (2).

Conclusion

AD is a complex medical emergency which requires accurate and prompt medical intervention to avoid catastrophic outcomes. In the context of the emergency department, understanding the neurological manifestations in both Stanford type A and B dissections emerges as a pivotal cornerstone for conducting precise assessments and efficient patient management. Given the wide spectrum of manifestations, we emphasize the importance of suspecting AD when neurological disorders are associated with the classic symptoms. In these cases, we encourage a complete neurological examination to be performed and a multidisciplinary group assembled to tackle this entity.

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Diagnosis and treatment of nerve injuries.

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Introduction

Peripheral and facial nerve injuries are frequently encountered in traumas caused by sharp objects. Primary and early primary repair is crucial in nerve injuries. Therefore, diagnosis at the initial presentation at the emergency room is important. Nerve injuries are one of the clinical conditions that can sometimes be overlooked in patients, leading to significant functional loss and morbidity. Furthermore, delays in diagnosis can lead to legal problems. Early diagnosis and appropriate treatment approaches are critical for functional recovery. Knowledge of the most frequently affected anatomical regions and current treatment methods will contribute to improved clinical outcomes. In this study, we retrospectively evaluated the diagnostic methods, most frequently affected anatomical regions, treatment approaches, and outcomes of facial and peripheral nerve injuries seen in our emergency department.

Materials and Methods:
This retrospective study included a total of 101 patients treated for facial and peripheral nerve injury at a single center. Amputations and brachial plexus injuries were excluded from the study. Patients were evaluated in terms of age, gender, injury localization, affected nerves, and outcomes.

Results:

The mean age of the patients was 41, with 84 male (83%) and 17 female (17%). The majority of injuries (96%) were located in the upper extremities. The most frequently injured nerves were the digital nerves at the finger level (40%), followed by median (19%) and ulnar nerve injuries (19%). Facial nerve injuries were less common (4%). Injuries were mostly due to penetrating trauma and lacerations (98%). Primary repair was the most common treatment %95), while nerve grafting and other surgical methods were preferred in selected cases. All patients underwent a physical therapy and rehabilitation program targeting the injured organs for the necessary duration. Rehabilitation programs included patient education, edema and pain control, range of motion maintenance and gentle stretching exercises, muscle strength maintenance and enhancement exercises, and sensory re-education. Hot/cold applications, electrical stimulation, bandaging, and orthotics were applied for these purposes. Treatment duration varied depending on the needs.

Discussion

Peripheral nerve injuries are relatively common following extremity trauma and lead to significant functional and sensory deficits, impairing daily activities and reducing quality of life. In the upper extremity, the median, ulnar, and radial nerves are associated with distinct clinical presentations. Median nerve injury typically causes numbness and weakness in the thumb, index, and middle fingers; ulnar nerve injury affects the ring and little fingers; and radial nerve injury results in impaired wrist extension and drop.

Early diagnosis remains challenging, as these injuries may be overlooked in emergency settings, particularly when masked by associated trauma. Early specialist consultation improves diagnostic accuracy, and injuries more severe than neurapraxia should be identified promptly to optimize management.

Treatment depends on injury type and severity, classified as open or closed. Open injuries require surgical exploration to assess nerve continuity. In sharp transections, primary repair within 72 hours is recommended. In cases without clear transection, serial clinical, electrodiagnostic, and imaging evaluations are necessary to monitor recovery. Electrodiagnostic studies are essential, especially in closed injuries, while MRI is increasingly valuable for assessment.

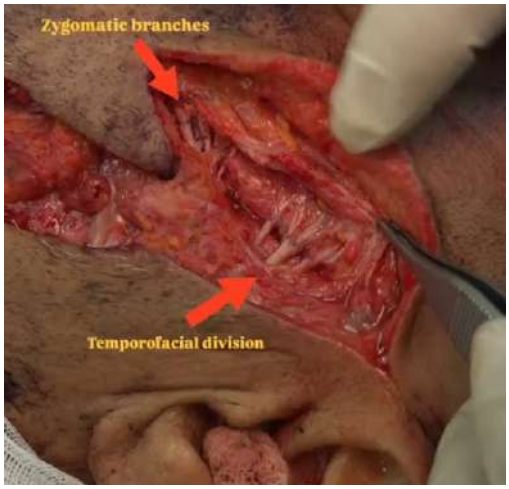
According to the Seddon classification, nerve injuries are categorized as neurapraxia, axonotmesis, and neurotmesis, the latter representing complete disruption requiring surgical repair. The Sunderland classification further defines injury severity from Grade I to V, with Grade VI describing mixed injuries.

Optimal management requires a multidisciplinary approach, including rehabilitation to maintain joint mobility, prevent contractures, and restore function. Prognosis depends on multiple factors such as injury severity, fibrosis, patient age, and repair quality. Axonal regeneration occurs at approximately 1 mm/day and can be monitored with the Tinel sign. In complete transections, meaningful recovery requires surgical repair.

These findings highlight the need to increase awareness and improve early recognition and referral of nerve injuries, emphasizing the importance of continuous medical education for clinicians.

Conclusion:

Early diagnosis and appropriate treatment approach in nerve injuries are critically important for functional recovery. The high frequency of upper extremity and digital nerve injuries in trauma patients necessitates increasing clinical awareness regarding these areas. Morbidity can be minimized with timely, correct, and atraumatic surgical techniques and follow-up physical therapy rehabilitation.



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Validation of an Artificial Intelligence-Based Model (APPENDIXPERT) for the Detection of Acute Appendicitis on Abdominal CT in the Emergency Department

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Introduction

Acute abdomen encompasses a spectrum of clinical conditions requiring prompt diagnosis in the emergency department (ED), as delays may result in significant morbidity and mortality. Acute appendicitis remains one of the most frequent surgical emergencies worldwide and may lead to severe complications such as perforation, peritonitis, abscess formation, and death (1).

Although ultrasonography is widely utilized as an initial imaging modality in the diagnosis of acute appendicitis, its diagnostic performance is limited by operator dependency and technical constraints, particularly in emergency settings (2). In contrast, computed tomography (CT) offers superior diagnostic accuracy but contributes to increased radiology

workload. Concurrently, ED overcrowding and rising imaging demands exacerbate physician workload and may increase the risk of delayed or missed diagnoses. In recent years, artificial intelligence (AI)-based models have emerged as promising tools to enhance diagnostic accuracy and improve workflow efficiency (3-6).

The aim of this study is to evaluate the diagnostic performance of the APPENDIXPERT artificial intelligence model for detecting acute appendicitis on contrast-enhanced and non-contrast abdominal CT scans in adult patients presenting to the emergency department with non-traumatic abdominal pain.

Materials and Methods

This retrospective methodological study included adult patients presenting with non-traumatic abdominal pain to the ED of Dokuz Eylül University Hospital between July 1, 2025, and October 30, 2025, who underwent both contrast-enhanced and non-contrast abdominal CT imaging.

Inclusion Criteria

- Age ≥18 years
- Presentation with non-traumatic abdominal pain
- Undergoing abdominal CT imaging

Exclusion Criteria

- Traumatic abdominal pain
- Presence of additional chief complaints
- Missing or incomplete data
- Inability to determine clinical outcomes

The APPENDIXPERT system (Version: BETA; HEALYSENSE Inc., İzmir, Turkey, 2025) is a deep learning-based algorithm designed to analyze abdominal CT images and detect regions suspicious for acute appendicitis. The system highlights areas of interest using heat maps and generates diagnostic reports with corresponding confidence scores. The model was trained on data from 395 patients comprising 34449 CT slices and validated using an independent dataset of 8612 slices. The gold standard for diagnosing acute appendicitis is pathological diagnosis of surgical specimens. Exclusion of acute appendicitis is performed by a specialist radiologist blinded to AI results.

Sample size calculation (G*Power/OpenEpi) indicated that a minimum of 254 cases was required (power: 80%, confidence level: 95%).

Results

A total of 1392 abdominal CT scans were analyzed, including 991 contrast-enhanced and 401 non-contrast studies. Acute appendicitis was radiologically and pathologically confirmed in 130 cases (119 contrast-enhanced and 11 non-contrast). The APPENDIXPERT model correctly identified 111 of these cases (102 contrast-enhanced and 9 non-contrast), yielding an overall sensitivity of 85.4% (Table-1).

Among 897 cases classified by the model as “no appendicitis” (572 contrast-enhanced and 325 non-contrast), 878 were correctly identified (555 contrast-enhanced and 323 non-contrast), corresponding to a specificity of 97.9% (Table-1).

Table-1. Diagnostic performance of the APPENDIXPERT model.

			Radiology & Pathology	**Radiology**	
			Yes (n)	No (n)	
Non-contrast CT	**Appendixpert**	Yes (n)	9	67	**11.8%**
		No (n)	2	323	**%99.4%**
			81.8%	**82.8%**	
Contrast-enhanced CT	**Appendixpert**	Yes (n)	102	317	**24.3%**
		No (n)	17	555	**97.0%**
			85.7%	**63.6%**	
Total	**Appendixpert**	Yes (n)	111	384	**22.4%**
		No (n)	19	878	**97.9%**
			85.4%	**69.6%**	

Discussion

In this study, we evaluated the diagnostic performance of the APPENDIXPERT artificial intelligence model for detecting acute appendicitis on abdominal CT scans in the emergency department setting. The model demonstrated a high sensitivity (85.4%) and excellent specificity (97.9%), suggesting that AI-assisted interpretation may provide reliable support in clinical decision-making.

The sensitivity observed in our study is comparable to previously reported AI-based appendicitis detection models, which typically range between 80% and 95%. Notably, the high specificity achieved by APPENDIXPERT indicates a strong ability to correctly exclude appendicitis, which is particularly valuable in reducing unnecessary surgical consultations and imaging overuse. In the context of emergency department overcrowding and increasing radiology workload, such performance characteristics are highly clinically relevant (7,8).

The model showed consistent performance across both contrast-enhanced and non-contrast CT scans, although slightly higher detection rates were observed in contrast-enhanced studies. This finding aligns with the known superiority of contrast-enhanced CT in visualizing appendiceal inflammation. However, the ability of the AI model to maintain acceptable performance in non-contrast imaging is of particular importance, as it may support diagnostic workflows in patients where contrast administration is contraindicated (7).

From a workflow perspective, AI-based tools such as APPENDIXPERT have the potential to function as a decision-support system rather than a replacement for radiologists. By highlighting suspicious regions via heat maps and providing confidence scores, the model may facilitate faster interpretation, reduce cognitive load, and minimize the risk of missed diagnoses, especially in high-volume emergency settings (9,10).

Future studies should focus on multicenter validation, real-time prospective implementation, and integration of AI systems into clinical workflows to assess their impact on patient outcomes, diagnostic turnaround times, and healthcare resource utilization.

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4244

A Rare Condition in the Etiology of Persistent Low Back Pain: Spinal Epidural Abscess (SEA)

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Although spinal epidural abscess (SEA) is a rare condition, it may result in severe neurological sequelae and mortality. Its annual incidence ranges between 0.2 and 2.8 per 10,000 hospital admissions. In recent years, the incidence has increased due to advancements in diagnostic modalities, a rise in invasive procedures, and the more frequent occurrence of predisposing risk factors.

The classical triad for SEA diagnosis—fever, low back pain, and neurological deficit—is reported to be present in only 10–15% of cases.

In this report, we present a patient who was admitted with persistent low back pain resistant to analgesic therapy and was subsequently diagnosed with spinal epidural abscess through advanced imaging. The aim is to emphasize the importance of early diagnosis, prompt surgical decompression, and appropriate antibiotic therapy.

Case Presentation

A 39-year-old female patient presented with severe low back pain and difficulty in movement persisting for one week. Her medical history was notable only for hypothyroidism. She denied any history of intravenous drug use, trauma, spinal surgery, or immunosuppressive conditions. It was learned that she had received intramuscular analgesic treatment at another center; however, her pain had not improved.

On admission, the patient was in good general condition. Her body temperature was 36.5°C, and her vital signs, as well as physical and neurological examinations, were within normal limits. Imaging revealed a fluid collection in the epidural space extending from L3 to S1 levels, causing compression of the thecal sac. Laboratory findings were as follows: WBC: 9,800/mm³, CRP: 208 mg/L, and ESR: 100 mm/hour.

Following contrast-enhanced lumbar magnetic resonance imaging (MRI), the patient was diagnosed with SEA and evaluated by the Neurosurgery and Infectious Diseases departments. She subsequently underwent surgery, including multilevel laminectomy and abscess drainage. Microbiological culture revealed growth of *Staphylococcus aureus*. After six weeks of intravenous antibiotic therapy in the postoperative period, the patient was discharged in good health.

Discussion

SEA is a clinical condition that may lead to neurological deficits and mortality and is often associated with diagnostic delays due to its initially nonspecific presentation. Pain that progresses despite analgesic treatment, elevated inflammatory markers, the presence of risk factors, or a recent history of invasive procedures should prompt consideration of SEA.

Studies have reported that elevated CRP levels are present in nearly all cases, while elevated ESR is observed in the majority. In the present case, despite a normal leukocyte count, both CRP and ESR levels were elevated.

Although diabetes mellitus, immunosuppression, intravenous drug use, spinal surgery, and epidural interventions are well-known risk factors, the literature also suggests that intramuscular injections and acupuncture may contribute to the development of SEA.

Contrast-enhanced MRI is the gold standard diagnostic modality due to its high sensitivity and specificity. *Staphylococcus aureus* is reported to be responsible for approximately half of all cases. The identification of *S. aureus* in this case is consistent with the literature.

Conclusion

Rare but serious conditions such as SEA should not be overlooked in patients presenting with persistent low back pain. The absence of fever, a normal leukocyte count, or the lack of clear risk factors does not exclude an infectious etiology.

Figure 1. preop lumbar epidural abscess

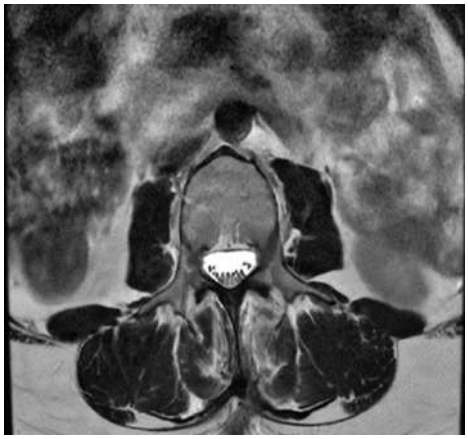

Figure 2.postop lumbar epidural abscess

4245

From Bedside Echo to Successful Resuscitation: Intra-CPR Thrombolysis in Massive Pulmonary Embolism

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INTRODUCTION

Acute pulmonary embolism (PE) is a life-threatening emergency caused by obstruction of the pulmonary arteries and their branches by a thrombus originating from the venous circulation. It is often difficult to diagnose and may present with a highly variable clinical course [1]. Patients may be asymptomatic or may present to the emergency department with dramatic manifestations such as chest pain, syncope, refractory hypotension, cardiogenic shock, or cardiac arrest [2]. Massive pulmonary embolism results in sudden right ventricular pressure overload and dilation, causing a leftward shift of the interventricular septum, which critically impairs left ventricular filling and cardiac output [1]. This vicious cycle rapidly leads to coronary ischemia, shock, and ultimately cardiac arrest. In cases of massive PE complicated by cardiac arrest, conventional cardiopulmonary resuscitation (CPR) alone is often insufficient because it cannot relieve the mechanical obstruction, and mortality exceeds 70% [3]. According to the European Resuscitation Council (ERC) and other current guidelines, systemic fibrinolytic (thrombolytic) therapy is strongly recommended when acute pulmonary embolism is a known or strongly suspected cause of cardiac arrest, in order to rapidly restore pulmonary circulation and achieve hemodynamic stabilization [1]. In this article, we aimed to present the case of a 45-year-old woman who developed cardiac arrest following syncope in the emergency department, was suspected of having massive PE based on bedside echocardiographic findings, received intravenous tissue plasminogen activator (alteplase) during resuscitation, was successfully resuscitated, and was discharged without neurological sequelae.

CASE REPORT

A 45-year-old woman with no known comorbidities was brought to the emergency department yellow zone by the 112 emergency medical services team with complaints of malaise that had begun earlier that morning and a syncopal episode occurring approximately 30 minutes before hospital arrival. It was learned that her consciousness had initially returned during prehospital management but deteriorated again during transport.

Upon arrival at the emergency department, the patient was unconscious and responded inappropriately to painful stimuli. She was rapidly connected to monitoring equipment. Initial evaluation revealed an unmeasurable blood pressure, weak peripheral pulses, and markedly

delayed capillary refill. Finger-stick blood glucose was measured at 145 mg/dL. Electrocardiography (ECG) demonstrated 1-mm ST-segment elevation in the inferior leads (Figure 1), raising suspicion for an inferior myocardial infarction, and the patient was transferred to the resuscitation room. Bedside focused echocardiography performed by our team revealed severe right ventricular dilatation, a markedly akinetic right ventricle, and significant leftward displacement of the interventricular septum. Based on these findings, massive pulmonary embolism was considered the leading diagnosis. Immediate consultations were obtained from the departments of pulmonology and cardiology. The patient underwent elective endotracheal intubation. During cardiology consultation, the ECG and echocardiographic findings were confirmed, and urgent intravenous thrombolytic therapy was planned. While preparations for thrombolytic treatment were ongoing, the patient developed cardiac arrest, and CPR was initiated immediately. Simultaneously with CPR, a total dose of 100 mg alteplase (Actilyse) was administered according to an accelerated thrombolytic protocol; 50 mg was given as an intravenous bolus, followed by infusion of the remaining 50 mg over two hours. Return of spontaneous circulation (ROSC) was achieved five minutes after intravenous alteplase bolus administration. However, during the first 15 minutes of follow-up, the patient experienced three additional episodes of cardiac arrest, requiring a cumulative CPR duration of approximately 10 minutes. After hemodynamic stabilization was achieved, computed tomography pulmonary angiography (CTPA) demonstrated findings consistent with massive pulmonary embolism (Figure 2). The patient was subsequently admitted to a tertiary cardiovascular surgery intensive care unit. Her clinical condition improved rapidly during follow-up, and she was successfully extubated on the second day of hospitalization. After six days of intensive care treatment, she was transferred to the inpatient ward. Following an additional two days of ward observation, she was discharged home in good health, ambulatory, and without any neurological or physical sequelae.

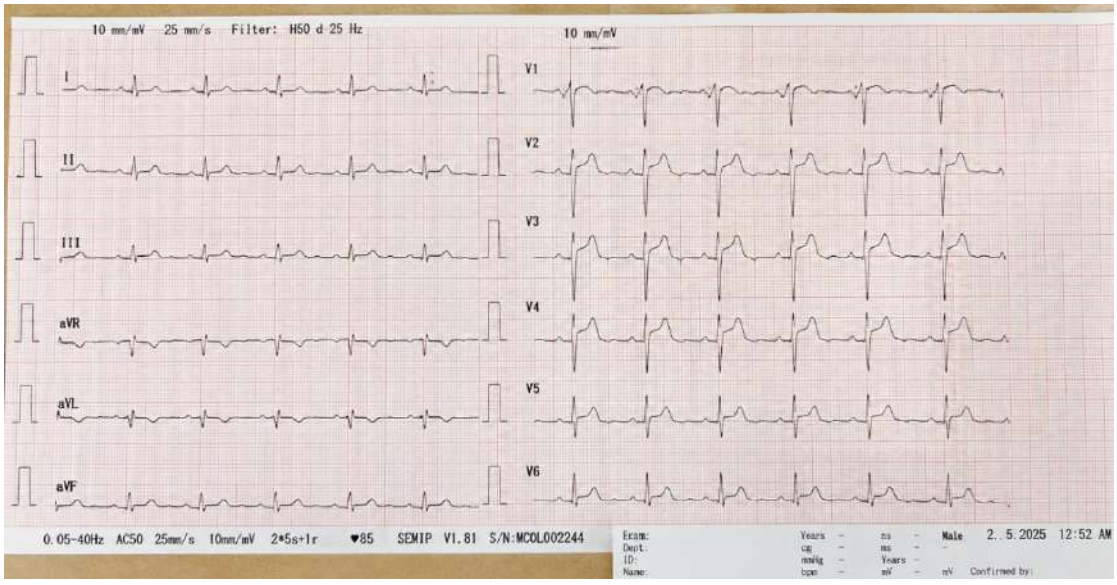

Figure 1 Patient's ECG Findings

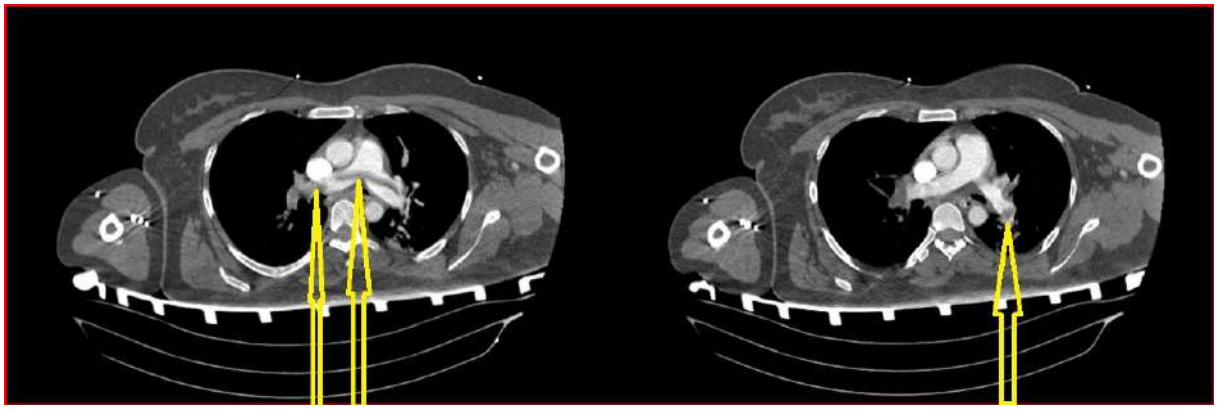

Figure 2: Patient's CT Angiography Images

DISCUSSION

Massive pulmonary embolism is an extremely challenging and often fatal clinical condition when it culminates in cardiac arrest [1,3]. In the literature, Aliyev et al. reported survival in a 70-year-old patient who developed refractory shock and cardiac arrest, following administration of alteplase during prolonged resuscitation lasting 55 minutes [1]. In our case, however, alteplase bolus was administered immediately at the onset of cardiac arrest in a younger patient, and an early response was achieved within 5 minutes. Nevertheless, recurrent episodes of cardiac arrest occurred within the following 15 minutes due to the persistent hemodynamic effects of the thrombus burden, demonstrating that achieving myocardial stability in cases of massive PE may require time. Currently, alternative and successful treatment modalities such as ultrasound-assisted catheter-directed thrombolysis (EKOS) and surgical embolectomy are also used in the management of pulmonary embolism [2,3,4]. However, the EKOS method reported by Açıkgöz et al. is generally preferred in patients who are relatively hemodynamically stable or in whom sufficient time is available for procedural preparation [4]. Furthermore, Uysal et al. emphasized that mortality is very high among patients undergoing surgical pulmonary embolectomy who experience preoperative cardiac arrest [3]. In light of these findings, it is evident that the time required for surgical or catheter-based preparation may be detrimental in emergency patients who suddenly develop cardiac arrest or experience rapidly deteriorating hemodynamics. The most important point is this: in cases of cardiac arrest caused by clinically highly probable or confirmed massive pulmonary embolism, systemic intravenous (IV) thrombolytic therapy (alteplase/Actilyse) is unquestionably indicated and represents the primary life-saving intervention. As clearly stated in the European Resuscitation Council (ERC) guidelines, ongoing CPR in the setting of suspected massive PE is not a contraindication to thrombolytic administration; on the contrary, fibrinolytic therapy provides a meaningful chance of survival [1]. Once fibrinolytic therapy has been administered, CPR should be continued for at least an additional 60–90 minutes to allow sufficient time for clot dissolution and restoration of circulation [1]. In our case, intravenous alteplase bolus therapy initiated immediately at the moment of cardiac arrest rapidly restored perfusion to the obstructed pulmonary vascular bed, making it possible for the patient to survive and recover without any sequelae.

CONCLUSION

Massive pulmonary embolism should always be considered in the differential diagnosis of patients presenting to the emergency department with cardiac arrest or developing cardiac arrest during their emergency department stay. Detection of signs of acute right heart strain on bedside echocardiography greatly facilitates the early diagnosis of this life-threatening condition. In patients with suspected pulmonary embolism who present with shock or develop cardiac arrest, administration of intravenous thrombolytic therapy (alteplase) simultaneously with resuscitation, without waiting for time-consuming interventions such as catheter-based procedures or surgery, is an absolute indication and the most critical intervention for dramatically reducing mortality.

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4246 **Rhabdomyolysis Developing Due to Hypothermia: A Case Report**

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Abstract
Rhabdomyolysis is a clinical condition characterized by the release of intracellular contents into the circulation as a result of damage to skeletal muscle cells. Trauma, drugs, infections, intense exercise, and hypothermia are among the important causes of rhabdomyolysis. Although rhabdomyolysis due to hypothermia is rare, it can lead to serious complications. This article presents the case of a 40-year-old male shepherd who developed rhabdomyolysis after being exposed to a cold environment for a long time.

Keywords: Rhabdomyolysis, hypothermia, myoglobinuria, acute renal injury, environmental exposure

Introduction

Rhabdomyolysis is a clinical syndrome characterized by the breakdown of skeletal muscle cells, resulting in the release of creatine kinase (CK), myoglobin, and other intracellular substances into the systemic circulation (1).

The most common causes of rhabdomyolysis include trauma, prolonged immobilization, drugs and toxins, intense exercise, electrolyte imbalances, infections, and hypothermia (2). Hypothermia can cause rhabdomyolysis, particularly in individuals exposed to low temperatures for extended periods, through decreased muscle perfusion and impaired cellular metabolism (3). This case report presents a case of rhabdomyolysis caused by hypothermia.

Case Presentation

A 40-year-old male shepherd was stranded for approximately 15 hours in a mountainous and cold region in November due to snowfall that began while he was grazing his sheep at -4 °C. After being found, the patient was kept at room temperature. Approximately 10 hours later, he developed increasing muscle and joint pain and was brought to the emergency room by 112 emergency medical services.

The patient's relatives stated that when the patient was found, both his upper and lower extremities were cold, pale, and rigid.

Initial assessment in the emergency department revealed: Glasgow Coma Score: 15, Blood pressure: 100/70 mmHg, Oxygen saturation: 98%, Heart rate: 120/min, ECG: Sinus tachycardia, Body temperature: 36.5 °C. Intravenous hydration and analgesic treatment were initiated.

Whole body examination showed: Skin color was normal, pulses were palpable in all four extremities, range of motion was normal, and there were no signs of trauma or ecchymosis. The patient described severe tenderness on touch and movement in both upper and lower extremities, back, waist, pelvis, and shoulder regions. Direct radiography and Doppler ultrasonography of the extremities revealed no pathology.

The patient reported that their urine had darkened. Blood parameters measured were: AST 443 U/L, ALT 105 U/L, LDH 1019 U/L, CK 25000 U/L, CK-MB 721 U/L, K 3.3 mmol/L, Ca 8.8 mg/Dl, Troponin <0.100, INR 1.54, BUN 28.5 mg/Dl, Creatinine 0.98 mg/Dl. It was learned that liver and kidney function tests and electrolyte values were normal at the patient's visit three weeks prior.

Based on clinical and laboratory findings, the patient was diagnosed with rhabdomyolysis.

The patient was admitted to the intensive care unit to prevent complications that may arise from rhabdomyolysis.

Treatment included intensive intravenous fluid therapy, electrolyte monitoring, renal function monitoring, and pain control. The patient was discharged after 5 days of treatment and observation without developing any complications.

DISCUSSION

Rhabdomyolysis is a syndrome that develops as a result of damage to the muscle cell membrane, causing cellular contents to leak into the circulation (1).

Hypothermic rhabdomyolysis can occur especially in individuals who remain in cold environments for a long time. Cold-induced vasoconstriction and decreased muscle perfusion can lead to muscle cell damage (4).

Clinically, muscle pain, muscle tenderness, weakness, and dark urine may be observed (2). In the diagnosis of rhabdomyolysis, an increase in serum CK levels more than 5 times the normal level is an important criterion (5). In this patient, the CK value was found to be 25000 U/L. The most serious complication of rhabdomyolysis is acute kidney injury, which can develop in approximately 10–40% of cases (3).

Aggressive fluid therapy initiated early in rhabdomyolysis reduces the development of acute kidney injury (6). Therefore, early diagnosis and aggressive fluid therapy are critically important. In this case, thanks to early diagnosis and rapid hydration therapy, no renal complications developed.

In individuals who have been exposed to cold for a long time and experience symptoms such as muscle pain and dark urine, rhabdomyolysis should always be considered. Early diagnosis and appropriate fluid therapy can prevent the development of serious complications.

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Multivessel spontaneous coronary artery dissection in the postpartum period: a case report

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INTRODUCTION

The postpartum period is associated with an increased risk of acute coronary events because of abrupt hemodynamic and hormonal changes in the cardiovascular system. Although pregnancy-associated myocardial infarction is rare, it is linked to substantial maternal morbidity and mortality [1]. In cases of acute myocardial infarction related to pregnancy and the postpartum period, spontaneous coronary artery dissection (SCAD) is considered one of the most important etiologies [2]. SCAD is characterized by separation of the coronary arterial wall and/or formation of an intramural hematoma in the absence of atherosclerosis, trauma, or iatrogenic intervention, and it is an important cause of acute coronary syndrome, particularly in young women without prominent traditional cardiovascular risk factors [3]. Pregnancy-associated SCAD most commonly occurs during the last trimester of pregnancy or in the early postpartum period, especially within the first month after delivery [2]. It is thought that the marked hemodynamic stress and hormonal changes during pregnancy and childbirth lead to structural fragility of the vessel wall [4]. In addition, fibromuscular dysplasia and other extracoronary arteriopathies may coexist in patients with SCAD [5]. While a conservative approach is generally preferred in hemodynamically stable patients, invasive intervention may be required in the presence of left main coronary artery involvement, multivessel disease, ongoing ischemia, or hemodynamic instability [5].

The aim of this case report is to highlight the diagnostic and management challenges posed by multivessel SCAD in the postpartum period in emergency department practice. The present case is notable from an emergency medicine perspective because it describes a rare postpartum patient who was initially hemodynamically stable, had no significant abnormalities on the first ECG, and had a normal initial troponin level, yet progressed within hours to severe coronary dissection.

CASE PRESENTATION

A 30-year-old woman presented to the emergency department at 7:30 with complaints of chest pain that started one hour before her arrival and spread to her left arm. She had no known comorbidities, no regular medication use, and no known allergies. Her history was notable for a spontaneous vaginal live birth 30 days earlier. There was no history of uterotonic or non-steroidal anti-inflammatory drug use. This was her third known pregnancy; she had

two live births and one previous abortion. At presentation to the emergency department, she was conscious, alert, oriented, and cooperative. Her blood pressure was 120/60 mmHg, pulse 90/min and regular, body temperature 36.7°C, oxygen saturation 98% on room air, and blood glucose 97 mg/dL. Electrocardiography (ECG) showed minimal ST-segment depression in leads I and aVL (Figure 1).

On initial examination, her Glasgow Coma Scale score was 15. Pupils were isochoric, and bilateral direct and indirect light reflexes were positive. Neurological examination was normal. Lung and heart sounds were normal. Abdominal examination was unremarkable. There was no guarding or rebound tenderness. Peripheral pulses were palpable.

Bedside echocardiography revealed an ejection fraction of 39% (measured by the Simpson method) and diffuse apical hypokinesia. The right heart chambers appeared normal. No D-shape or McConnell sign was observed. No significant valvular pathology was detected. No findings suggestive of aortic dissection were present.

Laboratory investigations at presentation showed a troponin level of 0.02 ng/mL (reference <0.06), CK-MB 25 U/L, D-dimer 0.4 µg/mL, and lactate 0.77 mmol/L. Complete blood count, electrolyte panel, and hepatic and renal function tests were within normal limits.

During monitored follow-up, systolic blood pressure ranged between 110 and 130 mmHg, and pulse rate ranged between 75 and 90/min. Intermittent follow-up ECGs demonstrated newly developed T-wave inversions in leads V1–V3 (Figure 2). Troponin increased to 0.20 ng/mL at the second hour and to 0.44 ng/mL at the third hour.

The patient received 300 mg acetylsalicylic acid orally. She was followed with a preliminary diagnosis of non-ST-elevation myocardial infarction (NSTEMI). Due to worsening chest pain, she underwent coronary angiography at the fourth hour. Coronary angiography demonstrated a long-segment dissection extending from the left main coronary artery (LMCA) to the left anterior descending (LAD) and circumflex (CX) arteries, with TIMI 0 flow. The right coronary artery could not be visualized. According to the Saw angiographic classification, the dissection was assessed as type 2B [6]. Because the dissection flap was visualized on angiography, heparin therapy was not administered.

During the procedure, the patient developed cardiac arrest four times, approximately 5 minutes apart, due to ventricular fibrillation secondary to reperfusion arrhythmia. Pulses were restored after defibrillation. Following resuscitation, two overlapping drug-eluting stents (DES, 3.0 × 28 mm and 3.5 × 18 mm) were implanted in the LAD-LMCA segment. After a brief period of stabilization, the patient was transferred to the intensive care unit; however, she died at the 16th hour because of recurrent ventricular arrhythmias.

DISCUSSION

In the present case, chest pain beginning on postpartum day 30 rapidly progressed to multivessel spontaneous coronary artery dissection extending from the left main coronary artery to the LAD and circumflex artery. The literature indicates that pregnancy-associated SCAD may follow a more severe course than non-pregnancy-related SCAD, with more frequent presentations as STEMI, and with left main coronary artery and multivessel involvement occurring more commonly. In an analysis of 120 contemporary cases by Havakuk et al., the reported rates were 75% for STEMI, 36% for left main coronary artery involvement, and 40% for multivessel involvement [7]. In this respect, our case reflects the high-risk clinical phenotype of postpartum SCAD.

One of the striking features of this case was the absence of marked ECG findings at presentation and the initially normal troponin level. Nevertheless, serial follow-up revealed evolving ECG changes and troponin elevation. The literature emphasizes that SCAD, particularly in young women and in the setting of pregnancy/postpartum status, may initially present with nonspecific findings, which may delay the diagnosis [3,8]. This demonstrates that normal initial tests do not exclude SCAD in postpartum chest pain. Therefore, serial ECGs, serial troponin measurements, and early cardiac evaluation are critically important.

The diagnosis was established by coronary angiography after clinical deterioration, and the lesion was angiographically consistent with a type 2B pattern. The literature identifies coronary angiography as the primary diagnostic modality for SCAD, whereas optical coherence tomography (OCT) and intravascular ultrasound (IVUS) may be helpful in selected cases, particularly when the diagnosis is uncertain [9]. This finding suggests that advanced cardiac imaging should not be delayed in postpartum patients with unexplained chest pain who develop dynamic changes during serial evaluation.

Because the dissection line was clearly visualized on angiography, heparin therapy was not administered, and thrombolytic therapy was not considered. The literature indicates that SCAD has a different pathophysiology from that of classic atherothrombotic acute coronary syndromes and that standard ACS algorithms should not be applied uncritically [4]. In particular, thrombolytic therapy should be considered with caution because it may promote progression of the intramural hematoma and extension of the dissection [4]. Therefore, in young postpartum women with suspected acute coronary syndrome, not only the presence of infarction but also the underlying mechanism should be carefully evaluated.

Because the dissection originated in the LMCA, involved multiple coronary territories, and was accompanied by recurrent ventricular fibrillation during the procedure, percutaneous coronary intervention became mandatory. Although the literature recommends conservative

management in stable SCAD patients without evidence of ongoing ischemia, invasive treatment may be necessary in the presence of left main coronary artery involvement, multivessel disease, ongoing ischemia, malignant arrhythmia, or hemodynamic deterioration [5]. Thus, SCAD management should be individualized according to anatomic extent and clinical stability rather than following a uniform treatment algorithm.

After the diagnosis of dissection was confirmed, the antithrombotic strategy was reassessed, and heparin was not continued. The literature indicates that there is still no clear consensus regarding the type and duration of antiplatelet therapy, especially in conservatively managed SCAD patients; in the DISCO registry, dual antiplatelet therapy was associated with a higher rate of adverse events compared with single antiplatelet therapy [10]. These findings suggest that antithrombotic treatment in SCAD should be approached differently from standard protocols used for atherosclerotic ACS.

Although the patient initially appeared hemodynamically stable, the clinical course rapidly culminated in malignant ventricular arrhythmias and death. The literature has shown that, although many SCAD patients are initially managed conservatively, serious cardiovascular complications may still develop in the early period [11]. Moreover, SCAD is not the only underlying mechanism of pregnancy-associated myocardial infarction; coronary thrombosis, embolism, vasospasm, and atherosclerosis should also be considered in the differential diagnosis [12]. Therefore, in postpartum chest pain, SCAD should be considered early, while other pregnancy-related coronary causes should also be systematically excluded.

This case demonstrates that initial test results may be misleading in young postpartum women presenting with chest pain and that maintaining clinical suspicion is crucial for diagnosis. When interpreted together with the literature, postpartum SCAD appears capable of following a severe course, particularly in the presence of multivessel and left main coronary artery involvement. Therefore, the key approach for the emergency physician should consist of early clinical suspicion, serial reassessment, timely advanced cardiac imaging, and cautious management.

CONCLUSION

Postpartum SCAD is a rare but fatal cause of sudden-onset chest pain in young women. Normal ECG and troponin results at the initial evaluation do not exclude the diagnosis. In the emergency department, maintaining a high index of suspicion in this patient group, together with serial troponin assessment, early consultation, and timely coronary angiography planning, may help prevent diagnostic delay.

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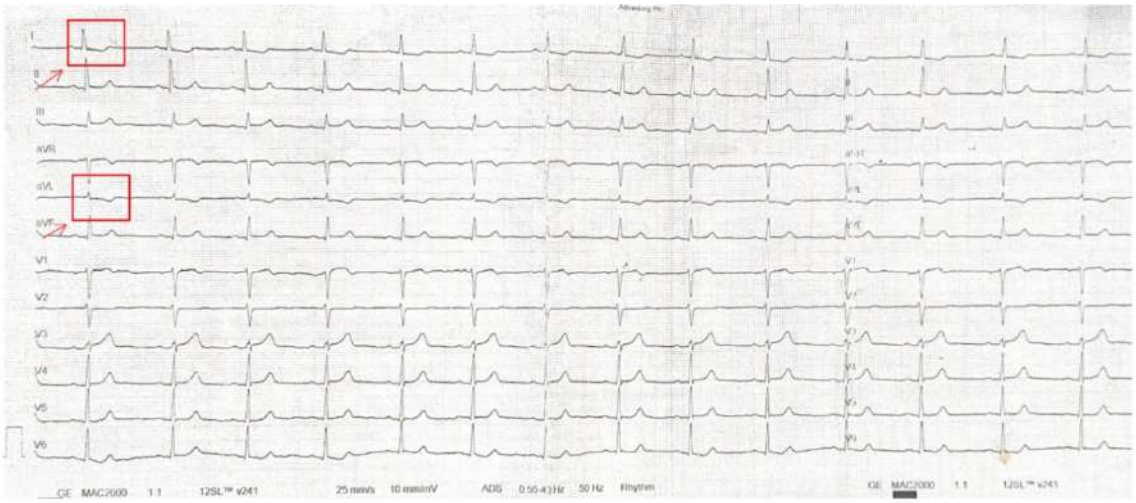

The red arrow and rectangular box indicate minimal ST segment depression in the DI-aVL derivations.

Figure 3. First ECG

The red rectangle contains newly developed T-wave negativity in derivations V1-2-3.

Figure 4. Control ECG

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Central Retinal Artery Occlusion Presenting with Sudden Vision Loss: A Case Report

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Introduction

Central retinal artery occlusion (CRAO) is an ophthalmic emergency characterized by sudden, painless, and severe vision loss, most commonly affecting elderly individuals with underlying vascular risk factors such as hypertension, diabetes mellitus, and atherosclerotic cardiovascular disease (1). Approximately 95% of CRAO cases are nonarteritic, while the remaining 5% are arteritic and usually related to inflammatory disorders, particularly giant cell arteritis (2). It is considered the ocular equivalent of acute ischemic stroke due to its shared thromboembolic mechanisms and the need for urgent evaluation (3).

In this report, we present a case of acute unilateral vision loss due to CRAO in a patient with multiple vascular risk factors and recent discontinuation of antiplatelet therapy, highlighting the importance of etiological evaluation and secondary prevention.

Case:

A 70-year-old female was admitted to the emergency department with sudden onset, painless vision loss in the left eye. She reported a similar episode of vision loss in the same eye two days prior, lasting approximately two hours. Additionally, she described intermittent brief episodes of pain over the left temporal region in the preceding two days. Her past medical history was significant for diabetes mellitus and coronary artery disease, and an intracranial aneurysm treated surgically 10 years earlier. The patient reported discontinuation of acetylsalicylic acid (ASA-100mg) 10 days prior to presentation.

On examination, the patient was conscious, oriented, and hemodynamically stable. Neurological examination revealed severe visual impairment in the left eye, with inability to perceive hand motion, while the right eye was intact. Confrontation testing demonstrated complete visual field loss in all quadrants of the left eye. Direct light reflex was present in the right eye, while no response was elicited in the left eye. Extraocular movements of the left eye were limited in upward and downward gaze, with medial deviation, consistent with prior deficits due to aneurysm. No other focal neurological deficits were identified. Initial vital

signs were as follows: blood pressure (BP): 171/67 mmHG, pulse: 72/min, oxygen saturation on room air: 97%. Ophthalmologic evaluation demonstrated retinal edema and optic disc pallor, raising suspicion for acute central retinal artery occlusion (CRAO). Ultrasonography of the temporal artery was performed to differentiate giant cell arteritis; however, no diagnostic findings were obtained. Computed tomography angiography revealed calcified plaques causing approximately 40% stenosis of the left carotid artery and 30% on the right, suggesting a possible embolic source.The patient's inital laboratory results were as follows: eGFR: 85, AST: 11 U/L, ALT: 13 U/L, LDH: 186 U/L, CRP 12.30 mg/L, hemoglobin (HGB): 11.1 g/dL, red blood cells (RBC): 4.58 million/uL, white blood cells (WBC): 9.91 10*3/uL, platelets (PLT): 448 10*3/uL, erythrocyte sedimentation rate (ESR): 31 mm/h. Cardiac biomarkers were elevated (troponin: 2202) and were attributed to the overall acute clinical condition rather than primary cardiac pathology.

Clinically, the patient was diagnosed with acute central retinal artery occlusion affecting the left eye, and hyperbaric oxygen therapy was recommended.

Discussion

Central retinal artery occlusion (CRAO) is a vision-threatening emergency. Patients typically present with sudden, painless, and profound unilateral vision loss, as seen in our case. The presence of vascular risk factors such as diabetes mellitus and coronary artery disease, along with recent discontinuation of antiplatelet therapy, likely contributed to an embolic event. Carotid imaging in this patient revealed moderate stenosis, unstable atherosclerotic plaques may serve as a source of microembolization leading to retinal artery occlusion.

An important consideration in the differential diagnosis is arteritic CRAO, commonly associated with giant cell arteritis, which requires urgent corticosteroid therapy (4). In our patient, the absence of supportive clinical and imaging findings made this diagnosis unlikely. Management of CRAO remains challenging; however, early initiation of hyperbaric oxygen therapy may provide potential benefit (5).

This case underscores the importance of rapid diagnosis, thorough etiological evaluation, and initiation of secondary prevention strategies, particularly antiplatelet therapy, to reduce the risk of further ischemic events.

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The Role of Oral Contraceptive Use and Recent Surgery in Mortality Among Female Pulmonary Embolism Patients Under 35 Years of Age

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Abstract

Objective:
Pulmonary embolism (PE) is a life-threatening clinical condition associated with substantial morbidity and mortality. Although oral contraceptive (OC) use and recent surgical procedures are well-established risk factors for the development of PE, their impact on mortality, particularly in young female patients, remains unclear. This study aimed to investigate the effects of OC use and recent surgical history on short-term mortality in female patients under 35 years of age diagnosed with PE.

Materials and Methods:
This retrospective observational study was conducted in the Emergency Department of Malatya Training and Research Hospital. Female patients younger than 35 years who were diagnosed with PE between September 2020 and September 2025 were screened. Of the 57 patients identified through ICD codes, 19 patients with radiologically confirmed PE and sufficient clinical data were included in the study. OC use was defined as having a prescription for oral contraceptives for at least three months before the diagnosis of PE, while recent surgery was defined as any surgical procedure performed within the previous six months. Demographic characteristics, laboratory findings, and blood gas parameters were analyzed. The primary outcome was 30-day mortality. Appropriate parametric and non-parametric statistical tests were used, and a p-value <0.05 was considered statistically significant.

Results:
Among the 19 patients included in the study, 3 (15.8%) died and 16 (84.2%) were discharged alive. OC use was present in 31.6% of patients, while 15.8% had a history of recent surgery. In mortality analyses, only serum albumin levels differed significantly between survivors and non-survivors. Patients who died had significantly lower albumin levels than survivors (2.97 ± 0.32 g/dL vs. 3.66 ± 0.51 g/dL, $p=0.040$). No significant associations were found between mortality and other laboratory or blood gas parameters (all $p>0.05$). Furthermore, neither OC use nor recent surgical history was significantly associated with mortality ($p=0.517$ and $p=0.422$, respectively). Although mortality appeared to be higher among patients who did not use OCs, this difference did not reach statistical significance.

Conclusion:
In female patients under 35 years of age with pulmonary embolism, oral contraceptive use and recent surgical history, despite being important risk factors for disease development, do not appear to be significant determinants of short-term mortality. In contrast, low serum albumin levels emerged as a potential prognostic marker in this patient population. These findings suggest that mortality in young PE patients may be more closely related to disease severity and physiological reserve than to the presence of traditional provoking risk factors. Larger multicenter studies are needed to validate these results.

Keywords:
Pulmonary embolism; oral contraceptives; surgery; mortality; albumin; young women.

Introduction

Pulmonary embolism (PE) is a life-threatening condition representing the most severe clinical manifestation of venous thromboembolism and is associated with high mortality when not diagnosed promptly.[1] Although it can occur in all age groups, its unexpected presentation in young and otherwise healthy individuals may lead to diagnostic delays and unfavorable clinical outcomes.[2] Mortality rates related to PE can reach up to 10% within the first 30 days, highlighting the importance of identifying early risk factors.[1]

The development of PE in women is associated with risk factors that differ from those in men. In particular, hormonal factors, pregnancy, the postpartum period, and oral contraceptive (OC) use are among the most important female-specific risk determinants.[3] Combined oral contraceptives have been shown to increase the risk of venous thromboembolism by two- to four-fold due to their procoagulant effects on the hemostatic system.[4] Furthermore, substantial evidence suggests that the use of third- and fourth-generation oral contraceptives may increase the risk of fatal pulmonary embolism.[5]

In addition, transient risk factors such as surgical procedures and immobilization play a critical role in the development of PE. Recent surgical interventions, in particular, promote thrombosis through endothelial injury and venous stasis, thereby significantly increasing the risk of PE.[3,6] When these risk factors coexist, both the frequency and severity of thromboembolic events may further increase.

Although PE is often perceived as a relatively low-risk condition in young women, serious and even fatal outcomes may occur in the presence of modifiable risk factors such as OC use and recent surgical history.[2,3] However, studies evaluating the combined impact of these risk factors on mortality, particularly among women younger than 35 years of age, remain limited.

Therefore, the present study aimed to investigate the effects of oral contraceptive use and recent surgical history on mortality among female patients under 35 years of age diagnosed with pulmonary embolism.

Materials and Methods

This retrospective observational study was conducted in the Emergency Department of Malatya Training and Research Hospital. We retrospectively reviewed the medical records of 57 female patients who were assigned an International Classification of Diseases (ICD) code for pulmonary embolism (PE) between September 2020 and September 2025. To ensure diagnostic accuracy, all imaging findings were re-evaluated in consultation with a radiologist. Following this review, 19 patients with radiologically confirmed PE who met the inclusion and exclusion criteria were included in the study.

Only female patients younger than 35 years of age with radiologically confirmed pulmonary embolism were included. Patients with incomplete data, unconfirmed diagnoses, or insufficient clinical records were excluded from the study.

Demographic characteristics, clinical data, and laboratory parameters were obtained from the hospital information management system. Oral contraceptive (OC) use was defined as having an active OC prescription for at least three months prior to the diagnosis of pulmonary embolism. Recent surgical history was defined as any surgical procedure performed within six months before the diagnosis of PE. The primary study endpoint was 30-day mortality.

The following variables were evaluated: age (years), OC use (yes/no), recent surgical history (yes/no), and mortality status (survivor/non-survivor). All laboratory and blood gas parameters were analyzed using the initial values obtained at the time of PE diagnosis. These included albumin (g/dL), C-reactive protein (CRP, mg/L), white blood cell count (WBC, $\times 10^9$/L), platelet count (PLT, $\times 10^9$/L), hemoglobin (Hb, g/dL), D-dimer (ng/mL), and troponin (ng/L) levels, as well as partial pressure of oxygen (pO_2, mmHg), partial pressure of carbon dioxide (pCO_2, mmHg), pH, and lactate (mmol/L).

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 27 (IBM Corp., Armonk, NY, USA).

The distribution of continuous variables was assessed using normality tests. Variables with a normal distribution were presented as mean ± standard deviation (SD), whereas non-normally distributed variables were expressed as median and interquartile range (IQR).

Associations between mortality and clinical or laboratory parameters were evaluated using appropriate parametric or non-parametric statistical tests according to data distribution.

The Chi-square test was used to assess relationships between categorical variables. In particular, the associations of mortality with oral contraceptive use and recent surgical history were analyzed, and risk assessments were performed.

A two-tailed p-value <0.05 was considered statistically significant for all analyses.

Results

Data from a total of 19 patients were analyzed. Of these, 3 patients (15.8%) died, while 16 patients (84.2%) were discharged alive.

A history of oral contraceptive (OC) use was present in 6 patients (31.6%), whereas 13 patients (68.4%) had no history of OC use. Recent surgical history was identified in 3 patients (15.8%), while 16 patients (84.2%) had not undergone surgery within the defined period.

Normality analyses demonstrated that the following variables were normally distributed: age (years) ($p=0.056$), albumin (g/dL) ($p=0.902$), white blood cell count (WBC, $\times 10^9$/L) ($p=0.192$), platelet count (PLT, $\times 10^9$/L) ($p=0.451$), hemoglobin (Hb, g/dL) ($p=0.938$), and partial pressure of oxygen (pO_2, mmHg) ($p=0.063$).

The following variables did not demonstrate a normal distribution: C-reactive protein (CRP, mg/L) ($p=0.001$), partial pressure of carbon dioxide (pCO_2, mmHg) ($p=0.005$), pH ($p=0.001$), lactate (mmol/L) ($p<0.001$), troponin (ng/L) ($p<0.001$), and D-dimer (ng/mL) ($p=0.037$).

Variables with a normal distribution are presented as mean ± standard deviation (SD), whereas non-normally distributed variables are presented as median and interquartile range (IQR). Detailed baseline characteristics and laboratory findings are presented in Table 1.

Table 1. Demographic and Laboratory Characteristics of the Study Population

Variables	n	Mean	SD	Median	IQR	
					25%	75%
Age (yıl)	19	29,1	±1,1			
Albumin (g/L)		3,5	±0,1			
CRP (mg/L)				2,7	0,7	5,9
WBC (×10⁹/L)		12,1	±0,9			
Plt (×10⁹/L)		279,1	±14,7			
Hb (g/dL)		12,7	±0,4			
pO_2 (mmHg)		34,8	±3,6			
pCO_2 (mmHg)				37	34	42
pH				7,38	7,36	7,42
Lactat (mmol/L)				1,8	1,4	3,8
Troponin (ng/L)				0,1	0,1	0,3
D-Dimer (ng/mL)				2,7	1,7	6

In the comparison between survivors and non-survivors, we found that only serum albumin levels differed significantly between the groups. Patients who died had significantly lower albumin levels than those who survived (2.97 ± 0.32 g/dL vs. 3.66 ± 0.51 g/dL, p=0.040).

No statistically significant differences were observed between mortality and the following parameters: age (p=0.506), WBC count (p=0.964), platelet count (p=0.868), hemoglobin level (p=0.128), pO_2 (p=0.361), D-dimer (p=0.737), troponin (p=0.618), lactate (p=0.501), pH (p=0.400), pCO_2 (p=0.499), and CRP (p=0.264).

When the relationship between recent surgical history and mortality was evaluated, the mortality rate appeared to be higher among patients with a history of recent surgery; however, this difference did not reach statistical significance (Fisher's exact test, p=0.422).

Similarly, when the association between oral contraceptive (OC) use and mortality was examined, none of the patients who died had a history of OC use. Nevertheless, this difference was not statistically significant (Fisher's exact test, p=0.517).

Risk analysis demonstrated a numerically higher mortality risk among patients who did not use oral contraceptives; however, this association was not statistically significant (RR=0.625, 95% CI: 0.428–0.914).

Discussion

In this study, the mortality rate among female patients under 35 years of age with pulmonary embolism was 15.8%. This finding is consistent with previously reported early mortality rates and demonstrates that pulmonary embolism remains a potentially fatal condition even in young patients.[1,7] Large emergency department–based studies have reported PE-related mortality rates ranging between 10% and 20%.[1,7]

In our cohort, 31.6% of patients had a history of oral contraceptive (OC) use, while 15.8% had undergone recent surgical procedures. Both OC use and surgery are well-established major risk factors for venous thromboembolism and play a significant role in the development of PE, particularly among young women.[3,4,6] Our findings support the presence of these recognized risk factors within our study population.

Normality analyses demonstrated that age, albumin, WBC count, platelet count, hemoglobin level, and pO_2 were normally distributed, whereas CRP, pCO_2, pH, lactate, troponin, and D-dimer levels showed non-normal distributions. Previous studies have reported that biomarkers reflecting inflammation and tissue perfusion, such as CRP, lactate, and troponin, may exhibit substantial heterogeneity in patients with acute PE, potentially reflecting differences in disease severity and clinical presentation.[8,9]

When survivors and non-survivors were compared, serum albumin was the only parameter that differed significantly between the groups. Albumin levels were significantly lower among patients who died (p=0.040). In contrast, no significant associations were observed between mortality and the remaining clinical or laboratory variables (all p>0.05).

Several studies have demonstrated that hypoalbuminemia is independently associated with mortality in patients with pulmonary embolism and may contribute to risk stratification.[10] Likewise, serum albumin has been reported as a strong predictor of both short- and long-term mortality in acute PE.[11] The finding that albumin was the only parameter associated with mortality in our study suggests that markers reflecting physiological reserve and systemic inflammatory status may have greater prognostic value than conventional laboratory parameters in younger patient populations.

Although mortality appeared to be higher among patients with a recent history of surgery, this difference did not reach statistical significance (p=0.422). Similarly, no significant association was found between OC use and mortality (p=0.517). Although mortality appeared numerically higher among patients who were not using OCs, this observation did not achieve statistical significance.

Oral contraceptive use and recent surgical procedures are recognized as major risk factors for venous thromboembolism and pulmonary embolism.[3–7] However, while their role in the development of PE is well established, their direct impact on mortality after PE has occurred remains less clear. Contemporary studies suggest that PE prognosis is primarily determined by factors such as hemodynamic instability, right ventricular dysfunction, and cardiac biomarkers, whereas provoking risk factors play a more limited role in predicting mortality.[7,12,13]

Taken together, our findings indicate that although oral contraceptive use and recent surgery are important risk factors for the development of pulmonary embolism, they do not appear to be direct determinants of mortality in young female patients. These results suggest that prognosis in this population may be driven more by disease severity and physiological reserve than by the presence of traditional provoking risk factors.

Limitations

The present study has several limitations. First, its retrospective and single-center design may limit the generalizability of the findings. Second, the relatively small sample size reduced the statistical power of the analyses, particularly for categorical comparisons, and may have limited our ability to detect potentially significant associations. Therefore, the results should be interpreted with caution and validated in larger, multicenter prospective studies.

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The Trap of Atypical Extremity Pain: Recognizing Hidden Substance Misuse in Complicated Soft Tissue Infections

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INTRODUCTION

The buprenorphine/naloxone combination, widely used in the treatment of substance use disorder, may be misused via intravenous administration (1–3). This practice can lead to serious complications, including local infection, abscess formation, necrotizing soft tissue infections, thrombophlebitis, and deep vein thrombosis (4,5). The risk is further increased by non-sterile injection techniques and the use of inappropriate solvents (6).

In this report, we present a case of complicated soft tissue infection accompanied by venous thrombosis following intravenous Suboxone injection.

CASE PRESENTATION

A 26-year-old male patient with a known history of substance use disorder treatment presented with swelling and pain in the left upper extremity after dissolving prescribed Suboxone in a spoon and administering it intravenously through the antecubital region.

At admission, laboratory findings revealed: C-reactive protein (CRP): 175 mg/L, White blood cell count (WBC): 12,000/mm³. During follow-up, CRP decreased to 85 mg/L.

Upper extremity venous Doppler ultrasonography demonstrated:Patent subclavian, radial, and ulnar veins.No thrombosis in the basilic vein.Chronic recanalized thrombus in the cephalic vein. A hypoechoic area measuring 40×27×80 mm in the medial arm (suggestive of hematoma/abscess). Acute, near-total occlusive thrombus in the axillary and brachial veins.

The patient underwent surgical debridement by the orthopedic team, and cultures were obtained. Compartment monitoring was initiated.

The infectious diseases team recommended triple antibiotic therapy with meropenem, vancomycin, and metronidazole.

The cardiovascular surgery team advised low-molecular-weight heparin therapy, while the plastic surgery team anticipated the potential need for microsurgical intervention.

DISCUSSION

Intravenous substance use is associated with severe infectious and vascular complications. Intravenous use of Suboxone increases the risk of endothelial injury and thrombosis (1,4,7).

The coexistence of acute and chronic thrombosis in this case suggests repeated vascular trauma. Soft tissue infections and abscess formation are also commonly observed in such patients (7–9).

Reported complications in the literature include: Septic thrombophlebitis. Deep vein thrombosis. Necrotizing infections. Tissue loss (10,11)

Management primarily involves:Early initiation of antibiotic therapy, Surgical debridement. Anticoagulation (12)

CONCLUSION

The coexistence of acute and chronic thrombosis observed in this case demonstrates that vascular damage in patients with substance use disorder is cumulative, and repeated injections may lead to irreversible deterioration of the venous system.

Management of such complex cases requires a multidisciplinary approach, as single-specialty intervention is often insufficient. Early initiation of broad-spectrum empirical antibiotic therapy, aggressive surgical debridement, anticoagulation to prevent pulmonary embolism, and the use of microsurgical techniques for tissue reconstruction are critical to improving prognosis and preventing amputation.

In conclusion, follow-up of patients undergoing addiction treatment should not be limited to psychiatric support alone. Patients must be thoroughly informed about the medical risks associated with improper medication use.

Healthcare professionals should consider hidden substance misuse in the differential diagnosis of patients presenting with atypical upper extremity pain and signs of infection, and should promptly utilize vascular and radiological imaging methods.

Intravenous Suboxone use can result in severe infections and thrombosis. Early and multidisciplinary management is essential for improving outcomes. Advanced cases may require microsurgical intervention.

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"From Self to System: A Multidimensional Analysis of Well-Being and Burnout Among Intern Physicians"

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Introduction

Emergency departments (EDs) represent one of the most demanding clinical environments in modern healthcare systems, characterized by high patient acuity, unpredictable workload, time pressure, and frequent exposure to critical decision-making. Intern physicians, who are at an early stage of their professional development, are particularly vulnerable to the cumulative effects of these stressors.

Physician well-being has traditionally been conceptualized through the lens of individual resilience, emphasizing personal coping mechanisms, stress tolerance, and self-care behaviors. However, recent literature increasingly recognizes burnout as a multifactorial phenomenon influenced not only by individual traits but also by organizational and systemic determinants. Burnout, defined by emotional exhaustion, depersonalization, and reduced personal accomplishment, is highly prevalent among emergency medicine professionals and has been associated with adverse outcomes including decreased job performance, medical errors, and reduced quality of patient care (1).

Emerging evidence suggests that structural factors such as workload intensity, shift patterns, institutional support, team communication, and workplace culture play a critical role in shaping physician well-being (2). Additionally, personality traits and individual differences may modulate susceptibility to burnout; however, these factors appear to interact with environmental stressors rather than act independently (3).

In this context, a comprehensive, multidimensional evaluation of well-being that integrates both individual and system-level factors is essential. The present study aimed to assess intern physicians' well-being in the emergency department using a structured framework encompassing personal well-being, system-related conditions, and burnout.

Furthermore, the study sought to determine the relative contributions of these domains and identify independent predictors of burnout.

Material and Methods

This prospective cross-sectional study was conducted at a tertiary academic hospital with a high-volume emergency department. The study population consisted of intern physicians who completed their mandatory ED rotation during the study period. Participation was voluntary, and all respondents provided informed consent prior to inclusion. Exclusion criteria were incomplete questionnaire responses and refusal to participate. A total of 71 intern physicians were included in the final analysis. Data were collected a structured, self-administered questionnaire consisting of 20 items. The questionnaire was developed based on the wellness framework proposed by the American College of Emergency Physicians (ACEP), which emphasizes a holistic approach to physician well-being incorporating both individual and organizational dimensions.

The questionnaire comprised three main domains:

Personal Well-Being and Self-Care (e.g., sleep quality, stress management, physical activity, emotional resilience)

System-Related Factors (e.g., workload, team communication, supervision quality, institutional support, psychological safety)

Burnout (assessing emotional exhaustion and work-related fatigue)

All items were measured using a 5-point Likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). Domain scores were calculated by summing the responses within each category, with higher scores indicating greater levels of the measured construct.

Statistical analyses were performed using standard statistical software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between groups were conducted using appropriate parametric or non-parametric tests based on data distribution. Correlation analyses were performed using Pearson or Spearman correlation coefficients to assess relationships between domain scores. To identify independent predictors of burnout, a multivariable linear regression model was constructed. Variables included in the model were personal well-being scores, system-related scores, age, and gender. Regression coefficients (β) and p-values were reported, with statistical significance defined as $p < 0.05$.

Results

A total of 71 intern physicians were included in the study. Of these, 37 (52.1%) were male and 33 (46.5%) were female. The mean scores were 26.37 ± 5.40 for personal well-being and self-care, 16.42 ± 4.70 for system-related factors, and 15.27 ± 3.85 for burnout. Gender-based comparisons revealed that male participants had significantly higher self-care scores than females (28.11 ± 5.73 vs. 24.33 ± 4.34, p=0.003). Similarly, total well-being scores were significantly higher in males (61.00 ± 12.05 vs. 54.33 ± 10.42, p=0.016). No statistically significant difference was observed between genders in system-related factors (p=0.331). Burnout scores showed a borderline difference, with higher values observed in males (16.08 ± 3.69 vs. 14.27 ± 3.89, p=0.050). Correlation analysis demonstrated a moderate association between personal well-being and burnout (r=0.49, p<0.001), while system-related factors exhibited a stronger correlation with burnout (r=0.59, p<0.001). A multivariable linear regression model was constructed to identify independent predictors of burnout scores. Variables included personal well-being scores, system-related scores, age, and gender. In multivariable linear regression analysis, system-related factors remained a significant independent predictor of burnout (β = 0.48, p < 0.001), whereas personal well-being showed a weaker but still significant association (β = 0.29, p = 0.012). Gender was not an independent predictor after adjustment (p > 0.05).

Table 1. Demographic Characteristics of Participants

Variable	n	%
Male	37	52.1
Female	33	46.5
Unspecified	1	1.4

Table 2. Descriptive Statistics of Scale Scores

Variable	Mean ± SD	Median (IQR)
Group 1 (Self-care)	26.37 ± 5.40	27 (22–30)
Group 2 (System)	16.42 ± 4.70	16 (13–19)
Group 3 (Burnout)	15.27 ± 3.85	16 (13–18)
Total Score	58.06 ± 11.75	57 (49.5–66.5)

Table 3. Comparison of Scale Scores by Gender

Variable	Male (Mean±SD)	Female (Mean±SD)	p-value
Group 1 (Self-care)	28.11 ± 5.73	24.33 ± 4.34	0.003
Group 2 (System)	16.81 ± 4.77	15.73 ± 4.44	0.331
Group 3 (Burnout)	16.08 ± 3.69	14.27 ± 3.89	0.050
Total Score	61.00 ± 12.05	54.33 ± 10.42	0.016

Table 4. Correlation Analysis Between Scale Scores

Variables	**r**	**p-value**	**Interpretation**
Self-care vs Burnout	0.49	<0.001	Moderate
System vs Burnout	0.59	<0.001	Strong

Discussion

This study provides a comprehensive multidimensional assessment of intern physician well-being in the emergency department, highlighting the relative contributions of individual and system-level determinants. The findings demonstrate that although personal well-being is associated with burnout, system-related factors exert a stronger and independent effect.

These results are consistent with the growing body of literature emphasizing that burnout in emergency medicine is not solely an individual-level phenomenon but is strongly influenced by structural and organizational factors (2). A large meta-analysis has shown that burnout is highly prevalent among emergency physicians and is closely linked to workplace conditions rather than individual resilience alone (1).

Traditionally, interventions targeting physician well-being have focused on individual strategies such as mindfulness, stress management, and self-care. However, our findings suggest that such approaches may be insufficient in high-intensity environments like the ED, where systemic pressures dominate. Instead, institutional strategies addressing workload, staffing, workflow efficiency, and organizational culture may have a greater impact.

The observed gender differences, particularly lower self-care scores among female participants, may reflect additional psychosocial responsibilities or differences in coping strategies. Nevertheless, the lack of an independent association between gender and burnout

in multivariable analysis suggests that systemic conditions may outweigh individual demographic characteristics. This observation aligns with previous findings indicating that personality traits and individual differences interact with environmental stressors rather than independently determining burnout risk (3).

Importantly, the stronger correlation between system-related factors and burnout underscores the need for targeted institutional interventions. Factors such as workload intensity, team dynamics, quality of supervision, and psychological safety appear central to shaping intern physicians' experiences.

Intern physician well-being in the emergency department is influenced by both individual and system-level factors, with system-related conditions demonstrating a stronger and independent association with burnout. These findings support a paradigm shift from individual-focused interventions toward system-based strategies aimed at improving working conditions, team dynamics, and institutional support mechanisms.

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4770 **Admission hyperglycemia and a composite glucose–age–blood pressure score for predicting DWI-confirmed ischemic stroke in the emergency department**

Running title: Hyperglycemia and DWI-confirmed stroke

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Highlights

- Admission glucose ≥ 140 mg/dL doubled the odds of DWI-confirmed ischemic stroke.
- A clear dose–response gradient was seen across four glucose categories for both DWI positivity and ICU admission.
- A composite glucose–age–SBP (GAS) score achieved an AUC of 0.81 for DWI positivity.
- The full multivariate model outperformed glucose alone (AUC 0.85 vs 0.65).
- Three routine emergency-department parameters can support early stroke risk stratification.

Abstract

Background: Admission hyperglycemia is consistently associated with adverse outcomes in acute ischemic stroke, but most studies have used clinical severity scales rather than radiologically confirmed injury as the outcome. Diffusion-weighted imaging (DWI) is the reference standard for early detection of acute ischemia, and DWI-stratified glucose analyses in the emergency department (ED) remain limited.

Objective: To investigate the association between admission blood glucose and DWI-confirmed ischemic stroke in ED patients with suspected stroke, evaluate the dose–response relationship across glucose categories, and explore whether combining glucose with age and systolic blood pressure (SBP) improves prediction.

Methods: Single-center, retrospective cohort of 322 consecutive adults presenting to a tertiary ED with suspected stroke between January 2021 and December 2024 who underwent admission glucose measurement and DWI during the index visit. The primary outcome was DWI positivity; the secondary outcome was intensive care unit (ICU) admission. Patients were stratified into four glucose categories (< 110, 110–139, 140–179, and ≥ 180 mg/dL). A composite Glucose–Age–SBP (GAS) score was calculated as (Glucose × Age × SBP) / 1,000,000 and compared with glucose alone using ROC analysis. Multivariate logistic regression was adjusted for age, SBP, neutrophil-to-lymphocyte ratio, creatinine, and sex.

Results: DWI positivity was present in 174 of 322 patients (54.0 %). Median admission glucose was higher in DWI-positive than DWI-negative patients (141 vs 119 mg/dL, $p < 0.001$). DWI positivity rose stepwise across glucose categories from 41.6 % (< 110 mg/dL) to 47.7 %, 59.2 %, and 75.0 % (≥ 180 mg/dL) (p for trend < 0.001). Glucose ≥ 140 mg/dL increased the odds of DWI positivity 2.48-fold (95 % CI 1.57–3.92, $p < 0.001$). In multivariate analysis, glucose remained an independent predictor (adjusted OR per 10 mg/dL 1.05, 95 % CI 1.01–1.10, $p = 0.016$). The multivariate model achieved an AUC of 0.851; the GAS composite score reached 0.809, both substantially exceeding glucose alone (AUC 0.647). ICU admission showed a parallel gradient (18.0 % to 40.6 %, $p = 0.014$).

Conclusion: Admission hyperglycemia independently predicts DWI-confirmed ischemic stroke with a clear dose–response. Combining glucose with age and SBP — three universally available ED parameters — markedly improves predictive accuracy and may support early neuroimaging prioritization. Prospective multicenter validation is warranted before clinical deployment.

Keywords: stroke; hyperglycemia; diffusion-weighted imaging; emergency department; blood glucose; risk stratification; composite score

1. Introduction

Stroke remains the second leading cause of death and the third leading cause of disability-adjusted life years worldwide, with ischemic stroke accounting for approximately 87 % of all cases [1,2]. In the emergency department (ED), rapid identification of confirmed ischemic stroke is essential for initiating time-sensitive reperfusion therapies [3]. Diffusion-weighted magnetic resonance imaging (DWI) has emerged as the reference standard for early detection of acute ischemic injury, with sensitivity exceeding 90 % within the first hours of symptom onset [4,5].

Admission hyperglycemia, defined variably as blood glucose above 126–180 mg/dL, occurs in 40–60 % of patients presenting with acute ischemic stroke [6,7]. Both stress-induced and diabetic hyperglycemia have been consistently associated with larger infarct volumes, increased hemorrhagic transformation, and higher mortality [7–9]. A dose–response meta-analysis demonstrated that each 1 mmol/L increase in admission glucose was associated with a 10 % increase in the risk of adverse outcomes [10]. Yet most studies have relied on clinical stroke severity scales or functional endpoints as outcome measures; only a handful have used DWI findings to objectively quantify the relationship between admission glucose and confirmed ischemic injury in the ED setting [11,12].

Whether combining glucose with other readily available ED parameters — specifically age and systolic blood pressure (SBP), both of which are established independent predictors of stroke severity — improves the identification of DWI-positive ischemic stroke has not been examined. We therefore aimed to (i) investigate the association between admission blood glucose and DWI-confirmed ischemic stroke in ED patients with suspected stroke, (ii) evaluate a dose–response relationship across predefined glucose categories, and (iii) explore whether a composite score integrating glucose, age, and SBP enhances predictive accuracy beyond glucose alone.

2. Methods

2.1. Study design and setting

This was a retrospective, single-center, observational cohort study conducted at the ED of Mersin City Training and Research Hospital, a tertiary care center in southern Turkey serving an urban catchment of approximately 1.9 million people. The study included consecutive patients who presented to the ED between January 2021 and December 2024 with suspected acute stroke and who underwent both non-contrast computed tomography (CT) and DWI during their ED evaluation. The study was conducted in accordance with the principles of the Declaration of Helsinki. Reporting follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

2.2. Study population

Patients aged 18 years or older who presented with acute neurological symptoms suggestive of stroke and had both an admission blood glucose measurement and DWI performed within the index ED visit were eligible for inclusion. Exclusion criteria were: (i) traumatic brain injury as the etiology of neurological symptoms, (ii) absence of DWI data, (iii) primary hemorrhagic stroke confirmed on initial CT, and (iv) missing key data precluding analysis. A total of 335 consecutive patients were identified through the electronic medical record system; after excluding 13 patients with incomplete or missing DWI data, the final analysis cohort comprised 322 patients.

2.3. Data collection

Demographic, clinical, laboratory, imaging, and disposition data were extracted from the electronic medical record by trained investigators using a standardized data collection form. The following variables were recorded:

Demographics: age and sex.

Vital signs at presentation: systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, and peripheral oxygen saturation (SpO_2).

Laboratory values: admission blood glucose (mg/dL), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), international normalized ratio (INR), and serum creatinine.

Time intervals: symptom-to-door time, door-to-CT time, and door-to-MRI time.

Imaging findings: presence of early ischemic changes on non-contrast CT, hyperdense middle cerebral artery (MCA) sign, CT angiography (CTA) vessel occlusion, and DWI positivity (defined as restricted diffusion consistent with acute ischemic injury, as interpreted by the on-call neuroradiologist).

Treatment and disposition: administration of intravenous tissue plasminogen activator (IV tPA), mechanical thrombectomy, and intensive care unit (ICU) admission during the index hospitalization.

2.4. Outcome definitions

The primary outcome was DWI positivity, defined as the presence of restricted diffusion on DWI consistent with acute ischemic stroke. The secondary outcome was ICU admission during the index hospitalization, used as a surrogate for in-hospital severity.

2.5. Composite score

An exploratory composite score — the Glucose–Age–SBP (GAS) score — was calculated as (Glucose [mg/dL] × Age [years] × SBP [mmHg]) / 1,000,000. This composite was derived post-hoc from the study data as a secondary analysis without a separate derivation cohort; it was examined to determine whether combining three readily available ED parameters could improve prediction of DWI positivity compared with glucose alone. A multiplicative formulation was chosen on the rationale that hyperglycemia, advanced age, and elevated blood pressure may interact synergistically rather than additively in conferring ischemic risk.

2.6. Statistical analysis

Continuous variables were assessed for normality using the Kolmogorov–Smirnov test. Normally distributed variables are reported as mean ± standard deviation (SD) and compared using the independent-samples t test; non-normally distributed variables are reported as median (interquartile range, IQR) and compared using the Mann–Whitney U test. Categorical variables are reported as frequency (percentage) and compared using the chi-square test or Fisher exact test, as appropriate.

To evaluate the dose–response relationship between admission glucose and outcomes, patients were stratified into four predefined categories: < 110, 110–139, 140–179, and ≥ 180 mg/dL. The Cochran–Armitage trend test and Spearman rank correlation were used to assess linear trends across categories.

Univariate logistic regression was performed for each candidate predictor. Variables with $p < 0.10$ in univariate analysis or with established clinical relevance were entered into a multivariate logistic regression model. Results are reported as odds ratios (OR) with 95 % confidence intervals (CI). The events-per-variable ratio was maintained above 10 to ensure model stability (174 events / 6 variables = 29).

Discriminative ability was evaluated using receiver operating characteristic (ROC) curves. The area under the curve (AUC) was calculated for (i) glucose alone, (ii) the GAS composite score, and (iii) the multivariate prediction model. Optimal cutoff values were determined using Youden's J statistic, and sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated at the identified cutoffs.

All statistical analyses were performed using Python 3.12 with SciPy 1.14, Statsmodels 0.14, and Scikit-learn 1.5. A two-tailed p value of < 0.05 was considered statistically significant.

3. Results

3.1. Baseline characteristics

Of 335 patients screened, 322 met the inclusion criteria and comprised the analysis cohort. The median age was 64 years (IQR 49–74), and 173 (53.7 %) were male. DWI positivity —

indicating confirmed acute ischemic stroke — was present in 174 patients (54.0 %). The overall median admission glucose was 130 mg/dL (IQR 107–167), and 94 patients (29.2 %) required ICU admission.

Patients with DWI-positive stroke were significantly older (median 70 vs 53 years, $p < 0.001$), had higher SBP (median 162 vs 131 mmHg, $p < 0.001$), and higher admission glucose (median 141 vs 119 mg/dL, $p < 0.001$) compared with DWI-negative patients. DWI-positive patients also had higher rates of early ischemic changes on CT (43.9 % vs 0.7 %, $p < 0.001$), CTA vessel occlusion (40.4 % vs 1.4 %, $p < 0.001$), and ICU admission (49.4 % vs 5.4 %, $p < 0.001$). Heart rate, MLR, and INR did not differ significantly between groups. Complete baseline characteristics are presented in Table 1.

3.2. Dose–response relationship

A stepwise increase in both DWI positivity and ICU admission rates was observed across the four glucose categories (Table 2, Figure 1). Among patients with glucose < 110 mg/dL, the DWI positivity rate was 41.6 %, compared with 47.7 % for 110–139 mg/dL, 59.2 % for 140–179 mg/dL, and 75.0 % for ≥ 180 mg/dL (chi-square 19.06, $p < 0.001$). A similar gradient was observed for ICU admission: 18.0 %, 28.4 %, 35.2 %, and 40.6 %, respectively (chi-square 10.66, $p = 0.014$). The trend was statistically significant for both DWI positivity (Spearman rho 0.239, $p < 0.001$) and ICU admission (rho 0.184, $p = 0.001$). Early ischemic changes on CT also increased with glucose category, from 19.1 % in the lowest to 43.8 % in the highest group ($p < 0.001$).

3.3. Hyperglycemia as a binary predictor

Using a cutoff of ≥ 140 mg/dL, hyperglycemia was present in 135 patients (43.2 %) and was associated with a 2.48-fold increased odds of DWI positivity (95 % CI 1.57–3.92, $p < 0.001$) and a 2.03-fold increased odds of ICU admission (95 % CI 1.23–3.36, $p = 0.006$). At the ≥ 180 mg/dL threshold, the odds ratio for DWI positivity increased to 3.15 (95 % CI 1.70–5.82, $p < 0.001$) with a specificity of 89.2 %, although sensitivity decreased to 27.6 %.

3.4. Multivariate analysis

In univariate logistic regression, glucose (OR per 10 mg/dL 1.07, 95 % CI 1.03–1.12, $p < 0.001$), age (OR 1.05, 95 % CI 1.03–1.07, $p < 0.001$), and SBP (OR per 10 mmHg 1.52, 95 % CI 1.36–1.69, $p < 0.001$) were each independently associated with DWI positivity. In the multivariate model adjusted for glucose, age, SBP, NLR, creatinine, and sex, glucose remained a significant independent predictor of DWI positivity (adjusted OR per 10 mg/dL 1.05, 95 % CI 1.01–1.10, $p = 0.016$). Age (adjusted OR 1.05, 95 % CI 1.03–1.07, $p < 0.001$) and SBP (adjusted OR per 10 mmHg 1.50, 95 % CI 1.34–1.68, $p < 0.001$) retained the

strongest associations. NLR, creatinine, and sex were not significant in the adjusted model (Table 3).

For the secondary outcome of ICU admission, the multivariate model demonstrated independent associations for age (adjusted OR 1.04, $p < 0.001$), SBP (adjusted OR per 10 mmHg 1.16, $p < 0.001$), and creatinine (adjusted OR 3.59, 95 % CI 1.27–10.14, $p = 0.016$). Glucose showed a trend toward significance (adjusted OR per 10 mg/dL 1.03, $p = 0.068$).

3.5. ROC analysis and predictive accuracy

ROC analysis revealed a clear hierarchy of predictive accuracy for DWI positivity (Table 4, Figure 2). Glucose alone had an AUC of 0.647 (95 % CI 0.588–0.704), with an optimal cutoff at 151 mg/dL (sensitivity 46.7 %, specificity 78.3 %). The exploratory GAS composite score achieved a substantially higher AUC of 0.809 (95 % CI 0.761–0.855), with an optimal cutoff at 1.09 (sensitivity 78.1 %, specificity 67.8 %). The full multivariate prediction model incorporating glucose, age, SBP, NLR, creatinine, and sex yielded the highest AUC of 0.851 (95 % CI 0.807–0.891). DeLong tests confirmed that the GAS composite score and the multivariate model each significantly outperformed glucose alone ($p < 0.001$ for both comparisons), while the multivariate model and the GAS composite did not differ significantly from each other ($p = 0.09$). For ICU admission, a similar pattern was observed: glucose AUC 0.613, GAS composite AUC 0.705, and multivariate model AUC 0.751.

4. Discussion

In this retrospective study of 322 ED patients with suspected stroke, admission hyperglycemia was independently associated with DWI-confirmed ischemic stroke. A clear dose–response gradient was observed: DWI positivity rose from 41.6 % in the lowest glucose category to 75.0 % in the highest — a nearly 1.8-fold increase. Importantly, combining glucose with age and SBP — three parameters routinely obtained within minutes of ED arrival — substantially improved the discriminative ability for DWI-positive stroke (AUC 0.809 for the GAS composite versus 0.647 for glucose alone), and the full multivariate model reached an excellent AUC of 0.851.

4.1. Hyperglycemia and confirmed ischemic injury

The association between admission hyperglycemia and stroke severity is well established. Capes and colleagues demonstrated a threefold increase in mortality among non-diabetic stroke patients with hyperglycemia [7], and a recent dose–response meta-analysis confirmed a near-linear relationship between glucose levels and adverse outcomes [10]. Our findings extend this evidence by using DWI as an objective marker of confirmed ischemic injury rather than relying on clinical severity scales. Only a limited number of studies have

examined glucose in the context of DWI findings: Gur-Ozmen et al. reported higher glucose levels in Turkish stroke patients with DWI–perfusion mismatch [12], and Ribo et al. showed that hyperglycemia above 140 mg/dL accelerated DWI lesion growth in patients receiving thrombolysis [11]. Our study differs from these by treating DWI positivity itself as the outcome, effectively asking whether hyperglycemia at ED presentation predicts the presence of confirmed ischemic injury among patients with suspected stroke.

The 140 mg/dL threshold, which is the most widely used cutoff in the stroke–hyperglycemia literature [11,13], yielded an OR of 2.48 for DWI positivity in our cohort. This aligns with Nardi and colleagues, who reported an optimal glucose cutoff near 143 mg/dL for predicting 72-hour fatality after acute cerebral ischemia [14]. At 180 mg/dL, the OR rose to 3.15, suggesting a stronger association at higher thresholds — although at the cost of reduced sensitivity. Recent work has also highlighted the prognostic value of the stress hyperglycemia ratio, which adjusts admission glucose for chronic glycemic status using HbA1c [15]; while we could not compute this index because HbA1c was not routinely available, its emerging role supports the broader concept that admission glucose carries independent prognostic information across diabetic and non-diabetic patients.

4.2. The dose–response relationship

The stepwise increase across four glucose categories is consistent with the dose–response pattern reported in the recent meta-analysis by Huang and colleagues [10] and supports the concept that admission glucose lies on a continuous risk spectrum rather than functioning as a binary variable. This gradient persisted for both DWI positivity and ICU admission, reinforcing the clinical relevance of glucose-based risk stratification. Notably, even mild hyperglycemia (110–139 mg/dL) was associated with a higher DWI positivity rate (47.7 %) than the lowest category (41.6 %), suggesting that risk may begin to increase below the conventional 140 mg/dL threshold.

4.3. Composite score and multivariate model

The exploratory GAS composite score, calculated as Glucose × Age × SBP / 10^6, achieved an AUC of 0.809 — substantially higher than glucose alone (0.647). This improvement is clinically meaningful because it elevates predictive performance from “poor” to “good” by combining three synergistic risk factors. The product-based formulation was chosen on the rationale that these variables may interact multiplicatively: hyperglycemia in an elderly, hypertensive patient plausibly confers greater risk than the same glucose level in a young normotensive individual. The full multivariate model reached an AUC of 0.851, indicating that the simple three-variable composite captures most of the predictive information available from routine ED data.

We deliberately present the GAS score as an exploratory finding rather than a validated clinical tool. External validation in an independent cohort, ideally a prospective multicenter dataset, is required before any clinical recommendation can be made. Nevertheless, the practical advantage is notable: these three measurements are universally available within the first minutes of ED evaluation, require no additional cost or testing, and can be calculated mentally or with a basic calculator at the bedside.

4.4. Clinical implications

From an ED perspective, our findings suggest that admission glucose should be interpreted in clinical context rather than in isolation. A glucose level of 150 mg/dL carries different implications in a 75-year-old hypertensive patient than in a 40-year-old normotensive one. The multivariate approach — whether formalized as a composite score or simply as clinical awareness of the interplay between glucose, age, and blood pressure — may assist emergency physicians in prioritizing patients with suspected stroke for advanced imaging, especially in resource-limited settings where MRI access is constrained.

4.5. Limitations

Several limitations warrant consideration. First, this was a retrospective, single-center study, which limits generalizability to other populations and practice settings. Second, glycated hemoglobin (HbA1c) data were not available, preventing differentiation between stress hyperglycemia and pre-existing diabetes — a distinction that may influence prognostic interpretation [15]. Third, the National Institutes of Health Stroke Scale (NIHSS) was not consistently recorded in our dataset, precluding direct comparison with clinical severity. Fourth, mortality data were not available; ICU admission served as a surrogate for disease severity. Fifth, the GAS composite score was derived from and tested in the same cohort without external validation or bootstrap internal validation, and the choice of a multiplicative rather than an additive or weighted formulation was exploratory; the high multivariate AUC of 0.851 should therefore be interpreted with caution given the risk of overfitting, although the events-per-variable ratio of 29 exceeded the recommended minimum of 10. Finally, as this was a Turkish single-center cohort, results may not apply directly to populations with different prevalences of diabetes, hypertension, or stroke etiology.

5. Conclusion

Admission blood glucose was independently associated with DWI-confirmed ischemic stroke in ED patients, with a clear dose–response relationship across four predefined glucose categories. Combining glucose with age and SBP — three universally available ED parameters — improved predictive accuracy from poor (AUC 0.647) to excellent (AUC 0.851) in the multivariate model. These findings support the integration of glucose into early

ED stroke risk assessment. This readily available bedside approach may support early neuroimaging prioritization in resource-limited settings and warrants prospective, multicenter validation.

Declarations

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Competing interests: The author declares no competing interests.

Data availability: De-identified data supporting the findings of this study are available from the corresponding author on reasonable request.

Author contributions (CRediT): Zülküf Sergen Ünlü: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft, Writing – review and editing.

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Table 1. Baseline characteristics of the study population stratified by DWI status

Variable	All (n = 322)	DWI(+) (n = 174)	DWI(−) (n = 148)	p value
Demographics				
Age, years — median (IQR)	64 (49–74)	70 (58–77)	53 (44–67)	< 0.001
Male sex — n (%)	173 (53.7)	91 (52.3)	82 (55.4)	0.656
Vital signs at presentation				
SBP, mmHg — median (IQR)	145 (125–172)	162 (140–182)	131 (118–145)	< 0.001
DBP, mmHg — median (IQR)	80 (70–93)	82 (73–100)	76 (67–85)	< 0.001
Heart rate, bpm — median (IQR)	84 (72–98)	83 (71–97)	85 (73–98)	0.518
SpO_2, % — median (IQR)	96 (94–98)	96 (93–98)	97 (95–98)	0.018
Laboratory values				
Admission glucose, mg/dL	130 (107–167)	141 (112–189)	119 (101–146)	< 0.001
NLR — median (IQR)	3.09 (2.01–5.72)	3.40 (2.22–5.98)	2.80 (1.75–5.27)	0.043
MLR — median (IQR)	0.26 (0.18–0.38)	0.26 (0.19–0.36)	0.26 (0.18–0.39)	0.767
INR — median (IQR)	1.03 (0.96–1.10)	1.04 (0.96–1.11)	1.02 (0.96–1.09)	0.507
Creatinine, mg/dL	0.84 (0.71–1.00)	0.89 (0.72–1.04)	0.82 (0.69–0.96)	0.025

Variable	All (n = 322)	DWI(+) (n = 174)	DWI(−) (n = 148)	p value
Time intervals				
Symptom-to-door, min	214 (120–600)	180 (120–300)	300 (120–1440)	< 0.001
Door-to-CT, min	70 (46–107)	64 (44–98)	76 (50–113)	0.025
Door-to-MRI, min	90 (60–168)	80 (52–115)	118 (70–213)	< 0.001
Imaging findings — n (%)				
Early ischemic changes on CT	77 (24.1)	76 (43.9)	1 (0.7)	< 0.001
Hyperdense MCA sign	4 (1.3)	4 (2.3)	0 (0.0)	0.179
CTA vessel occlusion	67 (22.0)	65 (40.4)	2 (1.4)	< 0.001
Treatment & disposition — n (%)				
IV tPA administration	49 (15.2)	48 (27.6)	1 (0.7)	< 0.001
Mechanical thrombectomy	14 (4.3)	14 (8.0)	0 (0.0)	0.001
ICU admission	94 (29.2)	86 (49.4)	8 (5.4)	< 0.001

Continuous variables presented as median (interquartile range, IQR); categorical variables as n (%). Comparisons used the Mann–Whitney U test for continuous and chi-square or Fisher exact test for categorical variables. CTA = computed tomography angiography; DBP = diastolic blood pressure; DWI = diffusion-weighted imaging; ICU = intensive care unit; INR = international normalized ratio; IV tPA = intravenous tissue plasminogen activator; MCA = middle cerebral artery; MLR = monocyte-to-lymphocyte ratio; NLR = neutrophil-to-lymphocyte ratio; SBP = systolic blood pressure.

Table 2. Dose–response relationship between admission glucose categories and clinical outcomes

Glucose category (mg/dL)	n	DWI(+) — n (%)	ICU admission — n (%)	Early ischemic change on CT — n (%)
< 110	89	37 (41.6)	16 (18.0)	17 (19.1)
110–139	88	42 (47.7)	25 (28.4)	19 (21.6)
140–179	71	42 (59.2)	25 (35.2)	11 (15.5)
≥ 180	64	48 (75.0)	26 (40.6)	28 (43.8)
p (chi-square)		**< 0.001**	**0.014**	**< 0.001**
p (trend)		**< 0.001**	**0.001**	—

Linear trend across categories tested using Spearman rank correlation; categorical comparisons used the chi-square test. CT = computed tomography; DWI = diffusion-weighted imaging; ICU = intensive care unit.

Table 3. Univariate and multivariate logistic regression for DWI positivity

Variable	Univariate OR (95% CI)	p	Multivariate aOR (95% CI)	p
Glucose (per 10 mg/dL)	**1.07 (1.03–1.12)**	**< 0.001**	**1.05 (1.01–1.10)**	**0.016**
Age (per year)	1.05 (1.03–1.07)	< 0.001	1.05 (1.03–1.07)	< 0.001
SBP (per 10 mmHg)	1.52 (1.36–1.69)	< 0.001	1.50 (1.34–1.68)	< 0.001
NLR	1.00 (0.999–1.001)	0.735	1.00 (0.999–1.001)	0.936
Creatinine (per mg/dL)	1.88 (0.88–4.03)	0.105	1.16 (0.64–2.11)	0.624

Variable	Univariate OR (95% CI)	p	Multivariate aOR (95% CI)	p
Male sex (vs female)	0.88 (0.57–1.37)	0.578	1.43 (0.80–2.56)	0.225

Multivariate model: full AUC 0.851; Nagelkerke pseudo R^2 = 0.310; events-per-variable = 29. Continuous predictors entered as linear terms. aOR = adjusted odds ratio; CI = confidence interval; DWI = diffusion-weighted imaging; NLR = neutrophil-to-lymphocyte ratio; OR = odds ratio; SBP = systolic blood pressure.

Table 4. Discriminative accuracy of predictors for DWI positivity and ICU admission

Predictor	Outcome	AUC (95% CI)	Optimal cutoff	Sensitivity	Specificity
Glucose alone	DWI(+)	0.647 (0.588–0.704)	151 mg/dL	46.7 %	78.3 %
GAS composite score	**DWI(+)**	**0.809 (0.761–0.855)**	**1.09**	**78.1 %**	**67.8 %**
Multivariate model	**DWI(+)**	**0.851 (0.807–0.891)**	—	—	—
Glucose alone	ICU	0.613 (0.548–0.678)	151 mg/dL	50.0 %	70.9 %
GAS composite score	ICU	0.705 (0.646–0.764)	1.01	88.0 %	46.8 %
Multivariate model	ICU	0.751 (0.696–0.806)	—	—	—

AUC values were computed for each predictor; optimal cutoffs were identified using the Youden J statistic. The multivariate model included glucose, age, SBP, NLR, creatinine, and sex. AUC = area under the curve; CI = confidence interval; DWI = diffusion-weighted imaging; GAS = Glucose–Age–SBP; ICU = intensive care unit.

Figure legends

Figure 1. Dose–response relationship between admission glucose categories and rates of DWI positivity (filled bars) and ICU admission (open bars). A significant stepwise increase was observed across the four predefined glucose categories for both outcomes (p for trend < 0.001 and p = 0.001, respectively).

Figure 2. Receiver operating characteristic (ROC) curves comparing glucose alone, the GAS composite score, and the full multivariate model for predicting DWI-confirmed ischemic stroke. AUC values: glucose alone 0.647, GAS composite 0.809, multivariate model 0.851.

Figure 1

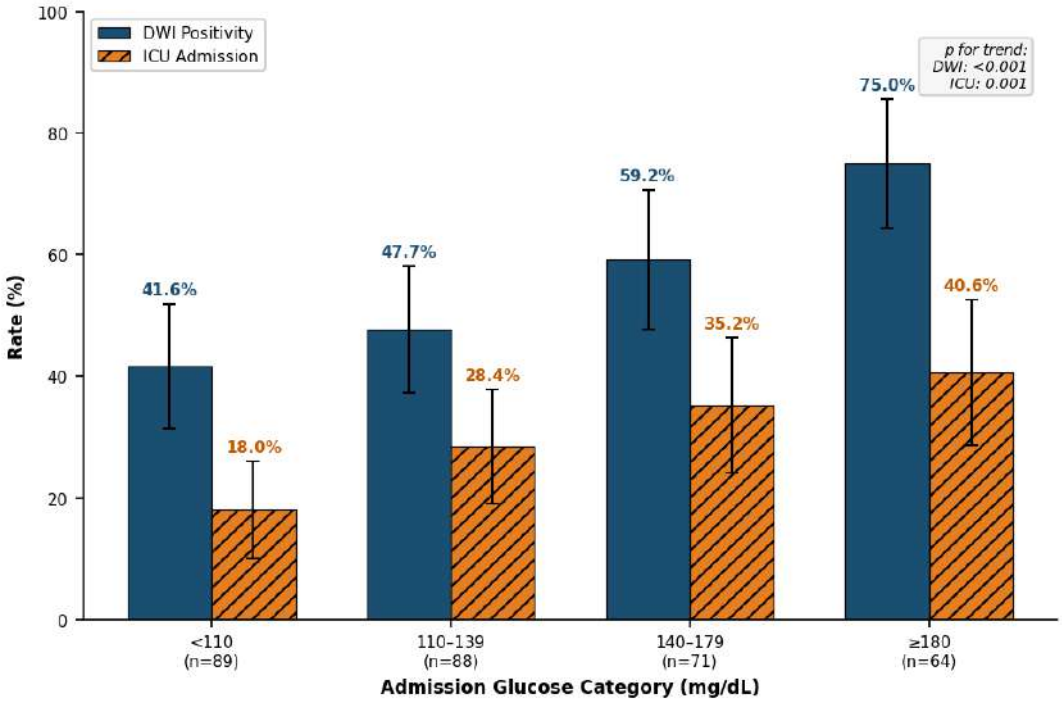

Figure 1. Dose–response relationship between admission glucose categories and rates of DWI positivity and ICU admission.

Figure 2

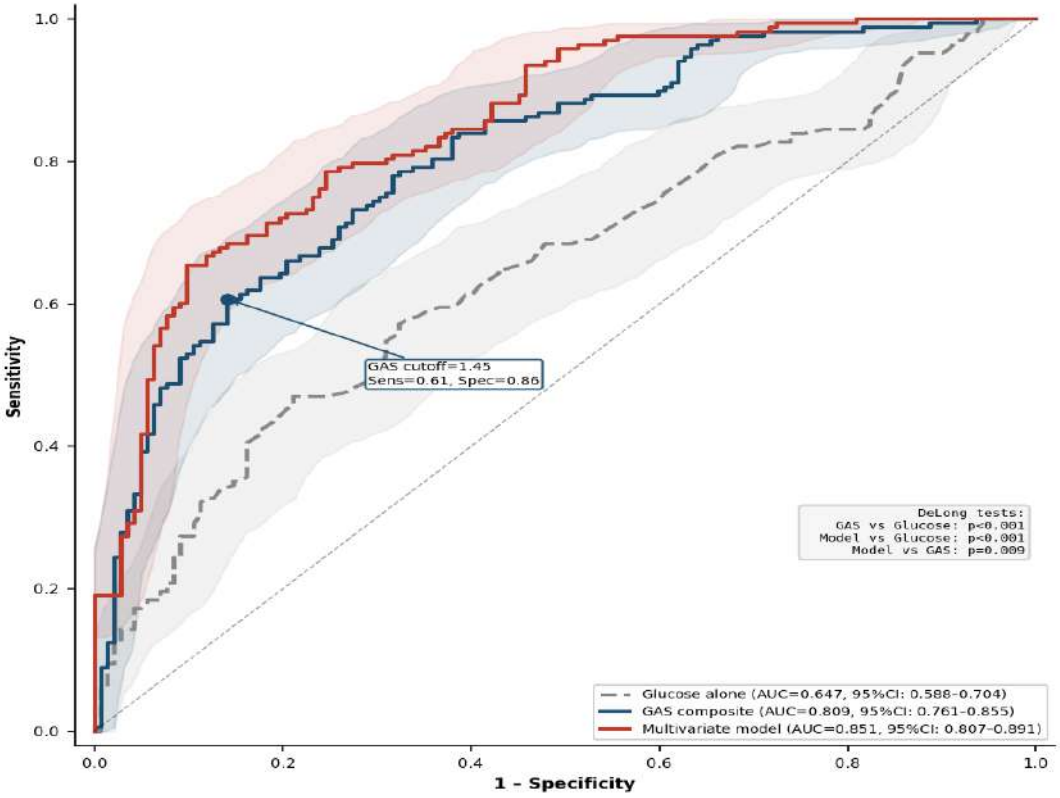

Figure 2. ROC curves comparing glucose alone, GAS composite score, and the multivariate model for predicting DWI-confirmed ischemic stroke.

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Early Risk Stratification in Traumatic Brain Injury: An Analysis of MEWS and rSIG Scores in Patients with Isolated Head Trauma

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Introduction

Traumatic brain injury (TBI) is a leading cause of trauma-related morbidity and mortality worldwide (1). Early identification of patients at risk for deterioration is critical for appropriate emergency management. The Modified Early Warning Score (MEWS) and the reverse Shock Index multiplied by the Glasgow Coma Scale (rSIG) are simple bedside tools that may assist in early prognostic a ssessment (2,3). This study aimed to evaluate the ability of MEWS and rSIG to predict poor outcomes in patients with isolated head trauma.

Methods

This retrospective observational study included patients presenting with isolated head trauma to the emergency department of Manisa Celal Bayar University Hospital between June 2021 and June 2024. Demographic and clinical data were obtained from the hospital information system. MEWS, Shock Index (SI), reverse Shock Index (rSI), and rSIG were calculated for each patient. Poor outcome was defined as intensive care unit admission and/or in-hospital mortality. Group comparisons were performed using nonparametric tests. Spearman correlation analysis, receiver operating characteristic (ROC) curves, and binary logistic regression models were used for statistical evaluation.

Results

A total of 705 patients (65.7% male; mean age 37.2±27.0 years) were analyzed. Pathological cranial CT findings were present in 24.7%, and overall mortality was 2.7%. Patients with poor outcomes had significantly higher MEWS and SI values and significantly lower GCS, rSI, and rSIG scores (all $p<0.001$). ROC analysis demonstrated moderate predictive ability for rSIG (AUC=0.701) and limited discrimination for MEWS (AUC=0.610). Optimal cut-off values were ≤21.35 for rSIG and ≥0.5 for MEWS. In multivariate analysis, both MEWS and rSIG showed independent prognostic significance in separate models, with rSIG remaining

significant in models excluding GCS. MEWS positively correlated with hospital length of stay (r=0.385, p<0.001), whereas rSIG showed a negative correlation (r=–0.252, p<0.001).

Discussion

Our study demonstrates that patients with poor outcomes after isolated head trauma exhibit higher MEWS and SI values and lower GCS, rSI, and rSIG scores. Both MEWS and rSIG are practical bedside tools for early risk stratification, with MEWS reflecting physiological deterioration and rSIG providing complementary hemodynamic–neurological information (4,5). These scores should be interpreted as adjuncts to standard clinical assessment rather than standalone triage instruments. Routine use may facilitate early clinical decision-making and closer patient monitoring in the emergency setting.

Keywords: Head Trauma, Modified Early Warning Score (MEWS), Reverse Shock Index multiplied by GCS (rSIG), Shock Index, Traumatic Brain Injury

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4789

A Rare Cause of Right Upper and Lower Quadrant Pain: A Case with Suspected Bilioenteric Fistula

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Keywords: Bilioenteric fistula, microperforation, adenomyomatosis

Introduction

Bilioenteric fistulas are abnormal transitions between the biliary tract and intestine and are rarely described clinically. The most common causes in the literature are chronic gallstone disease, recurrent cholecystitis attacks and biliary tract anomalies (1,2). These fistulas usually present with non-specific symptoms; abdominal pain, nausea, anorexia and sometimes fever may be observed. The diagnosis is often made with advanced imaging modalities and confirmed after surgery. Computed tomography (CT) and ultrasonography play a critical role in the diagnosis of bilioenteric fistulas. In addition, gallbladder wall changes such as adenomyomatosis may rarely contribute to fistula formation (3). This case reports a patient who presented with pain in the right upper and lower quadrant and suspected bilioenteric fistula with advanced imaging.

Case Report

A 37-year-old man with no known chronic disease presented with abdominal pain that had been intermittent for about a month and intensified last night. He had constipation and nausea, no vomiting and anorexia. Physical examination revealed tenderness in the right upper and lower quadrants, no defense and rebound, other quadrants were normal. Vital signs were stable. Gloskow Coma Scale was 15, the patient was coopere and oriented.

Laboratory findings were as follows: Leukocyte count (White Blood Cell, WBC) was $10.96\times10^3/\mu L$ with neutrophil predominance. C-reactive protein (C-Reactive Protein, CRP)

value was 10 mg/L. In liver function tests, alanine aminotransferase (ALT) was 25 U/L, aspartate aminotransferase (AST) was 21 U/L and total bilirubin (TBIL) was 0.22 mg/dL. Lactate dehydrogenase (LDH) level was 271 U/L. Hepatitis B surface antigen (HBsAg) was 5720.56 IU/mL. Lactate was 3.0 mmol/L and pH 7.34 in blood gas analysis. Other biochemistry and hemogram parameters were within normal limits.

Contrast-enhanced abdominal computed tomography showed that the colonic anus extending towards the gallbladder lumen was markedly distended and its transverse diameter reached approximately 10 cm. In this segment, increased intestinal wall thickness and adjacent free air were noted; the findings were considered to be compatible with microperforation. Hepatobiliary ultrasonography showed a transverse diameter of the gallbladder of 40 mm and cystic foci consistent with adenomyomatosis at the fundus level.

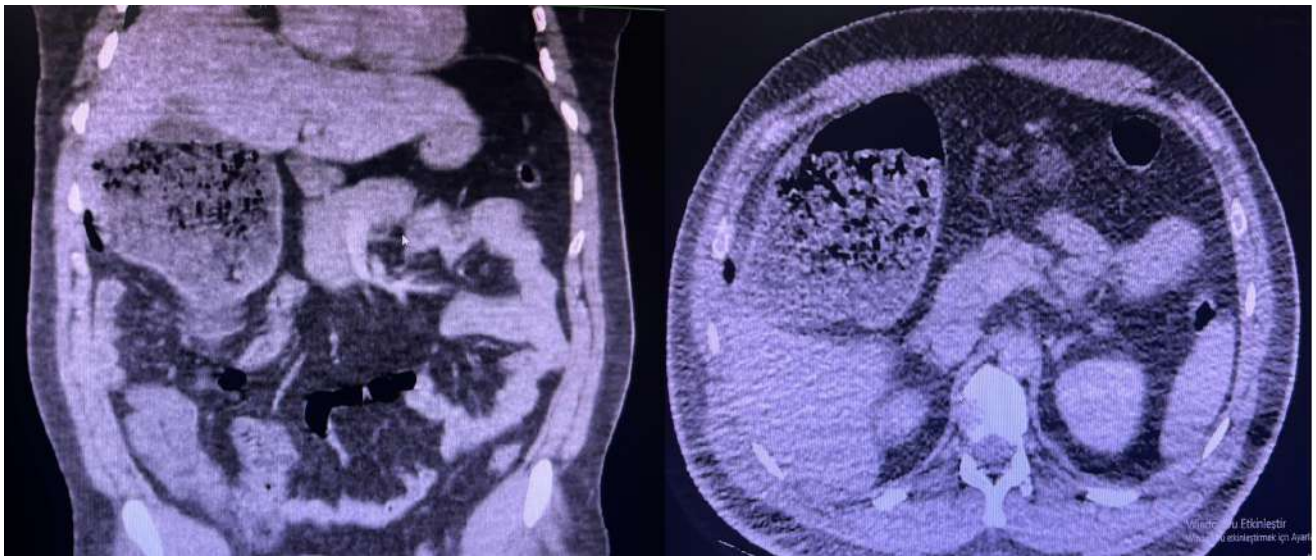

Figure 1. Distinct colonic anus dilatation and microperforation seen on contrast-enhanced abdominal CT sagittal and axial sections.

These findings were suggestive of bilioenteric fistula. The patient was consulted to the general surgery department and was interned to the ward and further evaluation was planned.

Discussion

Bilioenteric fistulas are abnormal connections between the biliary tract and the gastrointestinal system and are among the rare pathologies encountered in hepatobiliary surgery. In the literature, the etiology of these fistulas is mostly associated with chronic gallstone disease, recurrent cholecystitis attacks and biliary tract anomalies (1,2). However, gallbladder wall changes such as adenomyomatosis may rarely contribute to fistula development; cystic changes and wall thickening may predispose to fistula formation through chronic inflammation and mechanical irritation (3).

Clinically, bilioenteric fistulas usually present with non-specific symptoms. Abdominal pain, nausea, anorexia and sometimes low-grade fever are the most common findings. Therefore, the diagnosis is often delayed and routine physical examination and laboratory tests alone are not sufficient. In this case, the patient presented with pain in the right upper and lower quadrants, nausea and constipation. On physical examination, no defense or rebound was detected and the other quadrants were normal. These findings were in accordance with the "non-specific and atypical" symptom profile reported in the literature (4).

Imaging plays a critical role in the diagnosis of bilioenteric fistulas. Distention of the colonic anus, increase in wall thickness and presence of free air on contrast-enhanced CT are warning signs for the possibility of fistula. Increased transverse diameter of the gallbladder on ultrasonography and cystic foci compatible with adenomyomatosis at the fundus level are considered as additional findings supporting the diagnosis in the literature (5,6). In this case, both CT and USG findings confirmed the classical diagnostic criteria and provided additional information regarding fistula localization and possible microperforation.

Conclusion

Bilioenteric fistulas should be considered in the differential diagnosis of patients presenting with right upper and lower quadrant pain, especially in the presence of chronic biliary tract changes and adenomyomatosis. Early diagnosis, advanced imaging methods and multidisciplinary approach are critical to prevent complications and plan

appropriate surgical management. This case illustrates the importance of imaging findings and clinical evaluation strategy in the diagnostic and management of rare bilioenteric fistulas.

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Fatal Crush Syndrome Secondary to Carbon Monoxide Exposure and Prolonged Immobilization: A Case Report

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Introduction

Crush syndrome is a serious clinical condition characterized by extensive skeletal muscle necrosis resulting from prolonged compression or immobilization, with potentially fatal systemic complications (1,2). Destruction of muscle cells leads to the release of intracellular components such as creatine kinase (CK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), potassium, and myoglobin into the circulation. This process may result in rhabdomyolysis, electrolyte disturbances, metabolic acidosis, and acute kidney injury (AKI) (3,4).

Rhabdomyolysis is the primary pathophysiological component of crush syndrome. Clinically, it may present with muscle pain, weakness, and dark-colored urine; however, these symptoms may be subtle or absent in some cases (3,5). Therefore, the diagnosis of rhabdomyolysis is often based on laboratory findings, particularly markedly elevated CK levels accompanied by increased AST and LDH levels (3). AKI in rhabdomyolysis occurs through multiple

mechanisms, including hypovolemia, direct tubular toxicity of myoglobin, intratubular obstruction, and renal vasoconstriction (4,5).

Crush syndrome has traditionally been associated with traumatic events such as earthquakes, traffic accidents, and industrial disasters. However, non-traumatic causes have increasingly been reported in recent years (1,6). Prolonged immobilization due to alcohol or drug intoxication is among the most important non-traumatic etiologies (6,7). In particular, cases described as “Saturday night palsy” involve prolonged compression of muscle tissue after alcohol intoxication, potentially resulting in rhabdomyolysis and compartment syndrome (7).

Carbon monoxide (CO) poisoning causes tissue hypoxia due to its high affinity for hemoglobin, thereby impairing oxygen delivery to tissues (8,9). Additionally, CO disrupts mitochondrial oxidative phosphorylation, leading to cellular energy failure. Loss of consciousness caused by CO exposure may prolong immobilization and increase the risk of rhabdomyolysis and crush syndrome. Cases of crush syndrome associated with CO poisoning are rare but represent a potentially fatal clinical combination (8).

Acute compartment syndrome is a severe complication of crush syndrome and rhabdomyolysis. Increased intracompartmental pressure leads to impaired microcirculation and irreversible damage to muscle and nerve tissue (6–10). Early diagnosis and timely fasciotomy are critical for preventing limb loss and reducing mortality (5,11). However, delayed diagnosis and bilateral extremity involvement may worsen the prognosis despite surgical intervention (6).

In this report, we present a fatal case of crush syndrome accompanied by rhabdomyolysis, bilateral compartment syndrome, and acute kidney injury that developed following carbon monoxide exposure and prolonged immobilization after alcohol consumption in a closed environment.

Case Presentation

A 32-year-old male with no known chronic illnesses or regular medication use was found unconscious approximately 10 hours after entering a closed garage to consume alcohol. He was discovered in a semi-kneeling position leaning against a running vehicle. Upon arrival at the emergency department, the patient was somnolent with a Glasgow Coma Scale score of 14. Vital signs were as follows: blood pressure 100/60 mmHg, heart rate 75 beats/min, respiratory rate 18 breaths/min, peripheral oxygen saturation 95%, and blood glucose level 80 mg/dL.

The patient reported that he had begun drinking alcohol while the vehicle engine was running but did not remember subsequent events. Physical examination revealed erythema over both knees. The right great toe was pale and cold. Dorsalis pedis and posterior tibial pulses were absent in the right foot. The right leg was tense on palpation. Following Foley catheter insertion, the urine was observed to be dark and cola-colored.

Laboratory evaluation revealed creatinine 2.89 mg/dL, AST 2200 U/L, ALT 335 U/L, LDH 5600 U/L, potassium 7.8 mmol/L, high-sensitivity troponin T 489 ng/L, and CK 48,000 U/L. Arterial blood gas analysis demonstrated pH 7.05, lactate 16 mmol/L, bicarbonate 11.6 mmol/L, and carboxyhemoglobin level 19%.

Doppler ultrasonography showed patent lumens in the right popliteal artery but absence of a triphasic flow pattern. Acute thrombosis was detected in the right popliteal vein and its continuation. No flow was detected in the anterior tibial and dorsalis pedis arteries at the ankle level, while minimal low-velocity flow was observed in the posterior tibial artery.

Consultations were obtained from orthopedic surgery, cardiovascular surgery, and nephrology. Based on clinical and radiological findings, increased pressure in the right tibial compartment with associated circulatory compromise was suspected. The patient was diagnosed with crush syndrome, rhabdomyolysis, acute kidney injury, and compartment syndrome and was admitted to the intensive care unit. Emergency hemodialysis was initiated.

During follow-up, similar findings developed in the left lower extremity, and the patient underwent emergency surgery. Multiple fasciotomies were performed in bilateral femoral and tibial compartments under general anesthesia. Postoperatively, distal circulation could not be restored in the right lower extremity. Blood transfusion was administered due to decreased hemoglobin levels, and treatment for refractory hyperkalemia continued. Approximately eight hours after surgery, the patient developed cardiac arrest. Despite advanced cardiopulmonary resuscitation, return of spontaneous circulation could not be achieved and the patient was pronounced dead

Discussion

The presented case represents a rare clinical scenario in which multiple high-risk pathophysiological mechanisms contributed simultaneously to a fatal outcome. Alcohol-induced loss of consciousness, carbon monoxide exposure in a closed environment, and prolonged immobilization created conditions favorable for the development of severe rhabdomyolysis and crush syndrome.

Alcohol acts as a central nervous system depressant, preventing positional changes and resulting in prolonged pressure on muscle tissues. Previous reports have described alcohol-related immobilization leading to rhabdomyolysis and compartment syndrome, particularly affecting the lower extremities (6,7). In our case, the patient was found approximately 10 hours after the initial event, which likely contributed to the progression of muscle necrosis and systemic complications.

Carbon monoxide exposure was another important factor in this case. The COHb level of 19% indicated clinically significant exposure. CO causes tissue hypoxia and disrupts mitochondrial respiration, resulting in impaired cellular energy metabolism (8,9). When combined with mechanical compression due to immobilization, these mechanisms may exacerbate muscle injury and accelerate the development of rhabdomyolysis.

Severe hyperkalemia and metabolic acidosis secondary to rhabdomyolysis were the main contributors to the fatal outcome in this patient. Rapid release of intracellular potassium into the circulation can cause life-threatening cardiac arrhythmias, particularly in the presence of renal dysfunction. The potassium level of 7.8 mmol/L and severe metabolic acidosis observed in this case are consistent with findings reported in fatal rhabdomyolysis cases in the literature (12).
Acute kidney injury is one of the most serious complications of rhabdomyolysis. Myoglobin-induced tubular toxicity, intratubular obstruction, hypovolemia, and renal vasoconstriction all contribute to renal injury. Although early renal replacement therapy was initiated in our patient, the clinical condition could not be reversed due to extensive muscle necrosis and persistent metabolic derangements.

Another striking aspect of this case was bilateral lower extremity compartment syndrome. Early diagnosis and fasciotomy are essential to prevent irreversible tissue damage. However, bilateral involvement, venous thrombosis, and delayed presentation may limit the success of surgical intervention. In this case, despite bilateral multi-compartment fasciotomy, distal circulation could not be restored in the right lower extremity, suggesting severe microvascular injury.

Conclusion

This case demonstrates that alcohol-induced loss of consciousness combined with carbon monoxide exposure in a closed environment may lead to prolonged immobilization and severe crush syndrome. Emergency physicians should maintain a high index of suspicion for rhabdomyolysis, crush syndrome, and compartment syndrome in patients found unconscious in enclosed spaces. Early diagnosis and aggressive multidisciplinary management are essential, although mortality may remain high in advanced cases.

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Keywords: Crush syndrome, Rhabdomyolysis, Carbon monoxide poisoning, Compartment syndrome

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The Macklin Effect in the Elderly: Spontaneous Pneumomediastinum Triggered by Parenchymal Infiltration

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Introduction

Spontaneous pneumomediastinum (SPM) is a rare clinical entity characterized by the presence of free air within the mediastinal space and is generally considered a benign condition. SPM, also known as Hamman syndrome, results from alveolar rupture followed by the dissection of air along the peribronchial and perivascular sheaths into the mediastinum, a mechanism known as the Macklin effect. Although it typically presents in young adults with acute chest pain and dyspnea, elderly patients may present atypically with malnutrition, general deterioration, or underlying parenchymal lung diseases such as pulmonary infiltration or chronic obstructive pulmonary disease (COPD). We report the diagnostic evaluation and management of a 77-year-old man who presented with impaired oral intake and was found to have pneumomediastinum associated with pneumothorax and pulmonary infiltration.

Case Presentation

A 77-year-old male presented to the emergency department with a 10-day history of loss of appetite, difficulty eating and drinking, and generalized weakness. His medical history revealed risk factors for chronic parenchymal lung disease. Physical examination demonstrated decreased breath sounds bilaterally. No evidence of subcutaneous emphysema was noted. Vital signs were stable on admission. Laboratory investigations revealed a mild systemic inflammatory response, with a C-reactive protein (CRP) level of 48 mg/L. Arterial blood gas analysis and other laboratory parameters were within normal limits. Thoracic computed tomography (CT) demonstrated free air surrounding the mediastinal structures, consistent with pneumomediastinum. In addition, a right apical pneumothorax and diffuse pulmonary infiltrates suggestive of an infectious or inflammatory process were identified. Following multidisciplinary evaluation, the patient was consulted by the Thoracic Surgery Department and admitted to the intensive care unit for close monitoring and management.

Discussion

One of the most important considerations in patients with pneumomediastinum is the exclusion of esophageal rupture (Boerhaave syndrome). In the present case, the patient's prolonged history of impaired oral intake and only mildly elevated inflammatory markers favored alveolar rupture secondary to parenchymal lung pathology rather than an acute esophageal perforation. Furthermore, normal arterial blood gas values suggested that the accompanying pneumothorax was small and had not yet significantly impaired ventilation-perfusion balance. The pulmonary infiltrates observed on CT may have increased intra-alveolar pressure and predisposed the patient to alveolar rupture, resulting in air leakage into the mediastinum through the Macklin mechanism.

Conclusion

In elderly patients, spontaneous pneumomediastinum may present with nonspecific symptoms such as general deterioration or poor oral intake rather than the classic symptoms of chest pain and dyspnea. As demonstrated in this case, concomitant pulmonary infiltrates may act as a triggering factor for the Macklin effect. Management is usually conservative and consists of rest, oxygen therapy, and close observation. However, patients should be carefully monitored for progression of associated pneumothorax. Cases requiring intensive care support may necessitate transfer to, or follow-up within, a specialized thoracic surgery or critical care unit.

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5002 **Clinical Distribution and Imaging Trends in Patients Presenting with Dizziness (Vertigo): A Retrospective Study in a Tertiary Care Center**

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Introduction

Dizziness (vertigo) is a common presenting symptom in emergency departments, characterized by the absence of a specific diagnostic algorithm and the need for a multidisciplinary approach. It accounts for approximately 3.5% of all emergency department visits. Differentiation from central causes—most notably posterior circulation strokes—is essential. History taking and physical examination play a pivotal role in the diagnostic process. Contemporary diagnostic approaches include the HINTS examination (Head Impulse Test, nystagmus, test of skew), TiTrATE (timing, triggers, and targeted examination), and the Dix–Hallpike maneuver. The role of computed tomography in diagnosis has diminished and is now primarily used for exclusion purposes. Magnetic resonance imaging has become the preferred diagnostic modality. As most cases are attributable to benign etiologies and can be managed on an outpatient basis, hospitalization rates range between 14.8% and 20%. Nevertheless, high rates of imaging utilization contribute to increased healthcare costs (1–3).

Objective

This study aimed to evaluate the clinical distribution of patients presenting with dizziness, as well as the imaging modalities utilized across different departments, consultation requirements, and hospitalization rates.

Materials and Methods

In this study, the data were analyzed using descriptive statistical methods.

Results

In this retrospective study, one-month admissions of patients presenting with dizziness to a tertiary care hospital were analyzed, and both epidemiological characteristics and clinical management approaches were evaluated. Patients who presented between February 23, 2026, and March 23, 2026, and were assigned the ICD-10 diagnostic code R42 (dizziness/vertigo), were included in the analysis.

A total of 1,211 patients were enrolled in the study. Patients were categorized according to the departments they presented to: emergency department, otorhinolaryngology (ENT) outpatient clinic, neurology outpatient clinic, and other departments. The number of brain computed tomography (CT) and brain magnetic resonance imaging (MRI) studies requested in these departments was analyzed. Additionally, consultation rates and hospitalization rates were evaluated.

Among the 1,211 patients, the majority of presentations were to the emergency department (41.5%, n=503) and the ENT outpatient clinic (41.3%, n=500). Neurology accounted for 10.0% (n=121) and other specialties for 7.2% (n=87) of the presentations.

The overall CT positivity rate was 14.4% (174/1211). When analyzed by department, CT positivity was markedly higher in the emergency department (27.0%, 136/503), whereas lower rates were observed in ENT (3.2%, 16/500), neurology (10.7%, 13/121), and other departments (10.3%, 9/87).

The overall MRI positivity rate was 14.1% (171/1211), with the highest rate observed in the neurology department (28.9%, 35/121). This was followed by other departments (28.7%, 25/87), the emergency department (18.5%, 93/503), and ENT (3.6%, 18/500).

Consultation rates were generally low, with an overall rate of 2.8% (34/1211). The highest consultation rate was observed in ENT (5.0%, 25/500), followed by neurology (6.6%, 8/121) and other departments (1.1%, 1/87), while no consultations were recorded in the emergency department.

The overall hospitalization rate was notably low at 0.7% (8/1211). Hospitalizations were most frequent among neurology patients (3.3%, 4/121), followed by other departments (3.4%, 3/87) and ENT (0.2%, 1/500), while no hospitalizations were observed in the emergency department.

Overall, CT utilization and positivity rates were significantly higher among patients presenting to the emergency department, whereas MRI positivity rates were higher in the neurology department. The low consultation and hospitalization rates suggest that the majority of patients presenting with dizziness can be managed on an outpatient basis.

Limitations of the Study

The main limitations of this study include its single-center design, which may limit generalizability due to local practice variations; the possibility that diagnostic codes may have been reassigned following definitive diagnosis; limited availability of data regarding detailed history and physical examination findings; inability to evaluate repeat presentations; inclusion of the pediatric population; lack of assessment of comorbid conditions; and absence of follow-up data regarding patient prognosis. No conflicts of interest were declared in this study.

Discussion

In the multicenter study by Kerber et al., which evaluated patients presenting to the emergency department with vertigo between 1995 and 2004, both the number of patients presenting with vertigo and the rate of imaging utilization increased significantly ($p<0.01$). The use of CT/MRI increased by 169%, representing a greater rise than all other diagnostic tools. However, the incidence of central events such as malignancy and cerebrovascular disease did not increase. Although our study does not assess temporal trends, our findings are consistent with the existing literature (1).

In the study by Newman et al., dizziness accounted for approximately 3.3–4.4% of emergency department visits. The prevalence of stroke among patients presenting with dizziness was reported to be 3–5%, and more than 15% of patients were found to have serious underlying conditions. It has also been reported that neuroimaging may miss up to 50% of strokes smaller than 1 cm in diameter in patients presenting within the first 24–48 hours. Although CT and MRI are frequently used in current practice, their diagnostic yield remains limited. In particular, bedside ocular examination (HINTS) has been shown to be more sensitive than early MRI in acute vestibular syndrome. In this respect, our findings are consistent with the literature (3).

Edlow et al. emphasized that physical examination is critical in differentiating benign dizziness from posterior fossa lesions. They classified benign vertigo into acute vestibular syndrome, spontaneous episodic vestibular syndrome, and triggered episodic vestibular syndrome, demonstrating that these entities can be distinguished from posterior fossa lesions through clinical examination. Inadequate physical examination has been associated with higher rates of misdiagnosis and increased diagnostic costs. In our study, incomplete clinical documentation, along with high imaging rates but low consultation and hospitalization rates, suggests that insufficient physical examination may contribute to increased healthcare costs, in line with previous reports (4).

In the study by Xing et al., vertigo patients presenting to neurology outpatient clinics were evaluated etiologically. The majority of patients were female, with a mean age of 52.9±16.9 years. Subgroup analysis by age revealed that vestibular migraine (27.4%) was most common in the 0–30 age group, while benign paroxysmal positional vertigo (BPPV) predominated in the 31–60 (27.1%) and 61–100 (46.0%) age groups. Episodic vestibular syndrome accounted for 60.6% of all cases. In terms of localization, 56.1% of patients were diagnosed with peripheral causes, 17.0% with central causes, and 10.0% with psychiatric or functional vestibular disorders. Although such subclassification was not performed in our study, our findings are generally consistent with the literature (5).

Newman-Toker et al. demonstrated that the HINTS examination is superior to the ABCD2 score in predicting central causes. Quimby et al. highlighted that emergency physicians do not utilize the HINTS examination as frequently as neuroimaging. Similarly, our findings underscore the importance of clinical examination (6,7).

In the study by Adams et al., the use of neuroimaging in patients presenting with vertigo was evaluated in detail. The median time to neuroimaging was 0 days in the emergency department and 10 days in outpatient settings. Head CT was the most commonly used modality at presentation across all settings. In our study, 41.5% of patients presented to the emergency department, where CT was the most frequently utilized imaging modality (27%). Additionally, 58.5% of patients presented to outpatient clinics, where the overall imaging rate was 16.4%. These findings are consistent with the literature (8).

The majority of patients presenting with dizziness can be managed on an outpatient basis, with benign etiologies predominating. Although central causes are less frequent, they are

associated with higher morbidity and mortality, necessitating more frequent hospitalization. Higher imaging utilization rates in the emergency department may be attributed to the need for rapid exclusion of life-threatening conditions and the greater accessibility of imaging modalities. Nevertheless, the low consultation and hospitalization rates suggest that improvements in clinical evaluation are warranted.

Conclusion

Adopting a clinical approach based on thorough history taking and physical examination, along with the effective use of bedside vestibular and neurological assessments, may reduce unnecessary imaging rates and facilitate patient management.

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PNEUMOTHORAX: RETROSPECTIVE CASES ANALYSIS

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Abstract

Introduction: Pneumothorax is a clinical condition characterised by the accumulation of air in the pleural space, which can result in lung collapse. Its clinical course can vary widely, from asymptomatic cases to life-threatening respiratory failure. In recent years, biochemical and haematological parameters that reflect the inflammatory response have garnered attention as potential biomarkers for evaluating disease severity and clinical course in patients with pneumothorax.

Aim: This study aimed to evaluate the demographic characteristics and laboratory parameters of patients diagnosed with pneumothorax. It also aimed to investigate the relationships between inflammatory markers and albumin and haemoglobin levels. The goal was to reveal the relationships between these parameters and systemic inflammation and, the disease process.

Method: In this retrospective study, patients diagnosed with pneumothorax were examined. The demographic characteristics and laboratory parameters of the patients were evaluated. In addition, the relationships between inflammatory markers and albumin and haemoglobin levels were analysed.

Findings: The majority of pneumothorax patients included in the study were male, with an average age of 40 years. Right pneumothorax was observed in most cases, while left and bilateral pneumothorax were seen less frequently. In the parameters examined, a significant negative correlation was found between albumin and age, CRP, leukocyte, and monocyte values, while a significant positive correlation was found between albumin and haemoglobin. Furthermore, a significant negative relationship was detected between CRP and haemoglobin.

Conclusion: Routine laboratory parameters can be useful in evaluating the inflammatory process in pneumothorax patients. In particular, the relationships between albumin, hemoglobin, and inflammatory markers can provide important information about the disease process. However, further studies in larger patient groups are needed to more clearly establish the relationship of these parameters with clinical course and prognosis.

Keywords: Pneumothorax, Inflammation, Albumin, Haemoglobin, Haematological parameters

INTRODUCTION

Pneumothorax is a potentially life-threatening clinical condition characterised by partial or complete collapse of the lung due to air accumulation in the pleural space (1).

It can develop due to spontaneous or traumatic causes. Primary spontaneous pneumothorax is usually seen in young and tall men, while secondary pneumothorax is more frequently observed in elderly patients with underlying lung disease (2).

Pneumothorax is one of the most frequent reasons for emergency department visits with sudden chest pain and dyspnea. Respiratory failure may develop in some patients. Therefore, early diagnosis and appropriate treatment are of great importance (1).

Current studies show that not only bleb and bulla rupture, but also distal airway inflammation and structural lung changes play a role in the pathogenesis of primary spontaneous pneumothorax (3).

Routine laboratory parameters are important in clinical evaluation. Haematological indicators such as neutrophils and lymphocytes are associated with inflammation. In addition, biochemical markers such as C-reactive protein and albumin have been reported to be associated with inflammation and prognosis (4).

Combined evaluation of clinical and laboratory findings in pneumothorax patients presenting to the emergency department can contribute to predicting the course of the disease and clinical outcomes. However, studies on this subject are limited (5). This study aimed to determine the importance of demographic data and blood parameters of pneumothorax cases and their relationship with hospital stay and mortality.

MATERIALS AND METHODS

This study retrospectively examined patients diagnosed with pneumothorax who presented to our acute emergency department within the last year. Patients under 18 years of age and those for whom complete data were unavailable were excluded from the study.

STATISTICAL ANALYSIS

Statistical analysis of the data obtained in this study was performed using IBM SPSS 27 Statistics (Version 27; IBM Corp., Armonk, NY, USA) program. The distribution of continuous variables was evaluated using the Shapiro-Wilk test. Qualitative data were expressed as frequency and percentage. For quantitative data, parametric distributions were presented as mean and standard deviation, while parameters with nonparametric distributions were presented as IQR or minimum-maximum values. The t-test was applied to data with parametric distributions, and the Mann-Whitney U test was applied to data with nonparametric distributions. Correlation analysis was performed on quantitative data. $P<0.05$ was considered statistically significant.

RESULTS

A total of 39 pneumothorax patients were included in the study. 7.7% of the patients were female (n=3) and 92.3% were male (n=36). The average age of the patients included in our study was found to be 40. 33.3% (n=13) of the patients were diagnosed with left pneumothorax, 59% (n=23) with right pneumothorax, and 7.7% (n=3) with bilateral pneumothorax. The 2 patients who died were in the male gender group. The mean and SD value of our parametric data was albumin: 42.13±7.5 (Table 1).

Table 1. Mean and SD values of our parametric data.

Variables	n	Mean	SD±
Albumin(g/L)	39	42.13	7.5

The median and IQR values of our nonparametric data are: Patient age was 40 years (25–53), CRP 1 mg/L (1–3), amylase 65 U/L (45–75), leukocytes 10.9×10³/µL (8.8–13.8), platelets 255×10³/µL (217–295), hemoglobin 14.7 (13.2–15.4), monocytes 0.80×10³/µL (0.62–1.16), hospital stay 5 days (4–8), pH 7.37 (7.34–7.41), pCO_2 44 mmHg (37–47), lactate 2 mmol/L (1–2) and neutrophils 7.19×10³/µL (4.51–8.78). (Table 2)

Table 2. Median and IQR values of our non-parametric data.

			IQR	
Variables	n	Median	25%	75%
Age	39	40	25	53
CRP		1	1	3
Amylase		65	45	75
Leukocytes		10.9	8.8	13.8
Platelets		255	217	295
Hbg		14.7	13.2	15.4
Monocytes		0.80	0.62	1.16
HKS		5(days)	4(days)	8(days)
pH		7.37	7.34	7.41
pCO2		44	37	47
Lactate		2	1	2
Neutrophils		7.19	4.51	8.78

We evaluated the correlation between our data using Spearman correlation analysis. We found a moderate negative correlation between age and albumin (r=-0.691; p=0.0001). We also found significant moderate negative correlations between CRP (r=-0.463; p=0.003), monocyte (r=-0.342; p=0.033), and leukocyte (r=-0.346; p=0.031) values and albumin. We found a moderate negative correlation between age and haemoglobin (r=-0.441; p=0.005), a moderately significant negative correlation between CRP and haemoglobin (r=-0.557;

p=0.001), and a moderately significant positive correlation between albumin and haemoglobin (r=0.682; p=0.001) (Table 3).

Table 3. Results of Spearman correlation analysis.

		R	P
Age	Albumin	-0.691	0.0001
CRP	Albumin	-0.463	0.003
Monocytes	Albumin	-0.342	0.033
Leukocytes	Albumin	-0.346	0.031
Age	Hgb	-0.441	0.005
CRP	Hgb	-0.557	0.001
Albumin	Hgb	0.682	0.001

ARGUMENT

Pneumothorax is a potentially fatal clinical condition resulting from partial or complete collapse of the lung due to air accumulation in the pleural space (1).

In our study, the majority of pneumothorax cases were male (92.3%), and the mean age was 40 years. The literature reports that primary spontaneous pneumothorax is more common in young and middle-aged men (1,6). In this respect, the demographic distribution in our study is consistent with the literature and supports the known epidemiological characteristics of pneumothorax.

In our study, right pneumothorax was detected in 59% of cases, left pneumothorax in 33.3%, and bilateral pneumothorax in 7.7%. The literature reports that pneumothorax is slightly more common in the right hemithorax (2). Our findings also show a right-sided predominance and are consistent with existing studies.

When biochemical parameters were examined, it was found that the mean albumin level was 42.13±7.5 g/L and there was a strong negative correlation between age and albumin (r=-0.691; p<0.001). It is known in the literature that albumin is a negative acute-phase reactant and its level decreases with inflammation (7). In our study, the negative relationship between age and albumin can be explained by the inflammatory processes that increase with age and the decreased protein synthesis.

In our study, a significant negative correlation was found between CRP and albumin (r=-0.463; p=0.003). In the literature, it has been shown that CRP increases during inflammation as a positive acute-phase reactant, while albumin decreases (4,7). Therefore, our finding is consistent with the pathophysiology of the acute phase response.

We found a strong positive correlation between albumin and haemoglobin (r=0.682; p=0.001). It is emphasised in the literature that these two parameters are related to both nutritional status and inflammation level and that their evaluation together is important from a prognostic point of view (9). The results of our study support this information.

The negative correlations found between leukocyte and monocyte values and albumin (r=-0.346; p=0.031 and r=-0.342; p=0.033, respectively) reveal an inverse relationship between the inflammatory cellular response and albumin levels. Literature shows that albumin synthesis is suppressed along with an increase in inflammatory cells during the acute phase response (4). We believe this is due to the acute phase reactant secondary to inflammation.

In our study, both cases that resulted in mortality were male. Literature reports that pneumothorax generally has a low mortality rate, but mortality may increase in the presence of underlying lung disease, advanced age, and delayed treatment (1,6). In this context, the fact that the mortal cases in our study were male may be related to the fact that pneumothorax is more common in men.

CONCLUSION

In conclusion, this study found significant correlations between inflammatory markers and albumin and hemoglobin levels in pneumothorax patients. Specifically, the negative correlation of albumin levels with age and inflammation parameters, and the positive correlation with haemoglobin, suggests that albumin may be an important biomarker reflecting systemic inflammation and disease burden.

Our findings are consistent with the literature and support the effect of the inflammatory process on biochemical parameters in pneumothorax patients. Accordingly, we believe that albumin and related parameters can be used in clinical evaluation and possible prognostic prediction.

A noteworthy finding in our study is that all cases resulting in mortality were male. This may be related to the fact that pneumothorax is more common in men, or it may be related to accompanying clinical features and disease severity.

The limited sample size and retrospective nature of our study should be considered. Therefore, the generalizability of the results obtained is limited. Future studies with larger sample sizes and prospective designs are needed to more comprehensively evaluate the impact of these biomarkers on the clinical course, treatment response, and prognosis of pneumothorax.

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5052 **Possible Herbal Toxicity Following Consumption of Corn Poppy (Papaver rhoeas)**

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Introduction

The use of herbal products for traditional therapeutic purposes is widespread worldwide. According to the World Health Organization, a significant proportion of individuals in many societies benefit from herbal products in the treatment of various health problems (1). However, the natural origin of herbal products does not necessarily mean that they are always safe. The alkaloids, glycosides, and other biologically active components they contain may exert pharmacological effects and, in some cases, may lead to toxic outcomes (2).

Corn poppy (Papaver rhoeas) belongs to the Papaveraceae family and grows widely in Europe, the Middle East, and Anatolia. Traditionally, it has been used for the treatment of cough, pain, and sleep disorders due to its sedative, analgesic, and antitussive effects (3). However, because it contains alkaloid compounds, it may exert effects on the central nervous system and may cause side effects in some cases. In this study, we present a case that was

monitored in the toxicology intensive care unit due to symptoms that developed after the consumption of corn poppy.

Case

A 55-year-old female patient with a known diagnosis of hypertension reported that she had consumed a plant commonly known as “corn poppy” due to complaints of cough and generalized body pain persisting for approximately 3–4 days. Following the consumption of the plant, the patient developed dizziness, weakness, and palpitations and initially presented to an external healthcare center.

After the initial evaluation at the external center, the patient was referred to the toxicology intensive care unit of our hospital for further monitoring and management. Upon admission to our hospital, the patient's vital signs were stable and no significant pathological findings were detected during the physical examination.

Laboratory investigations revealed no pathological findings. Imaging studies were also evaluated as normal. The patient was admitted for observation in the toxicology intensive care unit due to possible herbal toxicity.

The patient’s clinical status, vital signs, and laboratory parameters were monitored for two days. Since no complications developed during the follow-up and the patient’s general condition remained stable, she was discharged with recommendations.

Discussion

With the increasing use of herbal products, cases of toxicity related to these products have been reported more frequently. Although herbal products are often perceived as natural and safe, the biologically active substances they contain may cause pharmacological and toxic effects (2). Adverse effects related to herbal products may affect gastrointestinal, neurological, cardiovascular, and hepatic systems (4).

Corn poppy (Papaver rhoeas) is a plant belonging to the Papaveraceae family and contains various isoquinoline alkaloids. Traditionally, it has been used for its sedative, analgesic, and antitussive properties (3). The alkaloid compounds contained in the plant may produce sedative effects on the central nervous system and may lead to clinical findings such as dizziness, weakness, and sedation in some cases (5).

Although serious toxicity related to the consumption of Papaver rhoeas has rarely been reported in the literature, it has been noted that neurological symptoms may occur particularly after high-dose consumption (6). The method of preparation of herbal products, the amount consumed, and individual sensitivity may cause variability in clinical findings (7).

In suspected cases of herbal product-related toxicity, obtaining a detailed medical history and monitoring the patient under appropriate clinical conditions are essential. In most cases, a specific antidote is not available, and treatment consists of supportive management and clinical observation (8). Therefore, close monitoring of symptomatic patients in terms of vital signs and possible complications is recommended.

Conclusion

In the presented case, possible herbal toxicity was considered after the patient developed dizziness, weakness, and palpitations following the consumption of corn poppy, and the patient was monitored in the toxicology intensive care unit. No pathological findings were detected in laboratory or imaging studies, and the patient's clinical course remained stable. The patient was discharged without complications after a two-day follow-up period.

The widespread and uncontrolled use of herbal products may lead to various toxic effects. Although traditionally used plants such as corn poppy are often considered safe, they may cause clinical symptoms due to the biologically active substances they contain. Therefore, it is important to question the use of herbal products in detail in patients presenting with symptoms. Additionally, monitoring patients in an appropriate clinical setting and providing supportive treatment in cases of possible herbal toxicity are essential to prevent complications.

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Keywords: Corn poppy; Papaver rhoeas; herbal toxicity; toxicology intensive care unit; herbal products

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Spontaneous Active Bleeding Secondary to Rupture of a Surrenal Adenoma: Diagnosis and Minimally Invasive Management

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Introduction and Purpose

Spontaneous adrenal hemorrhage (SAH) is a rare but critical clinical entity with high morbidity and mortality risk, requiring rapid diagnosis and multidisciplinary approach. Clinical findings are often non-specific, which may complicate the diagnosis. The aim of this case report is to emphasize the diagnostic process and minimally invasive treatment approach of spontaneous adrenal hemorrhage due to ruptured adenoma.

Case Presentation

A 71-year-old woman was referred to an external center for sudden onset of right flank pain and was referred to our center for further evaluation after a right perirenal hematoma was detected on abdominal computed tomography. It was learned that the patient had a history of hypertension, diabetes mellitus and chronic heart failure and was on antiaggregant therapy. Physical examination revealed right upper and lower quadrant tenderness and right costovertebral angle tenderness. Laboratory tests revealed decreased hemoglobin levels, metabolic acidosis and elevated lactate levels in arterial blood gas. Contrast-enhanced CT examination showed active extravasation from right surrenal adenoma and secondary retroperitoneal hematoma. Emergency blood product replacement was started due to ongoing bleeding and hemoglobin decrease. After multidisciplinary evaluation, interventional radiological treatment was planned as an alternative to surgery and successful transarterial embolization of the vasculature causing bleeding was performed.

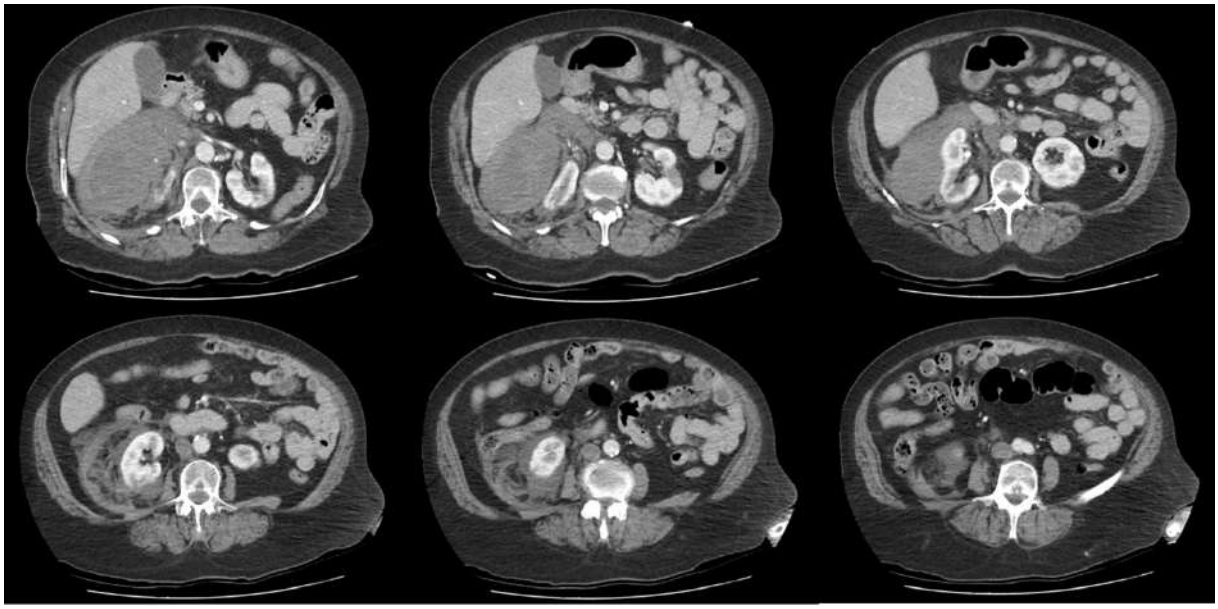

Figure 1. Contrast-enhanced abdominal CT scans showing heterogeneous hematoma and active contrast extravasation in the right surrenal lobe

Results and Conclusion

Spontaneous adrenal hemorrhage should be considered in the differential diagnosis, especially in elderly patients and patients on anticoagulant/antiaggregant therapy. While computed tomography plays a critical role in early diagnosis, interventional radiologic methods offer an effective and safe treatment option as an alternative to surgery in appropriate patients. Multidisciplinary approach is decisive in reducing mortality and morbidity.

Keywords: Adrenal adenoma, spontaneous adrenal hemorrhage, embolization, retroperitoneal hematoma, acute flank pain

Introduction

When surrenal adrenal hemorrhage (SAH) develops due to non-traumatic (spontaneous) causes, neoplasms (adenoma, pheochromocytoma), infections or anticoagulant/anti-aggregant use are frequently involved in the etiology (1-3). The clinical picture may vary in a wide range from adrenal insufficiency findings to hypovolemic shock. Non-specific clinical presentation (abdominal pain, nausea, hypotension) may lead to delays in diagnosis (1,4,5). The gold standard diagnostic method for adrenal hemorrhages is non-contrast and contrast-

enhanced computed tomography (CT) (1,2,6). Active hemorrhage foci detected in the early period can be managed with minimally invasive methods to avoid surgical morbidity. In this case, the management of SAH in an elderly patient with comorbidities was analyzed in the light of current literature.

Case Report

A 71-year-old woman with known hypertension (HT), diabetes mellitus (DM) and chronic heart failure (CHF) and a history of coronary angiography was admitted to an external center with sudden onset of right flank pain. It was learned that the patient regularly used apixaban, atorvastatin, metoprolol succinate, furosemide and metformin at home.

The patient was referred to our tertiary care hospital for further investigation and treatment after a right perirenal hematoma was observed on abdominal CT scan performed at an external center. In the initial physical examination at our center, the general condition was found to be moderate to poor, oriented and coherent. Vital signs; blood pressure arterial (BP): 106/72 mmHg, pulse rate: 102/min, room air saturation (spO2): 96% and temperature: 36.3°C. On abdominal examination, there was marked tenderness in the right upper and lower quadrants and no defense or rebound was detected. Right costovertebral angle tenderness (CVAH) was evaluated as positive.

Laboratory tests revealed that the hemoglobin (hb) level decreased from 9.6 g/dL in the outside center to 9.0 g/dL in our center. Arterial blood gas analysis showed base deficit supporting tissue hypoperfusion: -7, pH: 7.32 and lactate: 4.2 mmol/L findings were found. Coagulation parameters were within normal limits. On CT images re-evaluated by the radiologist; active extravasation (bleeding) from the right surrenal adenoma and secondary hematoma area were diagnosed. Upon detection of an active bleeding focus and continued Hb decline, an emergency erythrocyte suspension (ES) replacement protocol was initiated. As a result of the multidisciplinary evaluation of the urology and interventional radiology clinics, interventional radiologic intervention was planned primarily to reduce surgical risks. The patient was treated under emergency conditions and successful embolization of the vascular structure causing bleeding was performed. After the procedure, the patient was

hemodynamically stabilized and considered to be at risk for recurrence of bleeding, and he was interned to the intensive care unit for close follow-up.

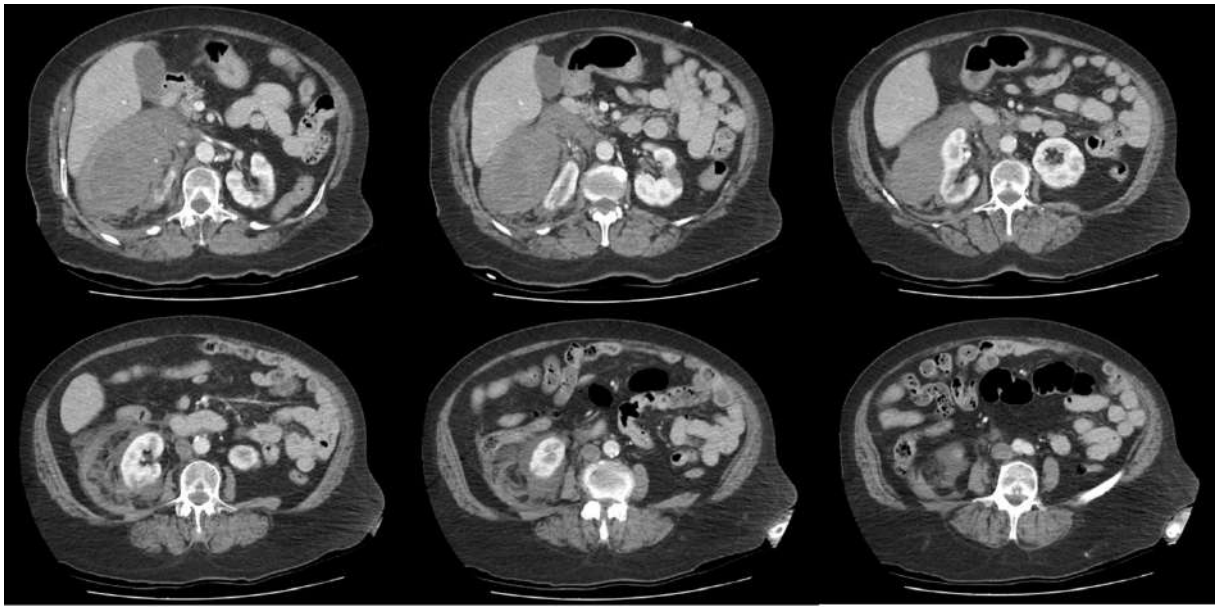

Figure 1. Contrast-enhanced abdominal CT scans showing heterogeneous hematoma and active contrast extravasation in the right surrenal lobe

Discussion

The pathophysiology of surrenal hemorrhage is closely related to the unique vascular anatomy of the gland. Although the adrenal gland is supplied by three separate arteries, it drains through a single central vein ; this creates a "vascular dam" effect that predisposes venous congestion and vascular rupture under physiologic stress conditions (7). It has also been suggested that venous vasoconstriction due to catecholamine discharge may trigger bleeding by increasing intravascular pressure (7). In the present case, antiaggregant therapy (acetylsalicylic acid) and chronic heart failure were considered as facilitating factors that increased the risk of bleeding by increasing vascular fragility.

Although primary adrenal tumors rank first in the etiology of spontaneous adrenal hemorrhage cases with a rate of approximately 40%, myelolipoma and pheochromocytoma are more common among these tumors, while ruptures caused by adrenocortical adenoma are

very rare and reported with a rate of approximately 4% (8). In this respect, the case presented represents one of the rare examples of adenoma rupture in the literature.

Computed tomography (CT) is the gold standard imaging method in the diagnostic process. Adrenal hemorrhage is characterized by heterogeneous density increase, active contrast extravasation and periadrenal infiltration findings in the adrenal region on CT (6). The base deficit (-7) and elevated lactate (4.2 mmol/L) found in our patient indicate that hemorrhage led to systemic hypoperfusion and support clinical severity. It has been reported in the literature that lactate elevation is associated with increased mortality and early intervention is critical for prognosis (3,5).

Although surgical adrenalectomy is the traditional method in the treatment approach, interventional radiologic techniques have recently come to the forefront as an effective and less invasive alternative, especially in hemodynamically unstable patients or patients with high surgical risk (2). In the present case, transarterial embolization, which was preferred as a result of multidisciplinary evaluation, prevented surgical morbidity by providing successful hemostasis. Post-procedure intensive care follow-up is of great importance for early detection of the risk of adrenal insufficiency, especially in the presence of large hematoma or the possibility of bilateral involvement.

Conclusion

Spontaneous adrenal hemorrhage may be considered as a "diagnostic pitfall" due to its clinical course and nonspecific findings. It should be considered in the differential diagnosis especially in elderly patients on antiaggregant or anticoagulant therapy and presenting with sudden flank pain. Early diagnosis with computed tomography and timely endovascular interventions offer an effective and safe treatment option in appropriate cases. In conclusion, patients with suspected SAH should be managed with a multidisciplinary approach including urology, radiology and intensive care units in terms of hormonal balance and surgical necessity.

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Relationship Between Laboratory Parameters And Mortality In Femoral Neck Fractures Presenting To The Emergency Department

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Introduction

Femoral neck fractures are an important cause of trauma characterized by high morbidity and mortality, especially in the elderly population (1). Mortality rates in these patients may reach up to 20–30% within the first year (2).
Among the factors determining prognosis, age, comorbid diseases, and duration of surgical delay are included; however, in recent years, the prognostic value of laboratory parameters has also been investigated (3,4).
In particular, inflammatory markers (CRP, WBC), indicators of renal function (urea, creatinine), and albumin levels reflecting nutritional status have been associated with mortality (5–7).
However, the role of these parameters, which can be obtained rapidly and objectively in the emergency department, in predicting mortality is still not clear. In this study, we aimed to evaluate the relationship between laboratory parameters and mortality in patients presenting with femoral neck fractures.

Materials and Methods

This study was conducted retrospectively on patients presenting to the emergency department with femoral neck fractures between December 1, 2025 and March 1, 2026.
A total of 43 patients were included in the study. Demographic data of the patients and laboratory parameters at admission were obtained from the hospital information system.
In the laboratory evaluation, creatinine (mg/dL), urea (mg/dL), albumin (g/dL), calcium (mg/dL), CRP (mg/L), WBC ($\times 10^3/\mu L$), platelet ($\times 10^3/\mu L$), and hemoglobin (g/dL) values were analyzed.
Normal distribution was assessed using the Shapiro–Wilk test. Parametric data were presented as mean ± standard deviation, and non-parametric data as median (IQR).
Student's t-test and Mann–Whitney U test were used for comparisons between two groups. Spearman and Pearson tests were preferred for correlation analysis. Statistical significance level was accepted as $p<0.05$.

Results

Table 1. Mean and standard deviation values of our parametric data

Variables	n	Mean	SD
Urea(mg/dL)	43	52.04	21.8
Plt (10^3/uL)		235.10	18.53
Hgb (mmol/L)		12.60	1.72

The mean age of the patients was 79 (68–89) years, and the elderly population was predominant.
The length of hospital stay was found to be 9 (7–12) days.
When laboratory parameters were examined:
Urea was 52.05 ± 21.80 mg/dL, platelet was 235.11 ± 18.53 ×10³/µL, and hemoglobin was 12.60 ± 1.73 g/dL.
Among non-parametric variables, creatinine was 0.92 (0.71–1.19) mg/dL, albumin was 3.8 (3.4–4.3) g/dL, CRP was 5.6 (1.0–47.2) mg/L, and WBC was 10.59 (8.28–15.08) ×10³/µL.

In the mortality analysis, no significant difference was found in terms of urea (p=0.388), hemoglobin (p=0.169), and platelet (p=0.433).
Similarly, no statistically significant relationship was found between mortality and age, length of hospital stay, creatinine, albumin, calcium, CRP, and WBC values (all p>0.05).
In the correlation analysis, a moderate positive correlation was found between albumin and calcium (r=0.500; p=0.001).
A weak negative correlation was found between creatinine and calcium (r=−0.363; p=0.017).
No significant relationship was observed among other parameters.

Discussion

In this study, it was shown that the main laboratory parameters examined in patients with femoral neck fractures were not significantly associated with mortality.
In the literature, low albumin levels have been reported to be associated with poor prognosis (6). Similarly, high CRP and leukocyte values have been reported to increase mortality by reflecting the inflammatory response (5,7).
However, in our study, these parameters were not found to be associated with mortality. This may be due to the limited sample size and the low number of mortality cases.
Although the prognostic value of renal parameters such as urea and creatinine has been previously demonstrated (8), our results do not support this relationship.
In the correlation analysis, the moderate relationship between albumin and calcium may be considered a physiological finding consistent with the calcium-binding role of albumin (9).
Overall, our findings indicate that laboratory parameters alone are not sufficient to predict mortality and should be evaluated together with clinical factors.

Conclusion

Laboratory parameters at admission were not found to be significant in predicting mortality in patients presenting with femoral neck fractures.
Clinical scores and comorbid conditions are considered to be more determinant in the evaluation of prognosis.

Limitations

The study has a single-center and retrospective design.
The sample size and the number of mortality cases are low.
Clinical scoring systems and comorbid diseases were excluded from the analysis.

Abstract

This retrospective study examined the prognostic relevance of admission laboratory parameters in 43 patients who presented to the emergency department with femoral neck fractures between December 2025 and March 2026. The median patient age was 79 (IQR: 68–89) years, and the median length of hospital stay was 9 (IQR: 7–12) days. Parametric variables included urea (52.05 ± 21.80 mg/dL), platelet count (235.11 ± 18.53 $\times 10^3/\mu L$), and hemoglobin (12.60 ± 1.73 g/dL), while creatinine, albumin, CRP, and WBC were analyzed as non-parametric data. Mortality analysis revealed no statistically significant association with any of the measured parameters, including urea ($p=0.388$), hemoglobin ($p=0.169$), platelet count ($p=0.433$), creatinine, albumin, calcium, CRP, and WBC (all $p>0.05$). Correlation analysis demonstrated a moderate positive relationship between albumin and calcium ($r=0.500$; $p=0.001$) and a weak negative correlation between creatinine and calcium ($r=-0.363$; $p=0.017$). These findings suggest that routine admission laboratory values alone are insufficient to predict in-hospital mortality in femoral neck fracture patients, and that comprehensive prognostic assessment should incorporate clinical scoring tools and comorbidity profiles.

Keywords: femoral neck fracture, emergency department, laboratory parameters, mortality, prognosis

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Differentiation Between Osteomyelitis and Malignancy in Bone Lesions: A Report of Four Cases

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Abstract

Aim: The differentiation between osteomyelitis and malignancy in bone lesions detected in patients presenting with bone pain is of critical clinical importance. This study aimed to present four cases in which diagnostic difficulty was encountered and to evaluate them in the context of the relevant literature.

Case Presentation: Three cases of osteomyelitis and one case of bone metastasis are presented. In the first case, osteomyelitis developing after trauma mimicked malignancy. In the second case, osteomyelitis was diagnosed despite the absence of prominent signs of infection. In the third case, bone metastasis was detected in a patient with no prior history of primary malignancy. In the fourth case, although osteomyelitis clinically mimicked malignancy following a traumatic femur fracture, the diagnosis was established pathologically by biopsy.

Conclusion: The differentiation between osteomyelitis and malignancy in bone lesions must be made through careful clinical evaluation, imaging, and histopathological examination. Misdiagnosis may lead to unnecessary surgical interventions or delays in treatment.

Keywords: Osteomyelitis, Bone metastasis, Bone tumor, Differential diagnosis, Case series

Introduction

With advances in medical treatment modalities and imaging techniques, there has been a notable increase in the number of patients presenting to emergency departments and orthopedic clinics with bone pain (1). Bone lesions represent one of the most significant causes of bone pain, and accurate differentiation of these lesions is of great importance for clinical management (2).

In the differential diagnosis of bone lesions, in addition to benign and malignant processes, infectious pathologies must also be considered. Osteomyelitis is an infection of bone tissue that may mimic malignant tumors, particularly in long bones. This situation can lead to misdiagnosis and unnecessarily aggressive treatments (3).

This study aimed to present four cases admitted to our clinic in which diagnostic difficulty was encountered in differentiating osteomyelitis from malignancy, and to evaluate them in the context of the relevant literature.

Case Reports

Case 1

A 16-year-old male patient, who had previously undergone surgery for a left femur subtrochanteric fracture, presented to our clinic with a complaint of recurrent and progressively worsening pain in the left knee. Imaging studies revealed a mass lesion at the proximal left tibia, and malignancy was considered as a preliminary diagnosis.

Biopsy and debridement were performed from the proximal left tibia. Histopathological examination of the specimens was reported as osteomyelitis. Appropriate antibiotic therapy was initiated according to culture results, and the patient was treated without the need for surgical resection.

The source of osteomyelitis was attributed to infection developing after skeletal traction applied following the prior femur fracture. No recurrence was observed during follow-up.

Case 2

A 9-year-old male patient presented with a complaint of pain radiating from the posterior of

the left thigh to the knee, persisting for approximately three months. Physical examination revealed no significant erythema or increased warmth.

A lesion was identified at the proximal femur based on imaging and laboratory findings. As differentiation between malignancy and osteomyelitis could not be established, a biopsy was obtained. Histopathological examination was consistent with osteomyelitis, and methicillin-sensitive *Staphylococcus aureus* grew in culture.

The patient was treated with appropriate antibiotic therapy without the need for surgical intervention. Clinical improvement was achieved during follow-up.

Case 3

A 59-year-old male patient presented with a complaint of left ankle pain and swelling persisting for one month. Imaging studies revealed a lytic lesion in the distal shaft of the left fibula, and the patient was operated on with a preliminary diagnosis of malignancy.

Resection and intramedullary nailing were performed. Histopathological examination of intraoperative specimens revealed metastasis of renal cell carcinoma (RCC). Following surgery, the patient was referred to the oncology clinic for treatment planning targeting the primary tumor.

Case 4

An 18-year-old male patient, who had previously undergone surgery for a right femur-tibia fracture, presented to our clinic with a complaint of recurrent and progressively worsening pain in the right knee. Imaging studies revealed a mass lesion at the proximal left tibia, and malignancy was considered as a preliminary diagnosis.

Biopsy and debridement were performed from the proximal left tibia. Histopathological examination of the specimens was reported as osteomyelitis. Appropriate antibiotic therapy was initiated according to culture results, and the patient was treated without the need for surgical resection.

The source of osteomyelitis was attributed to infection developing after skeletal traction applied following the prior femur fracture. No recurrence was observed during follow-up.

Discussion

In this study, three cases illustrating the difficulty of differentiating between osteomyelitis and malignancy in the differential diagnosis of bone lesions are presented. In two cases, osteomyelitis mimicked malignancy, while in one case, bone metastasis presented as the initial finding.

Osteomyelitis, particularly in its subacute and chronic forms, may exhibit tumor-like radiological appearances. This can raise suspicion of malignancy, especially in pediatric and young patients (3). In our first case, careful evaluation of the history of trauma and prior surgery enabled the correct diagnosis to be reached.

In the second case, the absence of prominent signs of infection was an important factor complicating the diagnosis. The literature also reports that osteomyelitis may not always follow the classic course of infection (2).

In the third case, bone metastasis presented as the initial finding in a patient with no history of primary malignancy. Metastatic bone disease is one of the important causes of bone pain, particularly in elderly patients, and may represent the first manifestation of systemic disease (1,4).

In the fourth case, a patient with a prior surgical history developed pain, and imaging revealed a suspicious mass appearance suggesting malignancy; however, following biopsy, the diagnosis of osteomyelitis was established pathologically. (2)

These cases demonstrate that imaging findings alone are insufficient in the evaluation of bone lesions and that histopathological confirmation is always required.

Conclusion

The differentiation between osteomyelitis and malignancy in patients presenting with bone lesions is of critical clinical importance. A detailed patient history, careful physical examination, appropriate imaging modalities, and histopathological examination must be evaluated together. Accurate diagnosis is of great importance for preventing unnecessary surgical interventions, ensuring appropriate treatment planning, and improving patient prognosis.

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Contrast-induced nephropathy following repeated contrast exposure: a case report

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ABSTRACT

In this case report, we aimed to present a patient who developed progressive acute kidney injury with a fatal outcome following repeated exposure to contrast media within a short period in the presence of advanced age, diabetes mellitus, and heart failure. A 75-year-old male presented to the emergency department with complaints of dyspnea, wheezing respiration, and abdominal distension. The patient's medical history revealed that he had undergone two contrast-enhanced computed tomography scans four days apart. It was also noted that colchicine and ibuprofen had been initiated due to pericardial effusion. Laboratory tests obtained in the emergency department showed a blood urea level of 93 mg/dL, serum creatinine level of 1.8 mg/dL, and C-reactive protein (CRP) level of 144 mg/L, while urine output was 50 mL/h. On the third day of intensive care unit follow-up, blood urea increased to 190 mg/dL and creatinine to 3.7 mg/dL. On the fourth day, urea was 240 mg/dL and creatinine was 4 mg/dL. Despite intensive care management, the patient's renal function progressively deteriorated and he died on the fifth day. In elderly patients receiving repeated contrast media administration, a history of diabetes mellitus, heart failure, and nonsteroidal anti-inflammatory drug use should be recognized as important risk factors for the development of contrast-induced nephropathy.

Keywords: Emergency, Acute kidney injury, Contrast-induced nephropathy.

INTRODUCTION

Iodinated contrast media are indispensable components of modern diagnostic imaging, particularly in computed tomography and angiographic procedures. However, acute

deterioration in renal function following contrast administration remains an important cause of morbidity and mortality in clinical practice.

Contrast-induced nephropathy (CIN) is defined as renal dysfunction characterized by either a 25% increase in serum creatinine from baseline or an absolute increase of 0.5 mg/dL (44 µmol/L) in serum creatinine within 48–72 hours following intravenous contrast administration (1). The renal failure associated with contrast exposure is typically acute and usually develops within 2–3 days after contrast administration (2).The incidence of CIN in the general population has been reported to be greater than 2%; however, in high-risk groups—such as patients with diabetes mellitus, congestive heart failure, chronic kidney disease, and advanced age—the incidence may exceed 20–30% (3).In this case report, we aimed to present a fatal case of progressive acute kidney injury developing after repeated contrast exposure in a patient with advanced age, diabetes mellitus, and heart failure.

CASE REPORT

A 75-year-old male with known diagnoses of diabetes mellitus and heart failure presented to the emergency department with complaints of dyspnea, wheezing respiration, and abdominal distension. The patient's medical history revealed that he had undergone two contrast-enhanced computed tomography examinations four days apart. In addition, colchicine and ibuprofen had been initiated due to pericardial effusion. At initial evaluation, vital signs were as follows: body temperature 36.8°C, heart rate 120 beats/min, blood pressure 140/90 mmHg, respiratory rate 22 breaths/min, and oxygen saturation 80%. Capillary blood glucose was measured at 250 mg/dL. Physical examination revealed coarse breath sounds with wheezing respiration, tachypnea, and abdominal breathing. Laboratory tests obtained in the emergency department demonstrated a blood urea level of 93 mg/dL, serum creatinine level of 1.8 mg/dL, and C-reactive protein (CRP) level of 144 mg/L. Urine output was measured at 50 mL/h. A nephrology consultation was requested, and the patient was subsequently admitted to the intensive care unit. Review of the patient's previous laboratory results revealed a baseline urea level of 46 mg/dL and serum creatinine level of 0.8 mg/dL. During intensive care follow-up, renal function deteriorated progressively. On the third day of

ICU hospitalization, blood urea was 190 mg/dL and creatinine was 3.7 mg/dL. On the fourth day, blood urea increased to 240 mg/dL and creatinine to 4 mg/dL. Despite aggressive supportive therapy, including fluid management and other intensive care interventions, renal function continued to worsen. The patient died on the fifth day of ICU follow-up.

DISCUSSION

Contrast-induced nephropathy is increasingly referred to in the recent literature as contrast-associated acute kidney injury (CA-AKI) (4). Contrast agents may trigger tubular epithelial injury by reducing renal blood flow and causing medullary hypoxia. This process tends to be more severe in conditions that impair renal perfusion, such as diabetes mellitus, advanced age, and heart failure (5). In our case, the patient had several predisposing factors for contrast-induced nephropathy, including diabetes mellitus, heart failure, and advanced age (75 years).

Repeated exposure to contrast media is also a significant risk factor. Administration of contrast agents within the critical 48–72 hour period may exacerbate renal injury and deepen kidney damage (6). In our case, the patient underwent two contrast-enhanced computed tomography examinations within a four-day interval.

In addition, the use of nonsteroidal anti-inflammatory drugs (NSAIDs) may inhibit prostaglandin synthesis in the renal afferent arteriole, leading to a reduction in glomerular filtration rate and potentially worsening contrast-related renal injury (7). It was also determined that our patient had been receiving colchicine and ibuprofen due to pericardial effusion.

The most effective strategy in the management of contrast-induced nephropathy is prevention. Isotonic fluid hydration, avoidance of nephrotoxic agents, and the use of the minimal effective contrast dose constitute the main preventive measures (8). In our case, all supportive treatments—including fluid hydration—were implemented; however, the patient's renal function continued to deteriorate.

Conclusion

In elderly patients receiving repeated contrast media administration, the presence of diabetes mellitus, heart failure, and a history of nonsteroidal anti-inflammatory drug use should be recognized as significant risk factors for the development of contrast-induced nephropathy.

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The Role of CRP, Albumin, and CAR in Predicting Mortality in Pneumonia Patients Aged ≥75 Years in the Intensive Care Unit

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Objective:
This study aimed to evaluate the association of C-reactive protein (CRP), albumin, and the CRP/albumin ratio (CAR) with mortality in patients aged ≥75 years with pneumonia admitted to the intensive care unit from the emergency department.

Design:
Retrospective observational study.

Setting:
Emergency department and intensive care unit of a tertiary care hospital.

Subjects:
Patients aged ≥75 years diagnosed with pneumonia and admitted to the intensive care unit.

Methods:
Demographic and laboratory data were obtained from hospital records. Statistical analyses were performed using IBM SPSS Statistics version 27. Normality was assessed using the Kolmogorov–Smirnov test. Parametric variables were expressed as mean ± standard deviation, and non-parametric variables as median (IQR). Independent samples t-test and Mann–Whitney U test were used for group comparisons.

Results:
A total of 69 patients were included. The mean age was 82.9 ± 5.4 years. The overall mortality rate was 26.1%. Age was significantly higher in non-survivors (p=0.028). CRP levels were significantly associated with mortality (p=0.001). No significant association was found between albumin levels and mortality (p=0.185). CAR values were significantly higher in non-survivors (p=0.001).

Conclusion:
CRP and especially CAR are significantly associated with mortality in elderly patients with

pneumonia. Albumin alone appears to have limited prognostic value. CAR may serve as a useful biomarker for mortality prediction by reflecting both inflammatory status and nutritional condition.

Keywords: Pneumonia, Intensive Care Unit, C-Reactive Protein, Albumin, C-Reactive Protein/Albumin Ratio, Mortality, Elderly

Introduction

Pneumonia is an important cause of morbidity and mortality, particularly in the elderly population. Age-related decline in physiological reserve, increased burden of comorbid diseases, and changes occurring in the immune system are associated with a more severe clinical course and increased mortality in patients with pneumonia.(1) "In elderly patients, pneumonia leads to a more complex clinical presentation, driven not only by the infection itself but also by the impact of concomitant chronic diseases."(2) "Early risk stratification in elderly pneumonia patients admitted to the intensive care unit from the emergency department is of critical importance for determining appropriate treatment strategies and reducing mortality. Although clinical scoring systems such as CURB-65 and PSI are widely used for this purpose, it has been reported that these scores may remain limited in certain patient populations, necessitating additional biomarkers."(3) In recent years, inflammatory biomarkers have been widely utilized to predict disease severity and mortality in patients with pneumonia. C-reactive protein (CRP) is a prominent acute-phase reactant known to reflect systemic inflammation and correlate with infection severity."(4) Albumin, on the other hand, is a negative acute-phase reactant and serves as a parameter that collectively reflects inflammation, nutritional status, and chronic disease burden."(5) The C-reactive protein/albumin ratio (CAR), a combination of these two parameters, has emerged as a potent prognostic marker in recent years due to its ability to simultaneously evaluate both inflammatory response and nutritional status. Particularly in critically ill patients and in the presence of infection, CAR has been demonstrated to correlate with mortality and disease severity."(6)

Aims

In this study, we aimed to evaluate the relationship between mortality and CRP, albumin, and CAR values in pneumonia patients aged 75 and older who were admitted to the intensive care unit from the emergency department.

Metods

In this study, we aimed to evaluate the relationship between mortality and CRP, albumin, and CAR values in pneumonia patients aged 75 and older who were admitted to the intensive care unit from the emergency department. This study was conducted by retrospectively reviewing pneumonia patients aged $\ge$75 years admitted from the emergency department to the

intensive care unit. Demographic characteristics and laboratory parameters of the included patients were obtained from the hospital registry system.

Statistical analyses were performed using IBM SPSS Statistics 27 software. The conformity of continuous variables to normal distribution was assessed using the Kolmogorov–Smirnov test. Parametric data were expressed as mean $\pm$ standard deviation, whereas non-parametric data were expressed as median and interquartile range (IQR, 25th–75th percentiles). For intergroup comparisons, the independent samples t-test was used for normally distributed data, and the Mann–Whitney U test was used for non-normally distributed data. Statistical significance was set at $p<0.05$."

Results

"A total of 69 patients were included in the study. The mean age of the patients was determined to be 82.9 $\pm$ 5.4 years. While the median CRP level was 12 (IQR: 6.3), the mean albumin level was found to be 2.8 $\pm$ 0.4. The median CAR value was 3.8 (IQR: 2.7–5.5). Detailed data regarding demographic and laboratory parameters are presented in Table 1."

Table 1. Demografik ve Laboratuvar Parametreleri (Parametrik Veriler Ortalama ± SS, Non-parametrik Veriler Medyan [IQR])

Parametreler	n	Mean	SD	Median	IQR %25-75
Yaş	69	82,9	5,4		
CRP		12	6,3		
ALB		2,8	0,4		
CAR				3,8	2,7-5,5

Of the patients, 60.9% (n=42) were female and 39.1% (n=27) were male. The overall mortality rate was determined to be 26.1% (n=18).

A statistically significant difference was found between mortality and age (p=0.028), with a higher mean age in patients who developed mortality. A statistically significant relationship was found between CRP level and mortality (p=0.001), and CRP values were higher in patients who deceased. No statistically significant difference was found between albumin and mortality (p=0.185). A statistically significant relationship was saptandı between CAR and mortality (p=0.001), and CAR values were higher in patients who developed mortality."

Discussion

Although there are studies reporting a male predominance among pneumonia patients, the higher proportion of females in our study may be attributed to the advanced age of the patient population. The fact that women have a longer life expectancy, and are thus more heavily represented in older age groups, could explain this situation.(7) "A statistically significant difference was found between mortality and age (p=0.028), with a higher mean age observed in patients who developed mortality. This finding is consistent with the literature demonstrating that increasing age is a significant risk factor for mortality in pneumonia (de Miguel-Yanes et al.). Advanced age is associated with a poor prognosis due to factors such as decreased physiological reserve, increased comorbidity burden, and a decline in immune response." In our study, CRP levels were found to be significantly higher in patients who developed mortality. This is consistent with studies demonstrating that increased systemic inflammation in pneumonia is associated with a poor prognosis.(8) "Previous studies have reported that elevated CRP is associated with mortality and disease severity."(9) The association of CRP with mortality in our cohort supports the significance of inflammatory burden on clinical outcomes in elderly pneumonia patients requiring intensive care.

In our study, no statistically significant relationship was found between albumin levels and mortality (p=0.185). Albumin is a negative acute-phase reactant, and its levels are expected to decrease during inflammatory states. Although our findings did not reach statistical significance, there are studies reporting that low albumin levels are associated with increased mortality and poor clinical outcomes in pneumonia patients."(10) "Particularly in studies conducted on patients with community-acquired pneumonia, hypoalbuminemia has been demonstrated to be an independent predictor of mortality."(11) "The lack of association between our albumin values and mortality may be explained by the fact that albumin levels reflect not only inflammation but also nutritional status, chronic disease burden, and liver functions. In the elderly population, albumin levels often follow a chronically low course."(12) "Therefore, the impact of the acute inflammatory response on albumin may have remained limited. The fact that our study exclusively included intensive care patients might have resulted in a similar degree of inflammation across all patients, which may have prevented the formation of a statistically significant difference between the groups. Furthermore, because albumin is a parameter with a long half-life, its capacity to reflect acute changes is limited. Consequently, serum albumin levels may have remained weaker in predicting acute mortality compared to rapidly changing biomarkers such as CRP.

In our study, a statistically significant relationship was found between the CAR and mortality (p=0.001), with higher CAR values observed in patients who developed mortality. CAR, a combination of CRP and albumin levels, is a biomarker that collectively reflects both inflammation and nutritional status. There are studies reporting that high CAR values are associated with increased disease severity and mortality in pneumonia patients. Particularly, CAR has been demonstrated to have a higher prognostic performance compared to CRP or albumin alone."(13) "This situation can be explained by the fact that the CAR reflects both an increased inflammatory response and a decreased albumin level simultaneously. Although no statistically significant relationship was found between albumin levels and mortality in our study, the significance of the CAR is noteworthy. The fact that albumin levels reflect not only inflammation but also nutritional status, chronic disease burden, and liver functions may have

limited its independent prognostic value. Evaluating elevated CRP and decreased albumin levels together can more accurately demonstrate the severity of systemic inflammation. Therefore, the CAR can be considered a more powerful biomarker in predicting mortality in pneumonia patients. The findings of our study support the prognostic significance of the CAR in elderly pneumonia patients monitored in the intensive care unit."

Conclusion

"In conclusion, it was found that CRP and especially CAR values were significantly associated with mortality in elderly pneumonia patients. In contrast, albumin levels alone were found to be insufficient in predicting mortality. Since CAR reflects both inflammatory response and nutritional status together, it is thought that it may be a more powerful biomarker in predicting mortality in elderly pneumonia patients requiring intensive care.

These findings support that CAR can be used for early risk stratification in clinical practice. This relationship needs to be confirmed by prospective studies with larger sample sizes."

Limitations

"This study has several limitations. First, the retrospective design may cause selection bias and limit causal interpretations. Second, the relatively small sample size and the fact that the study was conducted at a single center may limit the generalizability of the findings. Third, only laboratory parameters at the time of admission were evaluated, and the temporal changes in CRP, albumin, and CAR values were not examined. Fourth, potential confounding factors such as comorbidities, nutritional status, and disease severity scores (e.g., CURB-65 or APACHE II) were not fully analyzed. Lastly, the inclusion of only elderly patients admitted to the intensive care unit may limit the generalizability of the results to other patient populations."

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5453

Bradycardia, renal failure, AV nodal blockade, shock, and hyperkalemia (BRASH) syndrome presenting with cardiac arrest: a case report

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Introduction and Objective

BRASH syndrome is a clinical entity characterized by the synergistic effect of bradycardia, renal failure, atrioventricular (AV) nodal blocker use, shock, and hyperkalemia. It is often underrecognized in emergency settings and may rapidly progress to cardiac arrest. We aimed to present a case of BRASH syndrome with an atypical and severe presentation requiring prolonged resuscitation.

Materials and Methods

A 78-year-old female with a history of hypertension and diabetes mellitus, using metformin (Glifor) and trandolapril/verapamil (Tarka Forte), was brought to the emergency department due to generalized weakness and altered mental status. Initial prehospital vital signs included unmeasurable blood pressure, heart rate 79 bpm, oxygen saturation 97%, and blood glucose 189 mg/dL. The patient developed cardiac arrest during transfer.

Results

Cardiopulmonary resuscitation (CPR) was initiated immediately. Initial blood gas analysis revealed severe metabolic acidosis (pH 6.9, bicarbonate 6 mmol/L), hyperkalemia (6.5 mmol/L), and elevated lactate (unmeasurable). Calcium, sodium bicarbonate, and adrenaline were administered during CPR. Return of spontaneous circulation (ROSC) was achieved after 30 minutes. Post-ROSC, the patient remained hypotensive (40/20 mmHg) and bradycardic

(40 bpm) with slow atrial fibrillation on ECG. Despite atropine administration, aggressive fluid resuscitation, and vasopressor/inotropic support (noradrenaline, dopamine, dobutamine, and adrenaline infusion), the patient remained refractory. Laboratory findings showed acute kidney injury (creatinine 2.6 mg/dL), mild troponin elevation, and elevated liver enzymes. Imaging ruled out pulmonary embolism. The clinical picture was consistent with BRASH syndrome.

Conclusion

BRASH syndrome should be considered in elderly patients using AV nodal blocking agents who present with shock and bradycardia, especially in the presence of renal dysfunction and hyperkalemia. Early recognition and targeted treatment are critical, as conventional resuscitation strategies may be insufficient. This case highlights the potential for rapid deterioration and cardiac arrest in BRASH syndrome.

Keywords

BRASH syndrome; hyperkalemia; bradycardia; cardiac arrest; emergency medicine

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Conversion to Roux-en-Y and Endoscopic Stenting in the Management of Leak After Mini-Gastric Bypass: A Case Report Complicated by Stent Migration

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Introduction

Obesity is a major public health problem with an increasing global prevalence and is associated with numerous comorbidities, particularly type 2 diabetes mellitus, cardiovascular diseases, and certain malignancies (1). Compared with medical therapy, bariatric surgical methods are considered the most effective treatment option for achieving long-term weight loss and improving metabolic parameters (2).

Currently, the most commonly performed bariatric procedures include laparoscopic sleeve gastrectomy (LSG) and Roux-en-Y gastric bypass (RYGB) (3). While LSG is a restrictive procedure based on reducing gastric volume by approximately 75–80%, RYGB is a combined procedure with both restrictive and malabsorptive effects. Both techniques provide significant weight loss and marked improvement in obesity-related comorbidities (2,3). However, bariatric surgical interventions may be associated with serious complications. One of the most important and feared complications is postoperative leakage (4). Leaks developing from the staple line after sleeve gastrectomy or from anastomotic sites after gastric bypass may lead to severe morbidity, prolonged hospital stay, need for reintervention, and rarely mortality (5).

In the literature, the leak rate after sleeve gastrectomy is reported to be approximately 1–2.4%, decreasing to as low as 0.5% in experienced centers (4,6). Both mechanical factors (increased intraluminal pressure and technical errors) and ischemic factors (inadequate blood supply at the staple line) play a role in leak development (7). Leaks after gastric bypass

typically occur at the anastomotic line and may be associated with technical insufficiency, tissue ischemia, or distal obstruction (8). Early recognition and appropriate management of postoperative leaks represent one of the major challenges in bariatric surgery. Treatment approaches vary depending on the timing of the leak, the patient's clinical condition, and the severity of the leak, requiring a multidisciplinary approach. Radiological, endoscopic, and surgical treatment options should be considered together (4,5).

Endoscopic methods are increasingly preferred in the management of postoperative leaks, particularly self-expandable metal stents (SEMS), which have emerged as an effective and minimally invasive treatment option (1). Stents reduce intraluminal pressure by sealing the leak site and promote tissue healing. The success rate of stent application has been reported to be between 70–90%, with higher success rates when applied early (2,3). However, complications such as stent migration, pain, nausea, and rarely perforation may occur (3).

Stent migration is one of the most common complications associated with SEMS use in leak management after bariatric surgery. Migration may occur due to inadequate fixation in the tubular gastric structure after sleeve gastrectomy, low friction coefficient of fully covered stents, inappropriate stent diameter and length selection, and technical factors during placement. Additionally, gastric peristalsis, intraluminal pressure changes, and the absence of adjunct fixation methods increase the risk of migration (1–3).

In this study, a case complicated by a postoperative leak following bariatric surgery is presented and discussed in light of the current literature.

Case

A 47-year-old female patient presented to the emergency department with abdominal pain. She had no known comorbidities. Her history revealed that she had undergone sleeve gastrectomy 6 years earlier, and due to weight regain, revision surgery had been planned, leading to a mini-gastric bypass procedure performed at another center 6 days prior. The patient reported worsening abdominal pain over the last 24 hours, causing limitation of movement.

On physical examination, there was diffuse abdominal tenderness with positive guarding and rebound. Vital signs were as follows: temperature 37.6°C, heart rate 102/min, blood pressure 117/72 mmHg, and oxygen saturation 96% on room air. Laboratory analysis showed a leukocyte count of 22,000/mm³. Oral and intravenous contrast-enhanced abdominal CT revealed widespread contrast extravasation in the abdomen, and the patient was taken for emergency surgery (Figure1-3).

After prophylactic antibiotic administration (third-generation cephalosporin), the patient was operated on under general anesthesia. Entry into the abdomen was achieved through the previous incision using a visiport, and additional trocars were placed at prior port sites. Exploration revealed widespread purulent fluid and dense adhesions, which were carefully dissected. The gastroenterostomy line was evaluated, and no pathology was detected at the anastomosis.

A “bird’s eye” shaped leak was identified at the remnant stomach staple line. Intraoperative endoscopy confirmed the leak and excluded additional leak sites. Based on these findings, Roux-en-Y gastric bypass revision and endoscopic stenting were planned. The mini-gastric bypass anatomy was evaluated, intestinal limbs were counted, and a biliopancreatic limb was created approximately 60 cm distal to the gastroenterostomy. The anastomosis was performed using a stapler, and the opening was closed with 3/0 PDS sutures. Omentopexy was applied to the leak site. A gastric stent was placed endoscopically during the operation. The abdomen was irrigated with saline, and two drains were placed in the operative field and pelvis before concluding the procedure.

Postoperatively, antibiotic therapy was arranged in consultation with infectious diseases specialists. Drain output was serohemorrhagic. Stent position was monitored by direct radiographs on postoperative days 1, 3, and 7. Laboratory parameters improved rapidly; leukocyte levels normalized by postoperative day 2, and CRP decreased to 50 mg/L. Following completion of antibiotic therapy, the patient was discharged in good condition on postoperative day 10.

On postoperative day 27, the patient reported recurrent abdominal pain after jumping into a pool from a height of approximately 3 meters while swimming. She was invited for

evaluation. Direct radiography revealed distal migration of the stent into the small intestine (Figure 4). After appropriate fasting, endoscopy was performed under sedation. The migrated stent was successfully retrieved and a new stent was placed during the same session without complications.

During follow-up, no stent migration was observed on postoperative days 1, 3, and 7. At the fourth-week evaluation, the stent was removed, and the patient was discharged in complete clinical recovery.

Discussion

Leaks following bariatric surgery, although relatively rare, are among the most significant complications due to their high morbidity and mortality risk (1). Particularly in revision bariatric surgeries, the risk of leakage is higher compared to primary procedures due to tissue changes, adhesions, and vascular alterations caused by previous operations (2). In the present case, prior sleeve gastrectomy followed by mini-gastric bypass created a predisposed environment for leak development.

The patient's clinical status is the primary determinant in leak management, and minimally invasive approaches are preferred in hemodynamically stable patients (3). In this context, endoscopic methods, particularly SEMS applications, have become widely used as alternatives to surgery. Stents reduce intraluminal pressure by sealing the leak site and facilitate healing. Reported success rates of SEMS range between 70–90% (4,5).

However, one of the most important limiting factors of stent therapy is migration. Stent migration is more common due to factors such as tubular gastric anatomy after sleeve gastrectomy, wide lumen, low friction of fully covered stents, and insufficient fixation (4,6). In this case, late postoperative stent migration was noteworthy, and the onset of symptoms following physical activity was considered a triggering factor. Literature also suggests that intraluminal pressure changes and sudden physical movements may increase the risk of migration (6).

In addition to endoscopic approaches, surgical revision remains an important option in leak management when necessary. Particularly in cases where leak control cannot be achieved or

anatomical correction is required, surgical intervention becomes unavoidable (3). In this case, revision to Roux-en-Y gastric bypass combined with simultaneous stent placement was performed. This combined strategy successfully controlled the leak and accelerated clinical recovery.

In conclusion, the management of leaks after bariatric surgery is a complex process requiring a multidisciplinary approach. While endoscopic stent applications provide high success rates with appropriate patient selection, complications such as stent migration should be carefully monitored. This case contributes to the literature by demonstrating the effectiveness of combined revision surgery and endoscopic treatment and by highlighting the significant complication of stent migration.

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Figure1. Patient's CT scan image

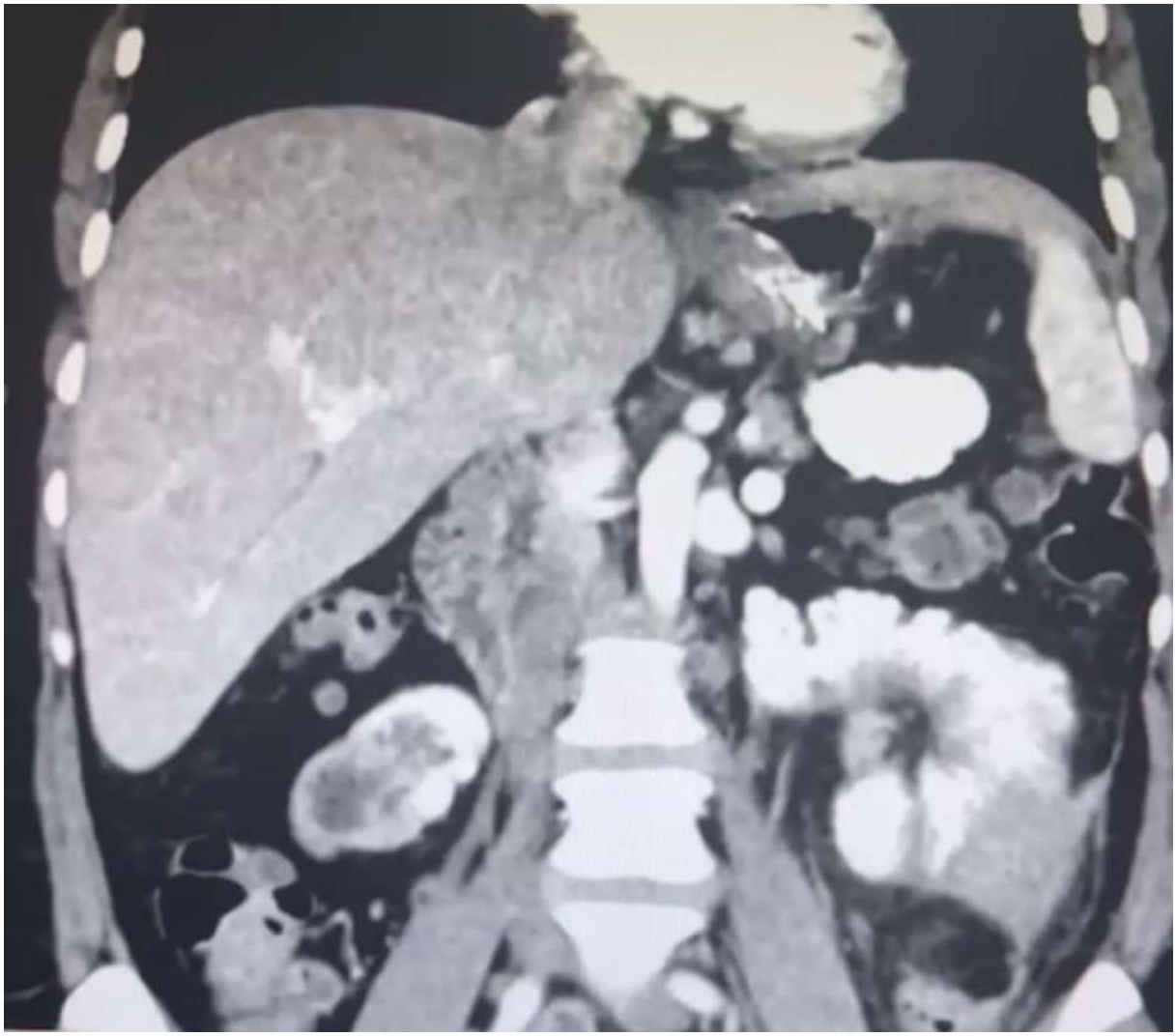

Figure 2. Patient's CT scan image

Figure 3. Patient's X-ray scan image

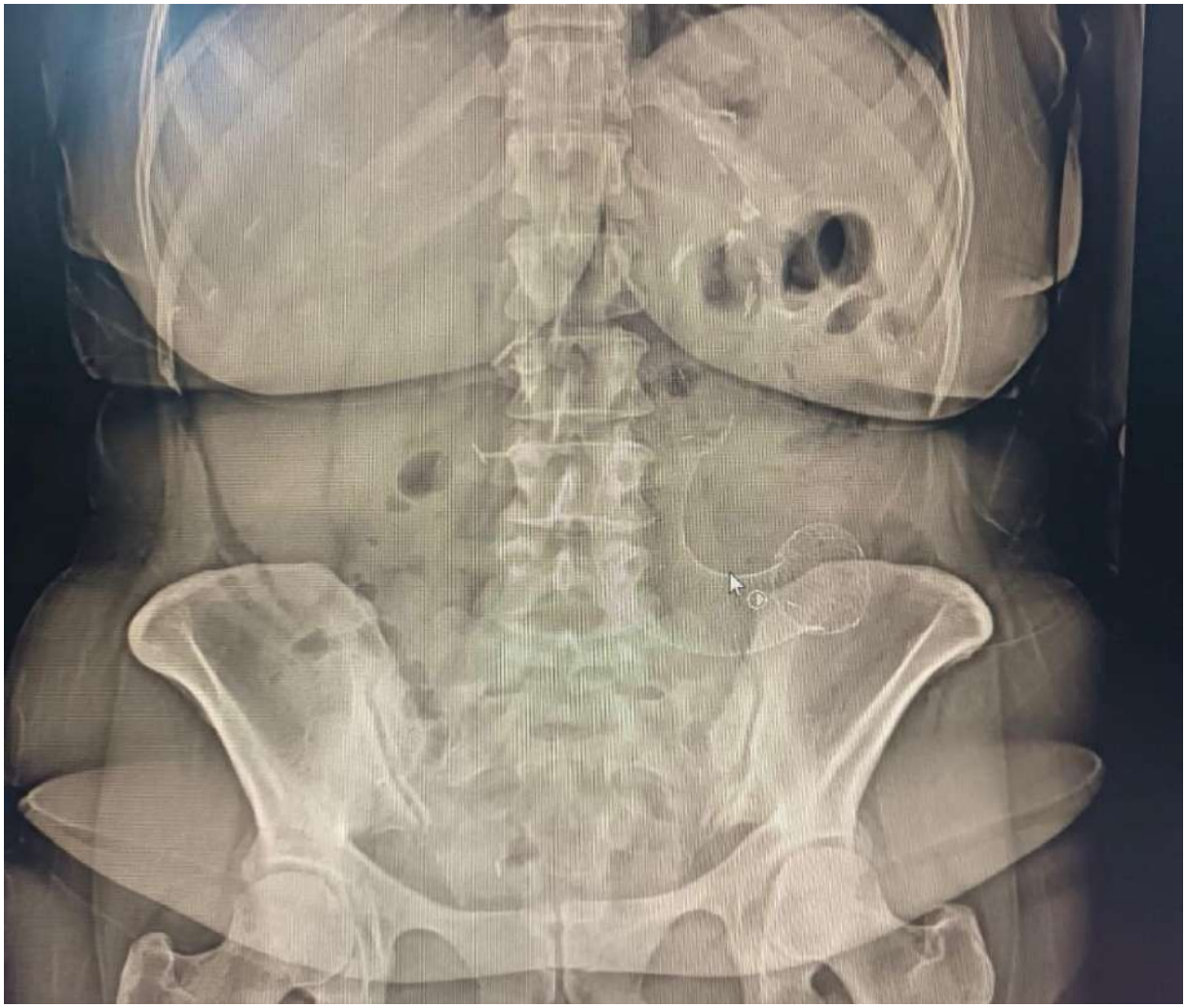

Figure 4. Patient's X-ray scan image (stent migration)

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Evaluation of the Relationship Between Inflammatory Markers and Metabolic Parameters in Diabetic Patients

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Abstract

Objective: This study aims to evaluate the relationship between inflammatory markers (CRP, ferritin, sedimentation rate) and metabolic parameters, as well as albumin levels, in diabetic patients.

Materials and Methods: A total of 31 diabetic patients were enrolled in the study. Demographic characteristics, anthropometric measurements, and laboratory parameters of the patients were analyzed retrospectively. The distribution of data was assessed using the Kolmogorov-Smirnov test. Due to the non-parametric distribution, the Spearman correlation test was utilized for correlation analysis. Independent samples t-test was performed for comparisons between genders.

Results: The mean age of the patients was 65.45 ± 9.97 years, and 67.7% were male. The mean BMI was found to be 28.95 ± 4.58 kg/m². No significant difference was found between genders in terms of BMI ($t = -1.53$, $p = 0.150$). A significant positive correlation was found between CRP and ferritin ($r = 0.62$, $p < 0.001$). Similarly, a significant positive correlation was found between CRP and sedimentation rate ($r = 0.58$, $p < 0.001$). A negative correlation was found between albumin levels and CRP ($r = -0.55$, $p < 0.01$), while a negative relationship was also observed between albumin and ferritin ($r = -0.49$, $p < 0.01$).

Conclusion: Robust relationships exist among inflammatory markers in diabetic patients. When evaluated together, increased inflammation combined with decreased albumin levels may serve as an indicator of chronic inflammation and poor metabolic control.
Keywords: diabetic foot, ferritin, crp

INTRODUCTION

Diabetes mellitus is a systemic metabolic disease characterized by chronic inflammation. Oxidative stress and inflammatory processes secondary to hyperglycemia form the basis of both microvascular and macrovascular complications. In recent years, inflammatory markers such as CRP, ferritin, and sedimentation rate have been shown to play a critical role in predicting diabetic complications (1-3). Albumin, acting as a negative acute-phase reactant, decreases in the presence of inflammation and provides insight into both nutritional status and systemic inflammation. Therefore, evaluating the relationship between inflammatory markers and albumin is of substantial clinical significance (4).

OBJECTIVE

The primary objective of this study is to investigate the relationship between inflammatory markers (C-reactive protein [CRP], ferritin, and sedimentation rate) and albumin levels—a negative acute-phase reactant—in patients diagnosed with diabetes mellitus. Additionally, we aimed to elucidate the correlations among these parameters to determine the impact of inflammation on metabolic status and to evaluate their clinical prognostic value.

MATERIALS AND METHODS

This retrospective, descriptive study included 31 patients with a confirmed diagnosis of diabetes. Patients' age, gender, height, weight, and laboratory parameters (CRP, ferritin, sedimentation rate, albumin) were recorded. Statistical analyses were performed using SPSS v27 software. Continuous variables were expressed as mean ± standard deviation. Normal distribution was assessed using the Kolmogorov-Smirnov test. For data demonstrating a non-normal distribution, Spearman correlation analysis was applied. Independent samples t-test was utilized for intergroup comparisons. A p-value < 0.05 was considered statistically significant.

RESULTS

The mean age of the 31 patients included in the study was found to be 65.45 ± 9.97 years. 67.7% of the patients were male and 32.3% were female. The mean BMI was 28.95 ± 4.58 kg/m², and the study group was generally found to be in the overweight/obese category.

In the comparison of BMI between genders, the mean BMI was found to be 28.3 ± 4.3 kg/m² in men and 30.2 ± 5.0 kg/m² in women. However, this difference was not statistically significant ($t = -1.53$, $p = 0.150$).

In the correlation analysis, a moderate-to-high positive correlation was found between CRP and ferritin ($r = 0.62$, $p < 0.001$). Similarly, a significant positive relationship was found between CRP and sedimentation rate ($r = 0.58$, $p < 0.001$). A negative correlation was observed between albumin and CRP ($r = -0.55$, $p < 0.01$), while a negative relationship was also found between albumin and ferritin ($r = -0.49$, $p < 0.01$).

DISCUSSION

In this study, the relationship between inflammatory markers and metabolic parameters in diabetic patients was examined. Literature reports indicate that CRP and ferritin elevate concurrently during inflammatory processes, carrying significant prognostic value, particularly in chronic diseases (5). The positive correlation identified among CRP, ferritin, and sedimentation rate demonstrates that these parameters effectively reflect systemic inflammation.

Furthermore, a negative correlation was established between albumin and inflammatory markers. This phenomenon can be explained by the suppression of hepatic albumin synthesis and the acceleration of catabolism during active inflammation (6,7). Low albumin levels in

diabetic patients are closely linked to poor nutritional status and an escalated inflammatory burden.

The elevated BMI values observed in our study support the established impact of obesity on inflammation. Obesity induces chronic low-grade inflammation mediated by adipokines and pro-inflammatory cytokines (8). This state may directly contribute to the elevation of markers such as CRP and ferritin. When these findings are evaluated collectively, it is evident that inflammation in diabetic patients is interrelated not only with metabolic control but also with nutritional status and the systemic response. In particular, the coexistence of elevated CRP and ferritin with suppressed albumin levels may serve as a clinical indicator of poor prognosis.

CONCLUSION

In conclusion, significant relationships exist among inflammatory markers in diabetic patients. While the concurrent evaluation of CRP, ferritin, and sedimentation rate provides crucial insights into the overall inflammatory burden, albumin levels successfully reflect the negative acute-phase response of this process. The combined clinical utilization of these parameters may offer valuable utility in determining the risk of complications in patients with diabetes.

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D-Dimer/Albumin Ratio for Predicting Clinical Outcomes in Acute Pancreatitis: A Retrospective Study

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ABSTRACT

Introduction: Acute pancreatitis is an inflammatory disease that presents with a broad clinical spectrum, ranging from mild cases to severe forms complicated by organ failure. The occurrence of severe pancreatitis in approximately 15–20% of cases highlights the importance of early identification of high-risk patients. For this purpose, there remains a need for practical, easily applicable, and reliable biomarkers. D-dimer is a marker reflecting activation of coagulation and fibrinolysis and is associated with systemic inflammation, microvascular thrombosis, and poor prognosis. Albumin is a negative acute-phase reactant that provides information about inflammation, nutritional status, and intravascular volume. However, disturbances in fluid balance, such as dehydration and hemoconcentration, which are common in acute pancreatitis, may affect serum albumin levels; therefore, caution is required in the clinical interpretation of this parameter. In recent years, combined biomarkers reflecting different pathophysiological processes have gained attention, and the D-dimer/albumin (D/A) ratio has emerged as a noteworthy parameter in this context. In this study, it was aimed to evaluate the prognostic value of D-dimer, albumin, and the D/A ratio in terms of disease severity, intensive care requirement, and mortality in patients with acute pancreatitis.

Method: In this retrospective study, patients diagnosed with acute pancreatitis were included. The prognostic performance of D-dimer, albumin, and the D/A ratio was evaluated in terms of disease severity, intensive care requirement, and in-hospital mortality. Discriminative ability was analyzed using ROC analysis; AUC, sensitivity, specificity, and optimal cut-off values were calculated.

Results: A total of 220 patients were included in the study. Of the patients, 104 (47.3%) were male and 116 (52.7%) were female, and the mean age was 57.97 ± 14.28 years. Disease severity was observed in 67 cases (30.5%), intensive care requirement in 24 cases (10.9%), and mortality in 22 cases (10.0%). In general, the discriminative performance of all biomarkers was found to be limited, with AUC values ranging from 0.428 to 0.682. The highest performance in predicting intensive care requirement was obtained with the D/A ratio (AUC: 0.682). In multivariable analysis, D-dimer and the D/A ratio were identified as independent predictors of disease severity, intensive care requirement, and mortality. While the best biomarker in terms of disease severity and intensive care requirement was the D/A ratio, the highest performance in predicting mortality was observed with D-dimer. In contrast, albumin was not identified as an independent predictor in any model.

Discussion: In our study, although D-dimer and the D/A ratio were statistically significant predictors in regression analyses, their overall discriminative performance was found to be limited. The fact that the AUC value did not exceed 0.70 for any of the evaluated biomarkers indicates that the clinical use of these parameters alone is limited. The D/A ratio demonstrated the best overall performance among the evaluated parameters. This superiority is particularly evident in the prediction of intensive care admission. D-dimer reflects coagulation activation. Albumin provides information regarding inflammation and dehydration status. Considering these two markers together may better reflect the multidimensional pathophysiology of acute pancreatitis compared to individual measurements. D-dimer was found to be associated with poor prognosis. This may be explained by increased coagulation tendency and pronounced inflammation in severe pancreatitis. These processes may lead to microvascular thrombosis. As a result, organ perfusion may be impaired and organ dysfunction may develop. In contrast, albumin did not demonstrate significant prognostic value in either ROC analysis or regression models. Another important finding is that D-dimer and the D/A ratio have high specificity. In contrast, sensitivity is low. Therefore, these markers do not appear to be very suitable as screening tests. However, they may be more useful in supporting and confirming high-risk patients. In other words, they may be considered as markers that strengthen the diagnosis rather than exclude it.

Conclusion: The low AUC values in regression analyses indicate that these biomarkers should not be used alone. The D/A ratio, which stands out among the others, may be integrated into existing scoring systems such as BISAP, Ranson, and APACHE-II in future studies.

Keywords: Acute Pancreatitis; D-dimer; Albumin; D-dimer/Albumin Ratio; Prognosis

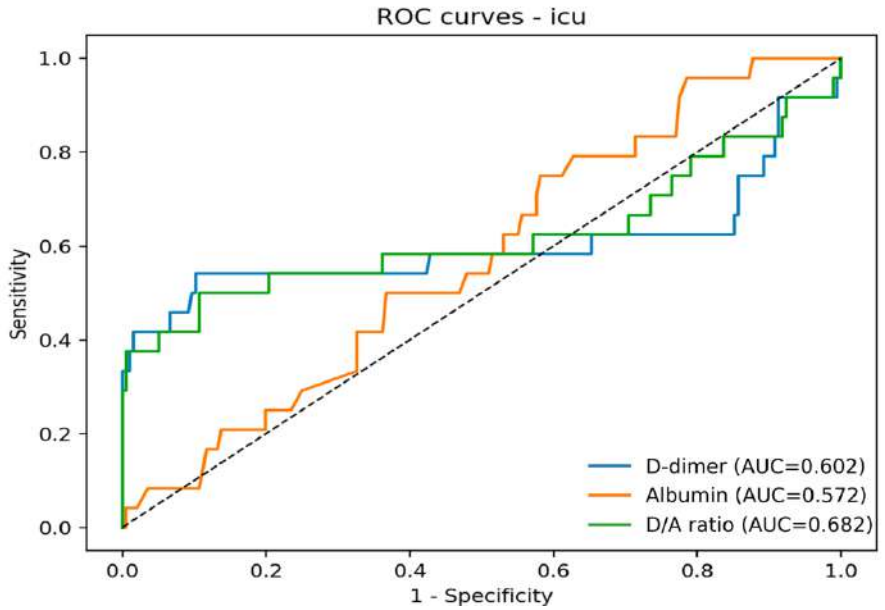

Figure 1: ROC curves of D-dimer, albumin, and D/A ratio for the prediction of intensive care unit requirement.

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Cerebral Hydatid Cyst: A Case-Based Review

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Keywords: cerebral hydatid cyst; cystic echinococcosis; Echinococcus granulosus; intracranial cyst; emergency medicine

Hydatid Cyst Disease

Hydatid cyst disease, also known as cystic echinococcosis, is an endemic zoonotic parasitic infection caused by Echinococcus granulosus. It remains an important public health problem in many regions of the world, particularly in Mediterranean countries [1,2]. The disease is encountered more frequently in communities engaged in livestock farming. Humans are usually infected through contact with dogs or through ingestion of parasite eggs contaminating food, water, or soil, and the disease may produce cystic lesions in various organs.

General Characteristics of the Parasite

Hydatid cyst disease is caused by the larval stage of the cestode Echinococcus granulosus (EG) [1,3]. Transmission to humans generally occurs by the fecal-oral route. Direct contact with infected animals, particularly dogs, or ingestion of contaminated food, water, or soil may lead to infection [1,4]. The adult tapeworm of EG lives in the intestine of the definitive host, most commonly dogs and other carnivores, and releases eggs into the environment through feces [3]. When herbivorous intermediate hosts such as sheep, goats, or cattle ingest these eggs, the larvae hatch, penetrate the intestinal wall, enter the portal circulation, and are transported to different organs. Humans act as accidental intermediate hosts; after larval dissemination, hydatid cysts may develop in multiple organs [1-3].

Dissemination and Pathogenesis

After ingestion, embryonated eggs pass through the intestinal wall and enter the portal circulation. Approximately 75% of embryos lodge in the liver, while 15-20% reach the lungs [3]. Although less common, metastatic dissemination may also involve the brain, spleen, kidney, muscle, bone, and other organs [5-7]. Cerebral hydatid cyst is a rare form of the disease, but it may cause severe neurological manifestations.

Cerebral Hydatid Cyst

Cerebral hydatid cyst develops when larval cysts of Echinococcus granulosus become established in the brain. These cysts generally enlarge slowly. As the cyst grows, it disrupts normal cerebral function and may produce various clinical symptoms [5,8].

Cerebral hydatid cyst is reported more commonly among individuals living in rural areas and those involved in animal husbandry. Although the disease can occur worldwide, it is more prevalent in regions such as the Middle East, South America, and Eastern Europe. Several cases of cerebral hydatid cyst have also been reported in Türkiye [6-16].

Intracranial hydatid cysts are most often located in the supratentorial compartment, particularly within the cerebral hemispheres [6,8]. They may cause increased intracranial pressure, headache, vomiting, seizures, and focal neurological deficits. Cerebral involvement is more frequent in childhood than in adulthood, partly because the blood-brain barrier in children is more permeable, facilitating access of the parasite to brain tissue [7,8].

Clinical Findings and Diagnosis

The clinical manifestations of hydatid cysts depend on the size and anatomical location of the cyst. Although the liver and lungs are the most commonly affected organs, cerebral involvement may present with serious neurological findings [5-11]. Symptoms observed in cerebral hydatid cyst disease may include:

- Headache
- Nausea and vomiting
- Epileptic seizures
- Visual disturbances
- Motor and sensory deficits
- Altered mental status

The diagnosis is established by imaging in the appropriate clinical context. Magnetic resonance imaging (MRI) and computed tomography (CT) are the most important diagnostic modalities for cerebral hydatid cysts [5]. MRI provides superior delineation of the internal structure of the cyst and its relationship with adjacent brain tissue, whereas CT may

demonstrate calcifications and intralesional calcified components more clearly [5-11]. Serological tests, particularly enzyme-linked immunosorbent assay (ELISA) and Western blot, may support the diagnosis; however, their sensitivity may be limited in cases with cerebral involvement. Definitive diagnosis is usually based on histopathological examination of cyst material obtained during surgery [1,3,4].

Treatment and Management

Surgery is the most effective treatment for cerebral hydatid cyst. The surgical objective is complete removal of the cyst without disrupting its integrity [14]. If the cyst ruptures, there is a risk of anaphylactic reaction and secondary dissemination [11]. Therefore, specialized surgical techniques, such as the Dowling technique, should be used to ensure safe removal of the cyst [8,13].

Pharmacological treatment may be used as an adjunct to surgery or as the primary strategy in patients who are not surgical candidates. Albendazole is the principal antiparasitic agent used to inhibit parasite growth and reduce cyst size. It is generally administered for 3-6 months and is also recommended postoperatively to decrease the risk of recurrence [4-11].

Case Examples

Case 1

A 6-year-old refugee was brought to the emergency department by emergency medical services because of headache and altered consciousness. On systemic examination, the patient was in good general condition, conscious, and had a Glasgow Coma Scale score of 13. Heart rate and blood pressure were appropriate for age. No cranial nerve involvement was detected; cerebellar examination was normal, and lung sounds were normal. Contrast-enhanced cranial CT demonstrated a well-defined, non-enhancing lesion measuring 11 x 10 x 9 cm, occupying the right frontotemporoparieto-occipital region, producing a marked midline shift, and compressing and obliterating the lateral ventricle (Figures 1a-b). Abdominal CT revealed a 7-cm well-defined hypodense cystic lesion in segment 2 of the left hepatic lobe, compatible with a type 1 hydatid cyst without septations, and a 5.5 x 3.5 cm multiseptated lesion in segment 3, compatible with a type 3 hydatid cyst (Figure 1c). In addition, opacity was observed on chest radiography. Thoracic CT showed a lesion compatible with hydatid cyst in the lower lobe of the left lung, containing membranous structures and demonstrating a water-lily sign (Figures 1d-e). The patient was subsequently referred to the neurosurgery department. The cerebral cyst was surgically removed intact by a neurosurgeon (Figure 1f).

Case 2

A 25-year-old pregnant Syrian patient was brought to the emergency department after being trapped under debris, presenting with altered consciousness and headache. On initial evaluation, her vital signs were stable (arterial blood pressure 110/50 mmHg, heart rate 95 beats/min, and SpO2 95%), and there were no signs of external bleeding. Cranial CT was performed using a fetal protective apron. A giant lesion in the right frontal region, potentially compatible with a hydatid cyst, was identified (Figures 2a-b). The diagnosis of hydatid cyst was supported by the clinical laboratory, and the patient was referred to the neurosurgery department for treatment. The cerebral cyst was surgically removed intact by a neurosurgeon (Figure 2c).

Examples From the Literature

Cerebral hydatid cysts are rare and account for approximately 1% of all hydatid cyst cases. Cerebral hydatid cyst may be primary or secondary to hepatic hydatid disease and is usually solitary. It is more common in children and young adults. Although it can occur anywhere in the brain, it is typically supratentorial, often located in the middle cerebral artery territory, and most frequently affects the parietal lobe. Large solitary cysts may sometimes be seen in the frontoparietotemporal region. Less common sites include the cerebellum, pons, ventricles, and cavernous sinus [1-5].

Hydatid cysts developing in the brain grow slowly. Because of the elastic nature of brain tissue and the gradual increase in pressure, the brain may adapt to the slowly expanding cyst. Consequently, the cyst may reach giant dimensions exceeding 10 cm in diameter, while the patient's symptoms may remain disproportionally mild relative to cyst size. Cerebral hydatid cysts may also lead to severe complications when rupture occurs [5-9].

In patients with cerebral hydatid cyst, a wide spectrum of symptoms may develop as a consequence of increased intracranial pressure and depending on the affected brain region. The disease may manifest with increased intracranial pressure syndrome, convulsions, unilateral weakness and sensory loss, headache, nausea, vomiting, strabismus, and confusion.

Serological and radiological methods are used in the diagnosis. Cerebral hydatid cyst is diagnosed by demonstrating the cyst on CT or MRI. On CT, a cerebral hydatid cyst appears as a well-demarcated, homogeneous, intraparenchymal, round lesion. On MRI, the cyst wall can be more clearly visualized on T1-weighted images. On T2-weighted images, these cysts show signal intensity similar to fluid. Radiologically, they may be confused with arachnoid cyst, abscess, porencephalic cyst, or cystic astrocytoma. Perilesional edema, peripheral enhancement, mural nodule, and lesion morphology are important parameters in the differential diagnosis.

Treatment is selected according to symptoms and cyst size and may be medical and/or surgical. The causative agent can be identified after cyst removal [5-10].

Among case reports published from Türkiye, Karabekir et al. [6] reported a large solitary cerebral hydatid cyst in a 10-year-old girl; Kılıç et al. [7] in a 5-year-old girl; Işıkay et al. [9] in a 5-year-old girl and a 13-year-old boy; Çullu et al. [10] in a 5-year-old girl; Uzun et al. [11] in a 24-year-old woman; Teber et al. [12] in a 10-year-old girl; and Tural-Kara [13] in an 8-year-old boy. Multiple localized cerebral hydatid cysts were reported by Hastürk et al. [14] in a 32-year-old man, Tamer et al. [15] in a 36-year-old man, and Angın et al. [16] in a 34-year-old woman.

Conclusion and Key Points

Although cerebral hydatid cyst is rare, it should be considered in the differential diagnosis of patients who present with neurological symptoms and have a single, or occasionally multiple, spherical homogeneous cystic brain lesions. Early diagnosis should be pursued using laboratory and imaging methods. Hydatid cyst disease remains an important public health problem, particularly in Mediterranean countries, and may cause serious complications in humans. Cerebral hydatid cyst is an uncommon but clinically significant form characterized by potentially severe neurological manifestations. Successful outcomes can be achieved through early diagnosis and appropriate treatment. Compliance with hygiene measures and effective public health interventions can substantially reduce transmission of the disease.

Figure Legends

Figure 1. Computed tomography images of Case 1.

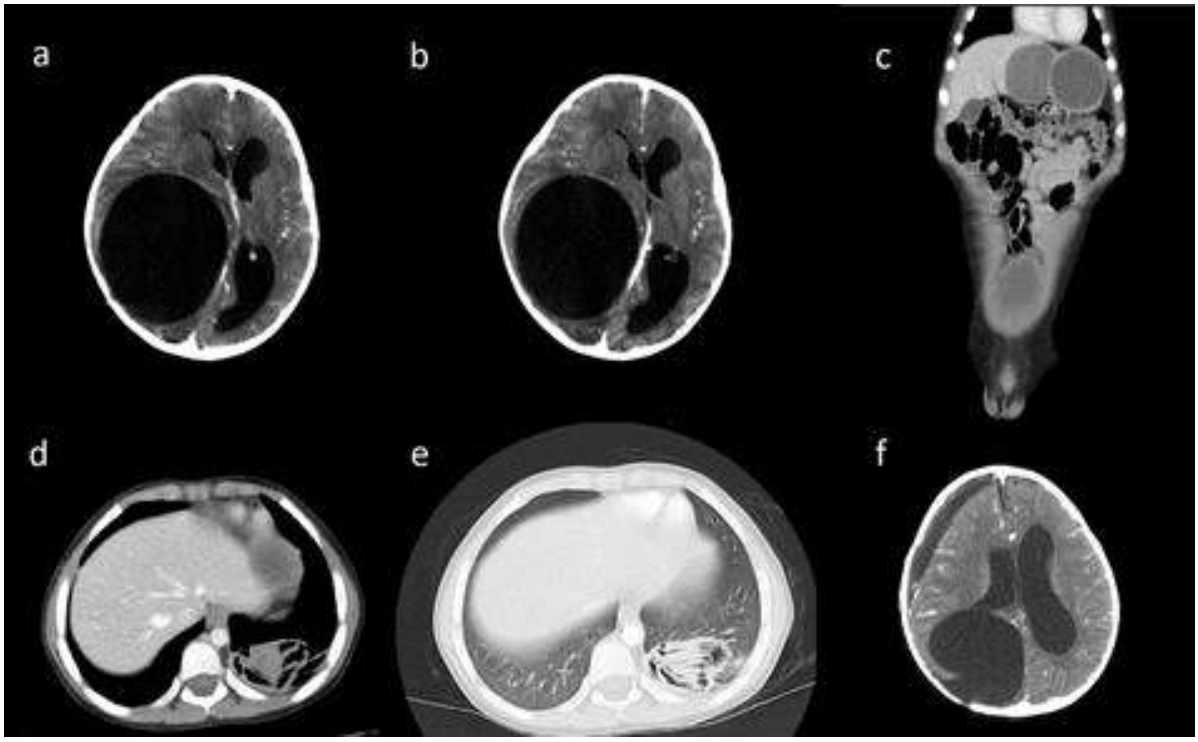
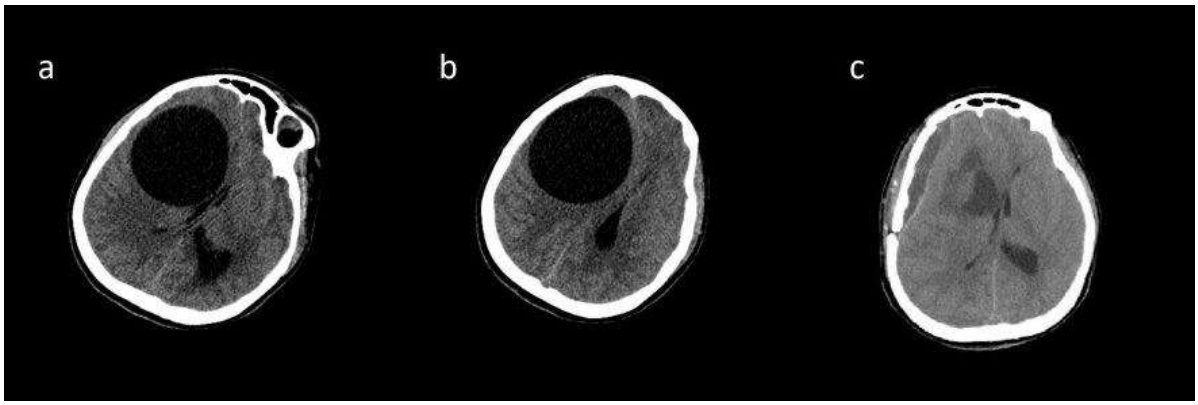


Figure 2. Computed tomography images of Case 2.



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Long-Term Trends of Climate-Related Disasters in Türkiye: An Analysis of the EM-DAT Database

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ABSTRACT

Background: Climate-related disasters pose increasing risks to public health and infrastructure globally. Türkiye, owing to its diverse topography and climate zones, is particularly susceptible to such events. This study examines long-term trends of climate-related disasters in Türkiye using the Emergency Events Database (EM-DAT).

Methods: A retrospective descriptive analysis was conducted on all climate-related disasters recorded in Türkiye in the EM-DAT database between 1948 and 2025. Data on disaster type, year of occurrence, mortality, and affected populations were extracted and analyzed using descriptive statistics.

Results: A total of 93 climate-related disasters were identified, including floods, storms, landslides, wildfires, extreme temperature events, and avalanches. Floods were the most frequent disaster type and accounted for the highest number of deaths and affected individuals. Avalanches demonstrated the highest average mortality per event. An increasing trend in disaster frequency was observed, particularly after 2000.

Conclusion: Floods represent the predominant climate-related hazard in Türkiye, while avalanches carry the highest per-event fatality risk. Strengthening flood risk management and early warning systems is essential for improving disaster resilience.

Keywords: climate-related disasters; floods; EM-DAT; disaster trends; Türkiye; disaster risk reduction

INTRODUCTİON

Climate-related disasters have become an increasingly significant global public health and environmental concern. Over recent decades, the frequency and severity of extreme weather events—including floods, storms, heatwaves, wildfires, and landslides—have risen across many regions. These events cause substantial human, economic, and environmental losses, affecting millions of people annually and placing considerable strain on healthcare systems, infrastructure, and disaster management capacities (1–3). Climate change is widely recognized as a primary driver of these trends, influencing both the occurrence and severity of hydrometeorological hazards through alterations in temperature, precipitation patterns, and atmospheric circulation (4,5).

Among climate-related hazards, floods, storms, and landslides account for a large share of recorded disasters worldwide. Floods, in particular, are among the most frequent and destructive natural hazards, often resulting in large numbers of affected individuals and significant infrastructure damage (6,7). Other hazards—such as wildfires, extreme temperature events, and avalanches—occur less frequently yet may still cause substantial human and economic losses depending on geographic and environmental conditions (2,3). Understanding the distribution and impacts of different climate-related disaster types is therefore essential for strengthening disaster preparedness, disaster risk reduction strategies, and climate adaptation policies.

The observed increase in climate-related disasters is influenced not only by climatic factors but also by growing human exposure and vulnerability. Rapid urbanization, population growth, and the expansion of settlements into hazard-prone areas have increased the number of people at risk from extreme weather events (8). Simultaneously, socioeconomic conditions, infrastructure quality, and disaster preparedness capacities strongly determine disaster outcomes. As a result, the same type of hazard may produce vastly different impacts across countries and regions depending on their level of resilience and adaptive capacity (9).

Large-scale disaster databases have become indispensable tools for evaluating long-term disaster trends. The Emergency Events Database (EM-DAT), maintained by the Centre for Research on the Epidemiology of Disasters (CRED), is one of the most widely used global

disaster databases and provides standardized information on disaster occurrence, mortality, and affected populations across countries and decades (10,11). By compiling records from national authorities, international organizations, and humanitarian agencies, EM-DAT enables consistent cross-country comparisons and long-term analyses of disaster patterns.

Türkiye is geographically and climatically diverse, and is exposed to multiple types of natural hazards. Its complex topography, varied climate zones, and rapidly growing urban population render the country vulnerable to a range of climate-related disasters, including floods, storms, landslides, wildfires, extreme temperature events, and avalanches (12). Mountainous terrain and intense precipitation events contribute to landslides and floods, while Mediterranean climatic conditions heighten the risk of large-scale wildfires during hot and dry summers.

Despite the growing importance of climate-related hazards in Türkiye, comprehensive national analyses examining their long-term patterns remain relatively limited. Most previous studies have focused on individual disaster types or regional case studies rather than providing a nationwide assessment of multiple climate-related hazards. A systematic evaluation of distribution, mortality patterns, and temporal trends across disaster types may therefore provide valuable insights for disaster risk management and climate adaptation.

The present study aimed to analyze all climate-related disasters recorded in Türkiye in the EM-DAT database between 1948 and 2025. Specifically, we evaluated the distribution of disaster types, mortality patterns, average deaths per event, affected populations, and temporal trends in disaster occurrence. By providing a long-term nationwide overview, this study aims to contribute to a better understanding of disaster risk and to support evidence-based disaster preparedness and climate adaptation policies.

METHODS

Data Source

All data were obtained from the Emergency Events Database (EM-DAT), maintained by CRED at the Université Catholique de Louvain, Belgium. EM-DAT provides standardized information on natural disasters worldwide, including disaster type, year of occurrence,

country, number of deaths, and number of affected individuals. The database compiles records from multiple sources, including United Nations agencies, national governments, research institutions, and humanitarian organizations.

An event is included in EM-DAT if at least one of the following criteria is met: (1) ten or more people reported killed; (2) one hundred or more people reported affected; (3) declaration of a state of emergency; or (4) a call for international assistance.

Study Design and Data Extraction

This study employed a retrospective descriptive design. All disasters categorized as climate-related events in Türkiye in the EM-DAT database between 1948 and 2025 were included. For each event, data on disaster type, year of occurrence, number of deaths, and number of affected individuals were extracted. Climate-related disasters were classified into six categories: floods, storms, landslides, wildfires, extreme temperature events, and avalanches.

Outcome Measures

The primary objective was to describe the distribution and impact of climate-related disasters in Türkiye. Outcome measures included: the distribution of disaster types; total deaths by disaster type; average deaths per disaster event; total affected individuals by disaster type; and temporal trends in disaster frequency.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics (version 29.0; IBM Corp., Armonk, NY, USA). Descriptive statistics were used to characterize the distribution and impact of climate-related disasters. The frequency and proportional distribution of disaster types were calculated, and total deaths and total affected individuals were summed for each disaster category. Average deaths per event were computed by dividing the total number of deaths by the number of recorded events for each disaster type. Temporal trends in annual disaster frequency were evaluated over the study period by examining the annual count of

recorded events. Graphical visualizations were generated to illustrate distributional patterns and temporal trends across disaster categories.

RESULTS

A total of 93 climate-related disasters were recorded in Türkiye between 1948 and 2025 according to the EM-DAT database. These events included floods, storms, landslides, wildfires, extreme temperature events, and avalanches, and resulted in substantial human impact in terms of mortality and affected populations.

Floods constituted the most frequently recorded disaster type, accounting for the largest proportion of all events. Storms were the second most common, followed by landslides. Extreme temperature events and wildfires were reported less frequently, while avalanches represented the least common category in the dataset (Figure 1).

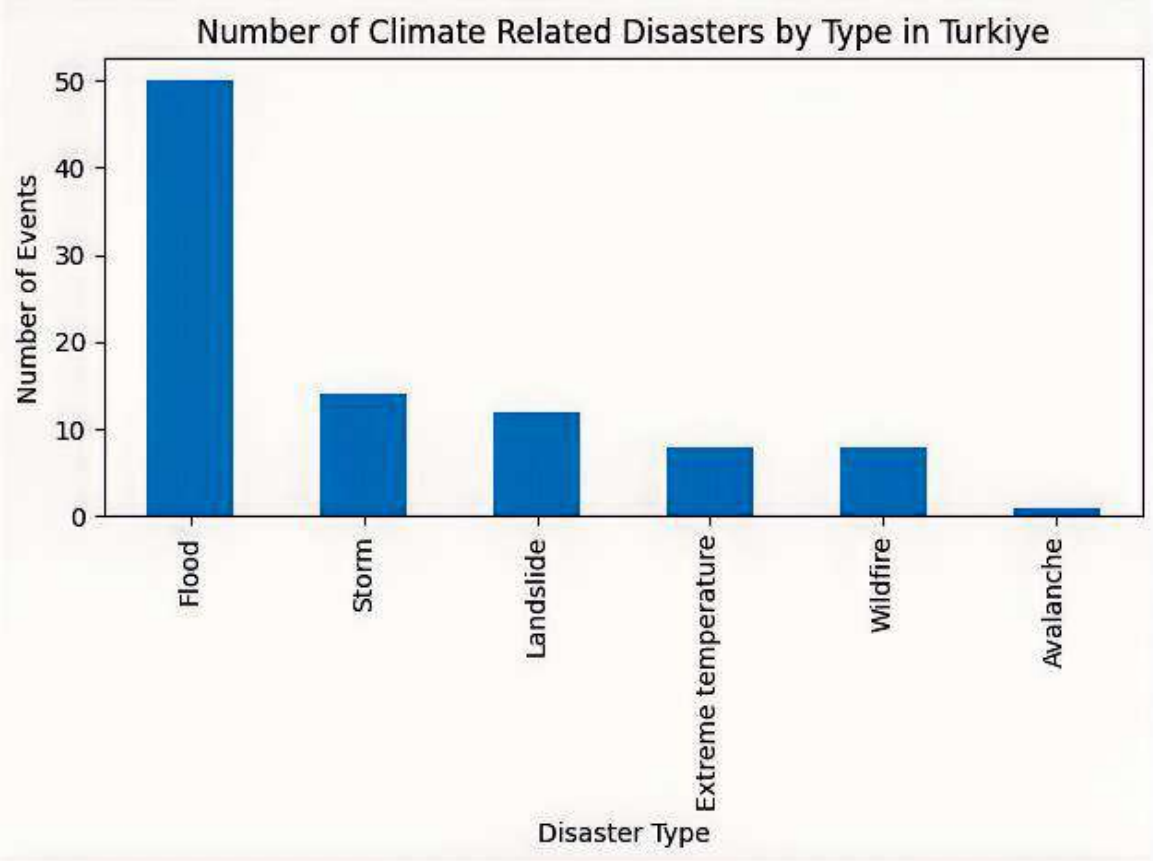

Figure 1. Distribution of climate-related disasters by type in Türkiye.

In terms of mortality, floods were responsible for the highest number of deaths. Landslides and avalanches were also associated with considerable mortality. Conversely, storms, extreme temperature events, and wildfires resulted in comparatively fewer deaths (Figure 2).

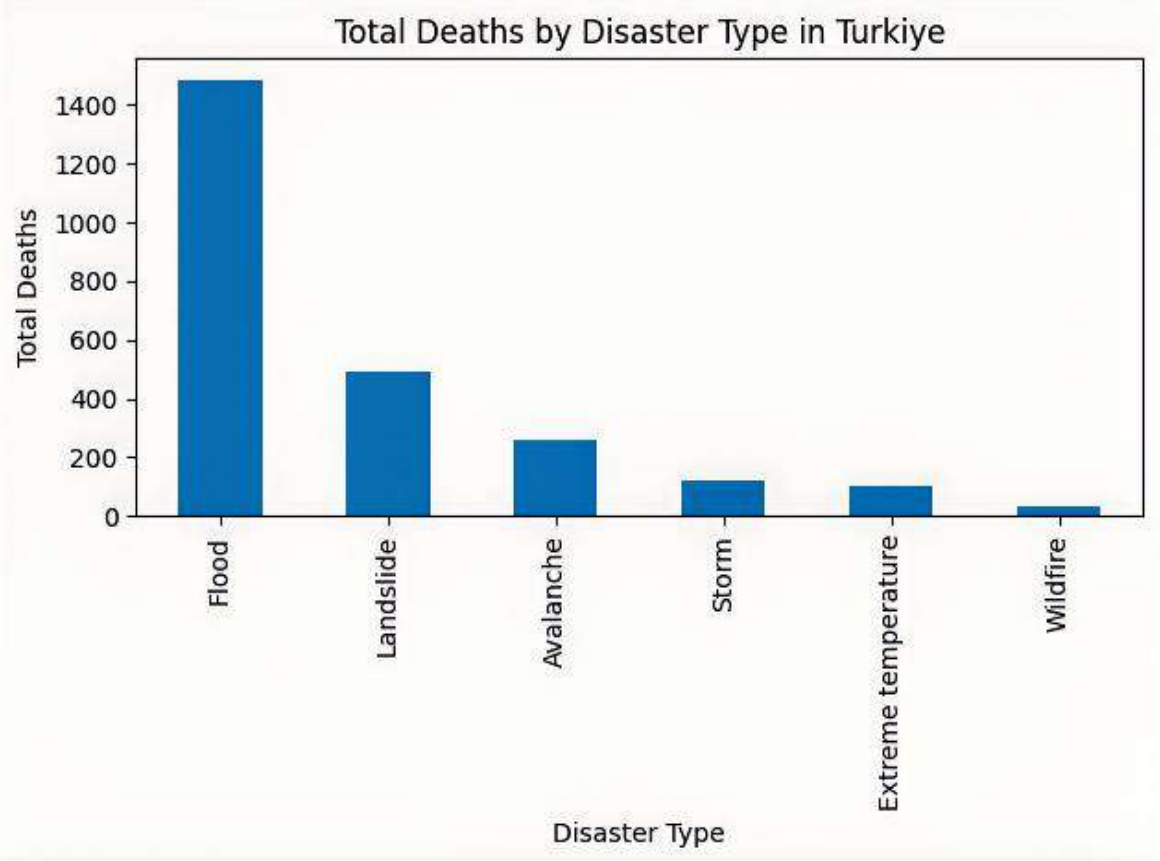

Figure 2. Total deaths by disaster type in Türkiye.

When mortality was assessed on a per-event basis, avalanches demonstrated the highest average number of deaths per disaster. Although avalanches occurred infrequently, they were associated with substantial fatality rates per event. Landslides and floods also exhibited comparatively high per-event mortality, whereas storms, extreme temperature events, and wildfires showed lower average fatality rates (Figure 3).

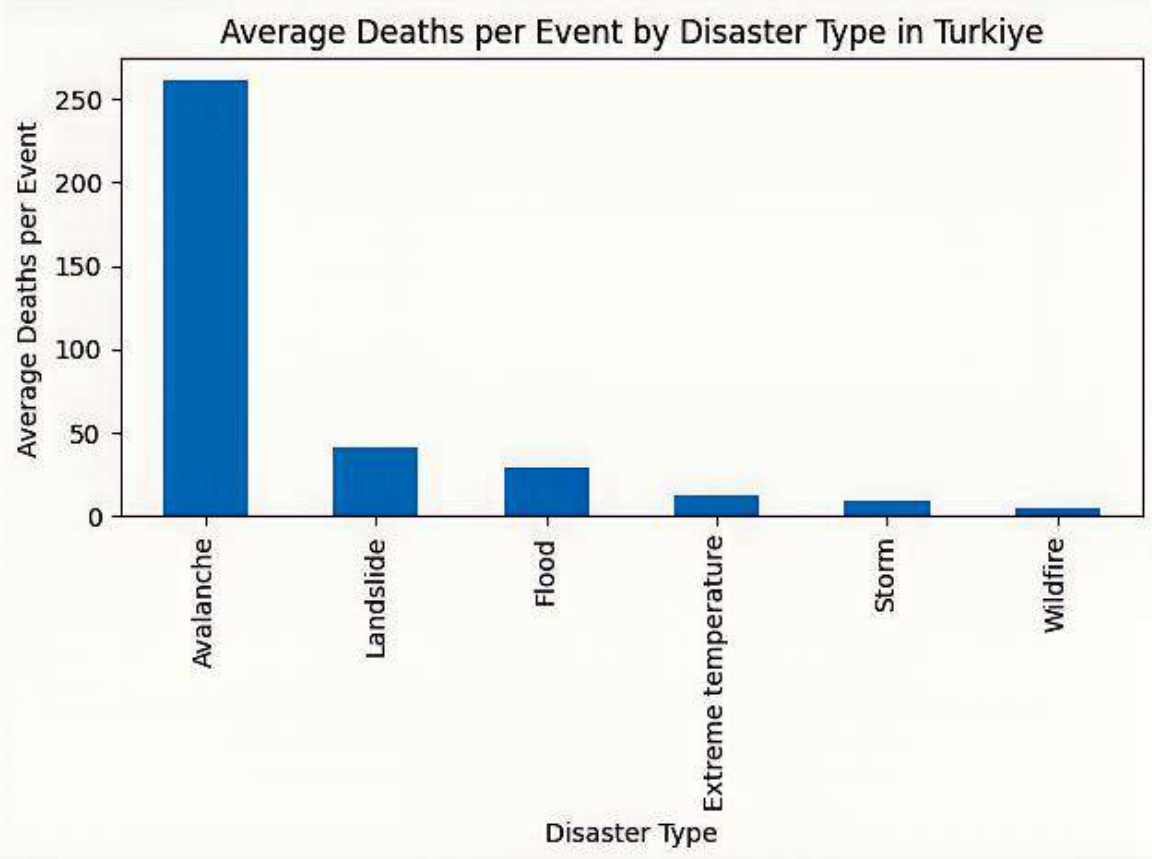

Figure 3. Average deaths per event by disaster type in Türkiye.

Analysis of the affected population revealed that floods impacted the largest number of individuals. Wildfires represented the second most impactful disaster type in terms of affected population. Other categories—storms, landslides, extreme temperature events, and avalanches—affected comparatively smaller populations (Figure 4).

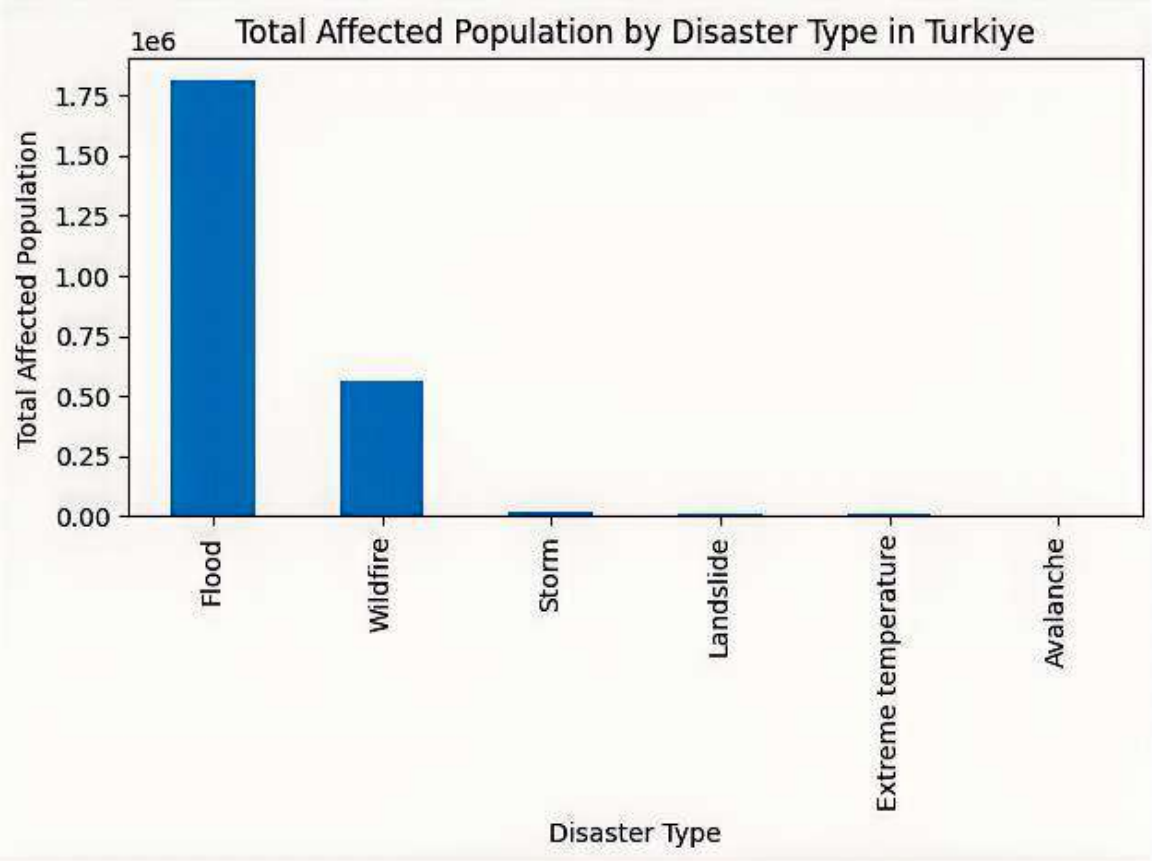

Figure 4. Total affected population by disaster type in Türkiye.

Temporal analysis demonstrated an increasing trend in the annual number of climate-related disasters in Türkiye over the study period. Although year-to-year fluctuations were observed, overall disaster frequency increased over time, with a particularly notable rise after the early 2000s (Figure 5).

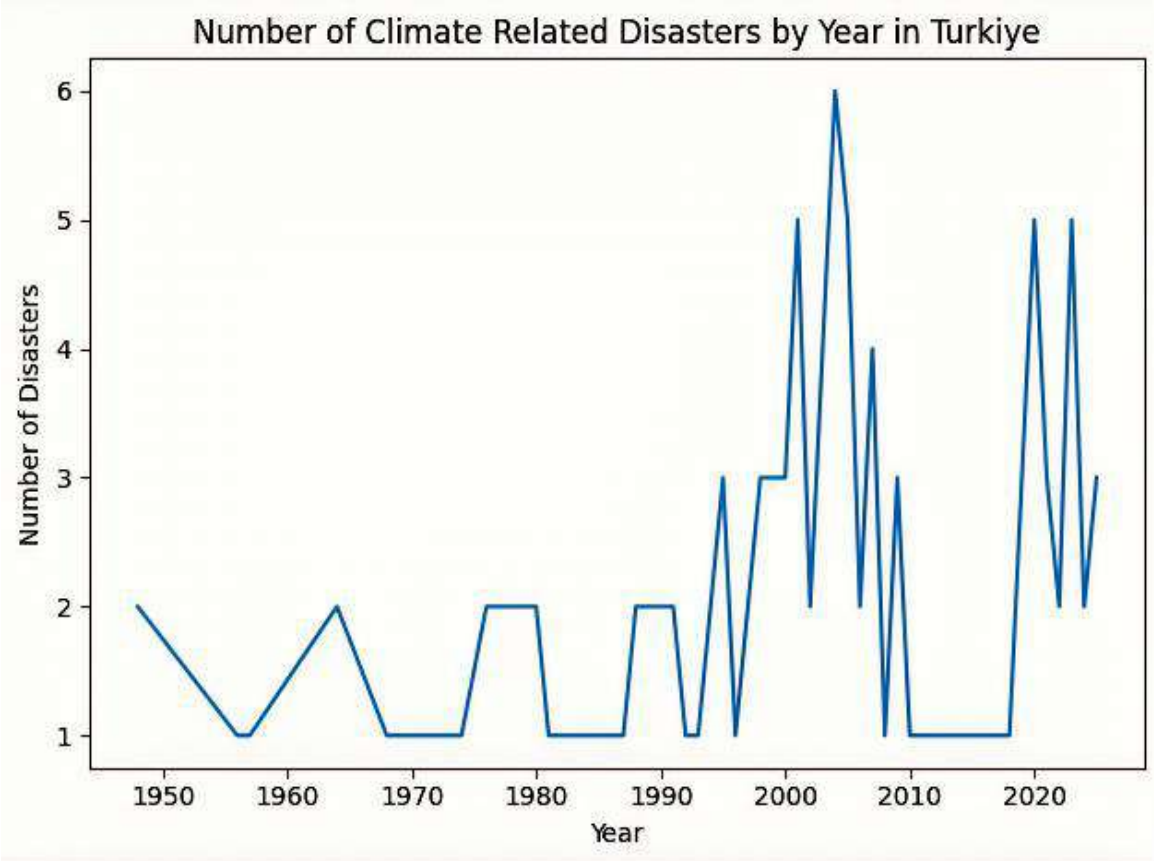

Figure 5. Annual number of climate-related disasters in Türkiye, 1948–2025.

DİSCUSSİON

This study provides a comprehensive long-term overview of climate-related disasters in Türkiye based on EM-DAT data spanning 1948 to 2025. A total of 93 events were identified, encompassing floods, storms, landslides, wildfires, extreme temperature events, and avalanches. Several key findings emerged. Floods were the most frequent disaster type and accounted for the highest total mortality and the largest number of affected individuals. Avalanches demonstrated the highest per-event fatality rate despite their relatively low frequency. A marked temporal increase in disaster frequency was observed, particularly after 2000.

The predominance of floods is consistent with global disaster statistics. Floods represent one of the most frequently recorded natural disasters worldwide and account for a disproportionately large share of affected populations (2,3,6). Global assessments have

consistently shown that hydrometeorological hazards—particularly floods and storms—dominate the disaster landscape across many regions, a pattern further supported by analyses linking climate anomalies to recorded disaster events (13,14). Because floods typically affect large geographic areas including densely populated zones, they frequently result in high levels of human exposure and infrastructure damage (7).

The high per-event mortality observed for avalanche disasters is a notable finding. Although avalanches are infrequent, their sudden onset and rapid destructive force leave little opportunity for evacuation, contributing to elevated fatality rates (15,16). Similar findings have been reported in studies of mountain hazards, where the combination of limited warning time and high energy release produces severe outcomes (15).

The finding that floods affected the largest number of individuals reflects their extensive spatial impact, often disrupting housing, transportation, and agricultural areas across wide regions. Previous research has similarly shown that floods tend to affect significantly larger populations than most other natural hazards (7). Wildfires ranked second in terms of affected population, a finding consistent with the heightened wildfire risk in Mediterranean ecosystems driven by hot, dry summers, prolonged drought, and land-use changes (17,18).

The increasing temporal trend in climate-related disasters documented in this study aligns with patterns reported globally over recent decades (3,13). Climate change is widely recognized as a key driver of this trend, influencing the frequency and intensity of heavy precipitation, storms, and heatwaves (1,4,5). However, improvements in disaster monitoring systems and reporting mechanisms may also contribute to the growing number of documented events (11). Additionally, rapid urbanization and population growth have placed more people in hazard-prone areas such as floodplains and wildfire-prone landscapes, amplifying potential societal impacts (8,19).

Implications for Disaster Risk Management

These findings underscore the importance of strengthening disaster risk reduction and climate adaptation strategies in Türkiye. Given the predominance of floods, improving flood risk management should remain a policy priority. Investments in early warning systems,

floodplain management, and resilient infrastructure could meaningfully reduce flood-related losses. The high per-event mortality associated with avalanches highlights the need for improved hazard mapping, risk-informed land-use planning, and public awareness programs in mountainous regions. The increasing trend in disaster frequency further reinforces the urgency of developing long-term, evidence-based climate adaptation frameworks.

Limitations

Several limitations should be acknowledged. EM-DAT relies on secondary data sources, and disaster reporting coverage may vary across time periods and geographic regions. Events occurring in earlier decades are likely underreported, which may affect long-term trend analyses. Furthermore, the database does not include detailed meteorological variables—such as precipitation intensity or temperature anomalies—which limits more granular hazard analysis. Future studies incorporating meteorological data and regional-level analyses would complement the national overview provided here.

CONCLUSİON

This study provides a comprehensive overview of climate-related disasters recorded in Türkiye between 1948 and 2025 using EM-DAT data. Floods emerged as the most frequent and impactful disaster type, accounting for the largest proportions of events, deaths, and affected populations. Avalanches, though rare, were associated with the highest per-event mortality. An increasing trend in disaster frequency was observed, particularly after 2000, underscoring the growing burden of climate-related hazards.

These findings highlight the critical importance of disaster risk reduction and climate adaptation in Türkiye. Strengthening flood risk management, expanding early warning systems, and enhancing preparedness measures across disaster types are essential steps toward reducing the human and societal costs of climate-related hazards. Long-term analyses of standardized disaster databases such as EM-DAT can provide policymakers and disaster management authorities with the evidence base needed to develop effective, forward-looking resilience strategies.

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5941

Septic Embolic Presentation of Enterococcus faecalis Endocarditis in a Hemodialysis Patient

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Introduction

Infective endocarditis (IE) is an uncommon but life-threatening clinical entity characterized by microbial infection of the cardiac valves, mural endocardium, or intracardiac prosthetic material. Although diagnostic imaging, microbiological methods, antimicrobial therapy, and surgical strategies have substantially improved, IE continues to cause severe complications, including sepsis, systemic or pulmonary embolization, heart failure, valvular destruction, and multiorgan dysfunction(1-3). Contemporary series report overall mortality rates of approximately 20–25%, with even higher rates among elderly patients, individuals with major comorbidities, and those with healthcare-associated infection(1, 3, 4).

The epidemiological profile of IE has changed considerably over recent decades. Whereas rheumatic valvular disease and community-acquired streptococcal infection were historically dominant predisposing factors, current cases are increasingly associated with advanced age, degenerative valvular disease, prosthetic valves, cardiac implantable electronic devices, invasive procedures, intravascular catheters, and healthcare-associated bacteremia(1-3). This transition has also altered the microbiological spectrum of IE. *Staphylococcus aureus*, coagulase-negative staphylococci, streptococci, and enterococci are now among the most frequently identified pathogens in contemporary cohorts (1, 2, 5). Because IE may initially present with nonspecific findings such as fever, malaise, new or changing murmur, embolic manifestations, sepsis, or altered mental status, early diagnosis remains challenging, especially in patients with complex comorbid conditions (1, 3).

Patients undergoing chronic hemodialysis constitute a particularly vulnerable subgroup. The incidence of IE in this population is markedly higher than in the general population, largely because of repeated vascular access, central venous catheter use, uremia-related immune dysfunction, endothelial injury, frequent healthcare exposure, and recurrent antibiotic use(4, 6, 7). IE in dialysis patients is clinically important not only because it occurs more frequently, but also because it is associated with a more severe course and poorer outcomes. In this group, staphylococcal pathogens, particularly *S. aureus*, remain predominant; however, enterococci are increasingly recognized, especially among elderly, comorbid, and healthcare-exposed patients(4, 6, 8). Central venous catheters represent a critical risk factor for both

bloodstream infection and subsequent endocardial involvement(6, 7). Therefore, in dialysis patients with unexplained or recurrent bacteremia—particularly *S. aureus* bacteremia—or with otherwise unexplained neurovascular embolic events, IE should be systematically considered and actively investigated.

Case

A 71-year-old male patient was brought to the emergency department by emergency medical services after a blood pressure of 80/50 mmHg was measured at home and he experienced a brief episode of loss of consciousness. He had been receiving hemodialysis twice weekly for end-stage renal disease for 2.5 years; however, his dialysis frequency had been increased to three sessions per week two weeks before admission. According to his relatives, he had recently developed intermittent confusion and difficulty recognizing people around him.

During the preceding week, he had experienced fatigue, gait disturbance, cough, and febrile episodes reaching 38 °C. His medical history included hypertension, chronic kidney disease, hyperparathyroidism, osteoporosis, and benign prostatic hyperplasia. His regular medications included calcium sulfonate, phosphate binders, calcium acetate, antihypertensive agents, potassium-lowering therapy, bicarbonate therapy, folic acid, vitamin supplementation, and treatment for hyperparathyroidism.

On physical examination, his general condition was moderate. He was conscious, cooperative, and oriented at the time of evaluation. His blood pressure was 80/50 mmHg, heart rate was 90 beats/min, oxygen saturation was 96% on room air, and body temperature was 38.2 °C. Cardiac auscultation did not reveal a clearly audible new murmur, and lung sounds were normal. He had a right subclavian hemodialysis catheter, and no erythema, discharge, tenderness, or other local skin changes were observed around the catheter site. Electrocardiography showed normal sinus rhythm without acute ischemic changes.

Laboratory testing revealed an elevated C-reactive protein level of 26.99 mg/L, creatinine of 6.55 mg/dL, blood urea nitrogen of 144 mg/dL, potassium of 5.38 mmol/L, and high-sensitivity troponin of 202.3 ng/L. Urinalysis was unremarkable. Blood cultures subsequently yielded *Enterococcus faecalis*, whereas urine culture showed no bacterial growth.

Thoracic computed tomography did not demonstrate a definite pneumonic focus. Because of transient loss of consciousness, gait disturbance, and recent cognitive changes, cranial computed tomography and diffusion-weighted magnetic resonance imaging were obtained to exclude central nervous system pathology. Diffusion-weighted imaging demonstrated multiple lacunar areas of restricted diffusion on

several sections, raising suspicion for embolic lesions, including possible septic emboli.

Bedside transthoracic echocardiography showed preserved left ventricular systolic function, with a left ventricular ejection fraction of approximately 60%. Additional findings included left ventricular hypertrophy, mild mitral regurgitation, calcific changes of the mitral and aortic valves, and mild tricuspid regurgitation. A mobile mass measuring 25.3 × 12.2 mm, compatible with vegetation, was visualized on the tricuspid valve.

Based on the presence of *E. faecalis* bacteremia, a large mobile tricuspid valve vegetation, fever, and neuroimaging findings suggestive of embolic involvement, the patient was diagnosed with right-sided infective endocarditis involving the tricuspid valve. The most probable source was considered to be the existing hemodialysis catheter. The infectious diseases department was consulted, and intravenous ceftriaxone plus vancomycin therapy was initiated. Serial blood culture monitoring and replacement of the hemodialysis catheter were recommended, and the patient was admitted to the infectious diseases ward for further management.

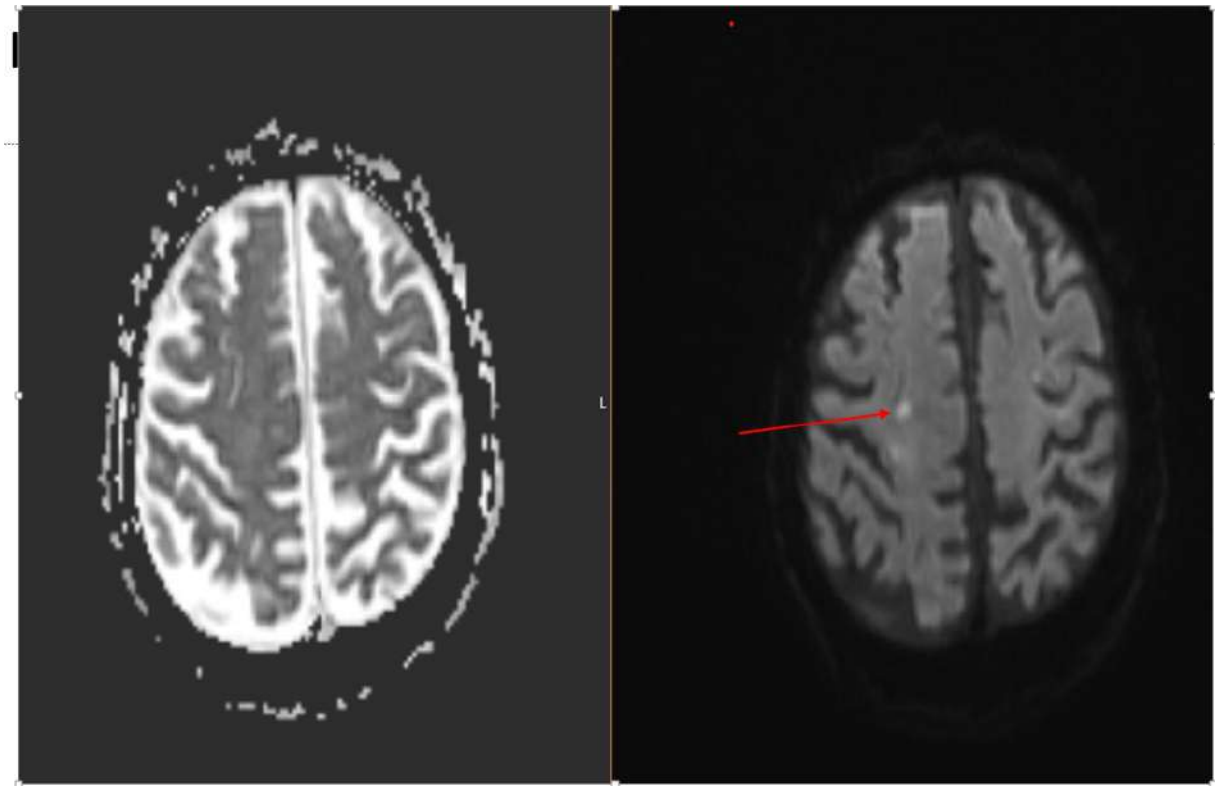

Figure 1. Diffusion-weighted brain magnetic resonance imaging shows a representative focus of restricted diffusion detected in the patient; in the clinical context, this finding is compatible with an ischemic embolic lesion.

Discussion

The incidence of infective endocarditis is estimated to be approximately 50–60 times higher in patients undergoing dialysis than in the general population (4, 6). In the present case, the patient presented to the emergency department primarily with hypotension, transient loss of consciousness, fatigue, and gait disturbance, while no clearly audible new murmur was detected during the initial examination. This clinical pattern highlights the diagnostic difficulty of infective endocarditis in chronic hemodialysis patients, particularly in elderly individuals with multiple comorbidities, in whom the disease may manifest predominantly with sepsis, neurological symptoms, or nonspecific systemic complaints rather than classic cardiac findings (3, 6). Current guidelines emphasize that the absence of a murmur does not exclude infective endocarditis when clinical suspicion is high, and that blood cultures and echocardiographic evaluation should not be delayed in such patients(1). The most notable risk factor in our case was the patient's long-term hemodialysis treatment for end-stage renal disease and the presence of a hemodialysis catheter. In hemodialysis patients, repeated vascular access, catheter-related bacteremia, uremia-associated impairment of immune response, and frequent hospital exposure may facilitate the development of infective endocarditis. Central venous catheters have been reported as one of the most important risk factors for both bacteremia and subsequent endocardial involvement in this patient population(4, 6). In healthcare-associated infective endocarditis, intravascular catheters and repeated vascular access have been identified as the source of infection in approximately 40–50% of cases (9). Therefore, the absence of erythema, discharge, or tenderness at the catheter insertion site does not completely exclude the possibility of catheter-related bacteremia. The identification of *Enterococcus faecalis* as the causative microorganism is another important aspect of this case. Although *Staphylococcus aureus* remains the most common pathogen in hemodialysis-associated infective endocarditis, enterococci are increasingly reported in patients with advanced age, multiple comorbidities, healthcare-associated bacteremia, and possible genitourinary or gastrointestinal sources (4, 5). In our patient, the absence of bacterial growth in the urine culture and the lack of a clear pneumonic focus on thoracic imaging made the existing hemodialysis catheter a more likely portal of entry. Nevertheless, in cases of *E. faecalis* bacteremia, non-catheter-related sources should also be considered, and further evaluation of the gastrointestinal and genitourinary systems should be guided by the clinical context(1, 5). Tricuspid valve involvement represents one of the distinguishing features of this case. Although right-sided infective endocarditis is classically associated with intravenous drug use or cardiac implantable devices, central venous catheters and hemodialysis access routes may also provide an important substrate for right-sided endocardial infection (6, 9). In our patient, the absence of intravenous drug use or a history of an intracardiac device suggests that the large and mobile vegetation on the tricuspid valve may have developed secondary to hemodialysis catheter-related bacteremia. In the literature, large vegetations, persistent bacteremia, recurrent embolic events, and severe valvular

dysfunction are considered high-risk features in right-sided infective endocarditis and may require surgical or interventional evaluation(1, 10). The detection of multiple lacunar foci with restricted diffusion on diffusion-weighted MRI raised concern for neurological involvement secondary to infective endocarditis. Neurological complications of infective endocarditis may present with a broad spectrum of manifestations, including ischemic embolism, septic embolism, intracranial hemorrhage, mycotic aneurysm, and encephalopathy. In the presence of fever, bacteremia, and valvular vegetation, multiple diffusion-restricted lesions strongly support an embolic pathophysiology(1, 3). Although embolic events in right-sided endocarditis are expected to involve the pulmonary circulation more frequently, paradoxical embolism through an intracardiac shunt, unrecognized concomitant left-sided involvement on transthoracic echocardiography, or ischemic injury related to sepsis and hypotension should also be considered. Accordingly, even when transthoracic echocardiography provides diagnostic evidence, transesophageal echocardiography may offer additional diagnostic value in patients with persistent clinical suspicion or embolic complications(1). Treatment of *E. faecalis* infective endocarditis should be individualized according to antimicrobial susceptibility, renal function, the involved valve, the presence of complications, and the patient's operative risk. In patients with renal failure, aminoglycoside-related toxicity represents an important limitation; therefore, in cases with an appropriate susceptibility profile, dual beta-lactam regimens such as ampicillin plus ceftriaxone have emerged in the current literature as an effective and safer therapeutic option(1, 8). During the empirical treatment phase, however, antimicrobial therapy should be adjusted by considering local resistance patterns, the severity of the patient's clinical condition, and subsequent culture results.

In conclusion, this case demonstrates that infective endocarditis should be considered early in hemodialysis patients presenting with fever, hypotension, transient alteration of consciousness, or otherwise unexplained neurological findings. The absence of obvious local signs of infection at the catheter insertion site should not exclude the diagnosis. Blood cultures, early echocardiographic assessment, central nervous system imaging, and catheter source control represent key steps that may reduce morbidity and mortality, particularly in patients with suspected embolic complications.

Keywords: Hemodialysis, Infective endocarditis, Septic embolism.

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6006

Myofascial Chest Pain That May Be Mistaken for Cardiac Pain in the Emergency Department: A Narrative Review

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ABSTRACT

Introduction and Purpose: A significant proportion of patients presenting to emergency departments (ED) with chest pain are found to have noncardiac chest pain (NCCP) after cardiac causes have been ruled out. Among these patients, musculoskeletal pain, particularly Myofascial Pain Syndrome (MPS), which can mimic angina pectoris, constitutes a clinically significant subgroup. The aim of this review is to present the prevalence of MPS cases mistaken for cardiac origin in ED, the diagnosis and treatment processes, and the critical role of physical examination in differential diagnosis, based on current literature data.

Method: This study was designed as a narrative review. Guidelines, systematic reviews, case reports, observational, and randomized controlled trials published between January 2007 and March 2026 were searched in the PubMed/MEDLINE and Google Scholar databases. Selected studies were synthesized based on themes according to the principles of contributing to the initial assessment of chest pain in ED, including MPS diagnostic criteria, and presenting applicable treatment approaches.

Results: The prevalence of musculoskeletal chest pain (MSCP) in ED series appears to be approximately 15-16% in most studies; however, it may vary between centers. Active MTPs in chest wall muscles, such as the pectoral muscles and serratus anterior, can cause referred pain mimicking cardiac ischemia through somatovisceral convergence and autonomic nervous system interactions. The diagnostic approach in the ED relies primarily on rapidly ruling out fatal cardiac conditions using electrocardiography and troponin. Subsequently, the identification of a taut band and hypersensitive focus during a focused examination, and the reproduction of the patient's “familiar/referred pain” with this pressure, supports the diagnosis of MPS. In acute treatment, reassurance, local heat, stretching, and ischemic compression are first-line; trigger point injection and dry needling are alternatives for refractory cases.

Conclusion: MPS is not merely a diagnosis of exclusion in the differential diagnosis of chest pain in ED; it is a clinical syndrome that should be actively sought with positive physical examination findings. Accurate diagnosis and management of MPS in ED can improve patient satisfaction by preventing unnecessary cardiac investigations and repeated in ED visits.

Keywords: Chest Pain, Myofascial Pain Syndrome, Trigger Points, Referred Pain

1. Introduction

Chest pain is one of the most common reasons for emergency department (ED) visits worldwide, accounting for approximately 5% to 12% of all visits (1,2). The primary priority in emergency medicine practice is the rapid identification of life-threatening "fatal" causes such as acute coronary syndrome (ACS), aortic dissection, and pulmonary embolism (1,3). However, despite the increasing use of technological capabilities and biomarkers (such as high-sensitivity troponins), no cardiac pathology is detected in 60% to 80% of chest pain presentations, a condition defined as "noncardiac chest pain" (NCCP) (3,4).

Musculoskeletal causes constitute a clinically significant subgroup among patients presenting to the ED with chest pain, rather than being one of the underlying causes in a significant proportion of patients with NCCP. Systematic review data indicate that the pooled prevalence of musculoskeletal chest pain (MSCP) in the ED is approximately 16%. Similarly, a retrospective review conducted at four centers in New Zealand found this rate to be 15%, with a range of 11% to 31% between centers. However, there is marked heterogeneity between individual studies, with some series reporting a prevalence as high as 49% (2,5). Despite this high prevalence, after risk stratification in EDs and exclusion of cardiac causes, patients are often discharged with a diagnosis of "nonspecific chest pain" without receiving a specific musculoskeletal diagnosis. It is not always easy to specifically identify MSCP after cardiac causes have been excluded; a significant proportion of cases may remain in the group of chest pain that cannot be causally differentiated at the initial evaluation (5).

Within this clinical picture, Myofascial Pain Syndrome (MPS) can cause referred pain mimicking angina pectoris, particularly through active myofascial trigger points (MTPs) in the chest wall muscles (e.g., serratus anterior, pectoral muscles). Active MTPs in chest wall muscles such as the pectoral muscles and serratus anterior can create pain patterns that may suggest cardiac-related pain and can radiate to surrounding areas (6-8). "Reproducible chest wall tenderness" detected during physical examination has a sensitivity of 92.9% and a very high negative predictive value of 98.1% in ruling out ACS. This finding alone is not a criterion for discharge; it should be evaluated in conjunction with clinical risk and biomarkers (9). However, the nature of MPS, which involves autonomic phenomena and sensory disturbances, may mislead the clinician toward a cardiac focus. Sensory changes and autonomic symptoms related to MTPs may be seen in myofascial pain syndrome; this picture can be clinically confused with cardiac-related pain, especially when the chest wall muscles are affected (10-12).

Patients with NCCP who do not receive a specific diagnosis in the ED make repeated visits to the ED due to recurrence of symptoms, undergo unnecessary invasive procedures, and enter a cycle of severe anxiety and low quality of life (4). MPS originating from the chest wall is also one of the most important hidden contributors to this picture and morbidity cycle due to its angina-mimicking nature.

This study aims to present the diagnostic approach, treatment processes, prevalence, and the critical role of physical examination in differential diagnosis of chest pain that is "mistaken for cardiac" but is actually myofascial in origin in EDs, based on current literature data.

2. Review Method

This study was prepared as a narrative review. Guidelines, systematic reviews, case reports, observational, and randomized controlled trials published between January 2007 and March 2026 were searched using the PubMed/MEDLINE and Google Scholar databases. The search terms consisted of "myofascial pain syndrome," "myofascial trigger point," "noncardiac chest pain," "musculoskeletal chest pain," "emergency department," and similar combinations.

Three principles were adopted when selecting studies: first, direct contribution to the initial assessment of chest pain in the ED; second, inclusion of the clinical definition and diagnostic criteria of MPS; and third, reporting of applicable results for treatment. Case reports and data confined solely to chronic pain clinic settings were de-emphasized when they did not make a meaningful contribution to emergency medicine practice. The data obtained were synthesized and presented based on themes.

3. Definition, pathophysiology, and mimicry of cardiac pain in MPS

MPS is a complex clinical condition characterized by taut bands containing MTPs within skeletal muscles and and associated fascial networks, which exhibit excessive sensitivity to palpation and cause localized and referred pain (13). It is increasingly accepted today that dysfunction within the body's myofascial continuity network plays a critical role in the etiology of NCCP, which can clinically mimic angina pectoris, the primary symptom of ischemic heart disease. The possible contribution of the myofascial continuity network is emphasized in some musculoskeletal cases of NCCP; however, the extent of this relationship is not yet fully clear and is often overlooked in clinical practice (11).

The particular importance of MPS within this spectrum stems from its ability to mimic cardiac pain due to referred pain patterns, despite being a locally muscle-related pain syndrome. MTPs originating from the pectoralis minor and pectoralis major, in particular, can cause pain in the precordial region, radiation to the shoulder and upper extremity, and a feeling of pressure or tightness; this can easily be interpreted by the patient as cardiac pain

(11,13,14). In the ED, this similarity can lead to unnecessary advanced cardiac investigations, repeated visits, and prolonged diagnostic uncertainty (4,15,16). In international consensus studies, taut bands, a hypersensitive spot, and referred pain have been highlighted as the core components of MTP diagnosis; the re-creation of the patient's familiar pain during examination has been accepted as an important finding in favor of an active MTP (14,17). Therefore, MPS is not merely an uncertain possibility considered after exclusion, but a clinical syndrome that can be positively identified with appropriate physical examination.

Autonomic nervous system (ANS) interactions, central sensitization related to peripheral nociceptive input, and intersegmental convergence processes may play a role in explaining this clinical similarity. Continuous nociceptive input from MTPs can sensitize dorsal horn neurons; conversely, cardiac pain can also manifest with referred pain patterns by its very nature. These shared processing mechanisms may contribute to the clinical overlap between chest wall-related myofascial pain and cardiac pain in some cases (10,12,14,18).

4. The role of MPS in the spectrum of chest pain in the emergency department

Chest pain is one of the most critical reasons for presentation to the ED, both in terms of clinical importance and time pressure. Therefore, the initial approach focuses on rapidly ruling out life-threatening causes, primarily ACS. The 2021 AHA/ACC chest pain guideline recommend the early use of Electrocardiography (ECG), serial cardiac troponin assessment, and the routine integration of structured clinical decision pathways based on high-sensitivity troponin into practice. The same guideline recommend defining chest pain as "cardiac/possible cardiac/noncardiac" rather than "typical/atypical," directing clinicians toward a broader differential diagnosis rather than focusing solely on coronary ischemia (1). Even after life-threatening cardiac conditions have been ruled out, patients may continue to experience severe pain radiating to the chest, left shoulder, and arm. Active MTPs developing in specific muscle groups surrounding the chest wall, such as the serratus anterior muscle, create serious referred pain patterns that give the patient the feeling of having a myocardial infarction, creating a cardiac illusion (pseudo-angina) that can mislead physicians and patients (6).

NCCP poses a greater burden in emergency medicine practice than commonly believed. Myofascial pain is recognized as a significant cause of disability in the working-age population. However, the frequently cited rate of approximately 30% in the literature does not apply to the ED population, but rather to a selected group of patients presenting with pain in primary care. Therefore, it is thought that the true frequency of MPS in the ED is not clearly defined, but is often overlooked in clinical practice (15). Frieling emphasized that the rate of NCCP can reach up to 70% at different levels of healthcare and that this condition can cause a deterioration in quality of life similar to cardiac chest pain, and in some cases even more

pronounced (4). Rushton and Carman noted that NCCP can be classified under gastrointestinal, musculoskeletal, psychiatric, and pulmonary/other causes, but that a clear etiological explanation cannot be reached in every patient (16). ED data also show that MSCP has a significant, rather than marginal, share within this broad spectrum: Mandrekar et al.'s systematic review and meta-analysis reported a pooled prevalence of MSCP of approximately 16%; Dacombe-Bird et al. found this rate to be 15% in their 2024 multicenter retrospective study and placed musculoskeletal causes second after cardiovascular causes (2,5). These data suggest that although MPS does not represent all MSCP, it should be considered a significant musculoskeletal subset within the spectrum of chest pain in the ED, rather than a "secondary" one (2,4,5,15).

5. Diagnostic approach in the emergency department

Before diagnosing MPS in the ED, the patient must be safely classified in terms of clinically dangerous causes. Current chest pain guidelines recommend rapid identification of life-threatening cardiac and non-cardiac causes during the initial assessment; this requires a detailed history, physical examination, 12-lead ECG, and high-sensitivity troponin in appropriate patients (1,6). Findings such as hemodynamic instability, new ischemic ECG changes, elevated troponin, significant hypoxemia, syncope, severe dyspnea, tearing-like pain suggestive of aortic dissection, or a history of trauma should not be interpreted in favor of MPS (1,15,16).

Historical clues in favor of MPS include: pain worsening with the use of a specific muscle, prolonged posture, exertion, or repetitive movements; the patient being able to localize the pain with one or two fingers; the pain recurring with shoulder-trunk movement or pressure on a specific point; and previous similar attacks having resulted in normal cardiac investigations (12-14,17). The goal of the examination is to identify tense bands and hypersensitive foci in areas such as the pectoralis major-minor, intercostal muscles, serratus anterior, scalene muscles, sternocleidomastoid, and upper trapezius; and to be able to reproduce the patient's familiar pain or referred pain with this pressure (6,11-14,17).

Therefore, the goal in the ED is to consider MPS not only as a "diagnosis of exclusion" but as a clinical diagnosis supported by positive physical examination findings. In practice, this requires a two-step approach: first, safe cardiac exclusion, followed by a focused musculoskeletal examination.

Table 1. Clinical findings in favor of MPS

Findings supporting MPS
Low-risk appearance with serial ECG and troponin

Recurrence of pain upon palpation of a specific muscle
Identification of an excessively sensitive trigger point within a taut band
Increase in discomfort with shoulder or trunk movement
History of posture, overuse, or repetitive movements
Partial relief with stretching, local heat, or manual pressure

MPS: Myofascial Pain Syndrome ECG: Electrocardiography

Note: This table was created to support practical clinical decision-making in the ED and does not, by itself, provide a definitive diagnosis.

6. Management and Treatment Approaches for MPS in the Emergency Department

Once the diagnosis of MPS is confirmed in the ED, the treatment strategy should aim to break the patient's pain cycle and prevent repeat visits to the ED (16). Despite the time and capacity constraints of the ED, basic interventions that can be performed by emergency physicians are both cost-effective and directly increase patient satisfaction (15).

First-line treatment generally involves non-invasive methods. In initial treatment, non-invasive methods may be considered first in selected patients. Reassuring the patient that their current pain is not cardiac in origin but stems from a benign musculoskeletal spasm and alleviating their anxiety is one of the most important steps (16). Providing the patient with explanatory and reassuring information after cardiac causes have been ruled out may contribute to reducing anxiety. In addition, local heat application to the affected muscle, ischemic compression (manual pressure) on the MTP, and simple posture/stretching exercises taught to the patient may alleviate symptoms (12,16,19).

Invasive methods are considered when conservative methods are insufficient or when the pain is very severe. The choice between invasive and noninvasive approaches varies depending on the severity of pain, previous treatment attempts, and the practical conditions of the ED. Dry needling therapy has been reported to be quite successful in the management of serratus

anterior-origin MTPs mimicking acute myocardial infarction. It has been reported that MTPs originating from the serratus anterior can mimic cardiac-related acute chest pain and that dry needling is one of the interventional options that can be applied with careful technique in these cases (6,15). Since methods such as dry needling carry the risk of hematoma and infection, "Trigger Point Injection" (TPI) stands out as another alternative method that can be applied in the ED, considering the limited time and resources of the ED. Roldan and Hu stated that MPS can be effectively treated by emergency physicians with TPI after correct diagnosis and emphasized that this application is a feasible/effective option in selected cases in EDs (15). A randomized controlled trial conducted on patients presenting with NCCP complaints and identified MTPs in their pectoral muscles demonstrated that MTP injection significantly reduced pain intensity in the short and medium term (7).

In addition to needling therapies, myofascial trigger point manual therapy is also an effective option. A recent meta-analysis comparing dry needling with myofascial trigger point manual therapy in patients with MPS in the neck and upper back region confirmed that both methods showed similar, high clinical efficacy in reducing pain intensity [Visual Analog Scale (VAS)] and disability [Neck Disability Index (NDI)] (19).

7. Post-Emergency Department Follow-Up and Multidisciplinary Approach

Symptomatic relief provided in the ED is generally insufficient for the long-term management of MPS (13). Unless the underlying causes of MSCP—poor posture, repetitive microtrauma, biomechanical dysfunction, or accompanying cervical pathology—are corrected, the risk of MTPs reactivating (recurrence) is quite high (13).

Therefore, the role of the emergency physician does not end with relieving acute pain and ruling out fatal causes; it also includes making the correct outpatient referral. In an ideal healthcare model, patients discharged from the emergency department with a preliminary diagnosis of NCCP and MPS should be referred to Physical Medicine and Rehabilitation (PMR) clinics for posture analysis, structured exercise prescriptions, advanced manual therapy techniques, and biomechanical corrections (13). This multidisciplinary bridge will reduce the unnecessary patient load on EDs while permanently preventing patients from entering a cycle of chronic pain and disability. Structured multidisciplinary referral after the ED can contribute to reducing repeat visits and healthcare utilization, as well as lowering the patient's risk of chronicity and functional loss (4,13).

8. Conclusion

MPS is a common but often underdiagnosed cause of chest pain mistaken for cardiac origin in the ED. The fundamental principle for the emergency physician is to first rule out life-threatening cardiac and thoracic causes; then, based on positive physical examination

findings, to look for MPS. An excessively sensitive MTP within a taut band, the reproduction of familiar pain on palpation, and a referred pain pattern are findings of high diagnostic value.

Patient education, stretching exercises, posture correction, and TPI in selected cases are the main treatment options supported by the current literature. More systematic recognition of MPS in the ED may contribute to reducing unnecessary investigations and repeat visits, as well as increasing patient satisfaction and symptom control. Therefore, MPS should not only be considered in the differential diagnosis of chest pain but should also be actively sought as a clinical possibility.

Ethics committee approval

Since this study is a review, it does not require ethics committee approval.

Abbreviations

ACC : American College of Cardiology

ACS: Acute Coronary Syndrome

AHA: American Heart Association

ANS: Autonomic Nervous System

ECG: Electrocardiography

ED: Emergency Department

MPS: Myofascial Pain Syndrome

MSCP: Musculo Skeletal Chest Pain

MTP: Myofascial Trigger Points

NCCP: Noncardiac Chest Pain

NDI: Neck Disability Index

PMR: Physical Medicine and Rehabilitation

TPI: Trigger Point Injection

VAS: Visual Analog Scale

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Inflammatory Biomarkers and Clinical Outcomes in Intensive Care Unit Pneumonia: The Role of Procalcitonin and Ferritin

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Abstract

Background:
Pneumonia remains a significant cause of morbidity and mortality in intensive care units (ICUs), particularly among elderly patients. Inflammatory biomarkers such as procalcitonin (PCT) and ferritin have been increasingly used to evaluate disease severity and prognosis. This study aimed to investigate the relationship between these biomarkers, clinical parameters, and outcomes in ICU patients with pneumonia.

Methods:
In this retrospective study, patients with pneumonia admitted to the reanimation ICU between January 1, 2025, and June 30, 2025, were evaluated. A total of 38 patients were screened, and 33 patients were included after excluding those with non-pneumonia-related mortality or confounding clinical conditions. Statistical analyses were performed using IBM SPSS Statistics 27. Data distribution was assessed with the Shapiro-Wilk test. Parametric variables were analyzed using the Student's t-test, and non-parametric variables were evaluated with the Mann–Whitney U test. Categorical variables were compared using the chi-square test or Fisher's Exact test where appropriate. Correlation analyses were performed using Spearman's test.

Results:
The median age of the patients was 74 years, and 66.7% were male. The overall mortality rate was 18.2%. Ferritin levels were significantly higher in male patients. Procalcitonin levels were significantly elevated in non-survivors, and both age and length of hospital stay were higher among patients who died. Although mortality was observed only in male patients, this difference was not statistically significant. Positive correlations were found between age, PCT, ferritin levels, and hospital length of stay.

Conclusion:
PCT and ferritin levels may serve as useful biomarkers in assessing disease severity and predicting clinical outcomes in ICU patients with pneumonia. The association between

increased age, inflammatory markers, and prolonged hospitalization highlights their potential role in clinical decision-making. Larger studies are needed to confirm these findings.

Keywords: Pneumonia, Procalcitonin, Ferritin, Intensive Care Unit, Mortality

Introduction

Pneumonia remains a major cause of morbidity and mortality in intensive care units (ICUs), particularly among elderly patients. Its clinical course may range from mild respiratory tract infections to life-threatening respiratory failure, with mortality rates increasing substantially in cases requiring intensive care support. Therefore, early and accurate assessment of disease severity and prediction of prognosis are of critical importance in clinical management.

Although several clinical scoring systems are used to determine the prognosis of patients with pneumonia, inflammatory biomarkers have gained increasing attention in recent years. Procalcitonin (PCT) is a biomarker reflecting the severity of bacterial infections and systemic inflammation and has been associated with disease severity and mortality, especially in patients with community-acquired pneumonia [1]. Ferritin, on the other hand, serves not only as an indicator of iron metabolism but also as an acute-phase reactant that increases during inflammatory processes and may correlate with the extent of infection [2].

Advanced age is an important risk factor for both the development of pneumonia and poor prognosis. The process of immunosenescence associated with aging is characterized by impaired immune response and chronic low-grade inflammation, which alters the host response to infections. This condition may also influence the clinical significance and prognostic value of inflammatory biomarkers.

The aim of this study was to evaluate the relationship between procalcitonin and ferritin levels and clinical parameters and outcomes in patients with pneumonia admitted to the intensive care unit. Additionally, the interaction between age, inflammatory response, and length of hospital stay was investigated to explore the potential clinical utility of these biomarkers.

Materials and Methods

In this retrospective study, patients with pneumonia who were followed in the intensive care unit were evaluated. A total of 38 patients admitted to the reanimation intensive care unit between January 1, 2025, and June 30, 2025, were initially reviewed. Five patients with additional pathologies worsening the clinical course and whose mortality was unrelated to pneumonia were excluded from the study. Consequently, 33 patients were included in the final analysis.

Statistical analyses were performed using IBM SPSS Statistics version 27. The normality of continuous variables was assessed using the Shapiro–Wilk test. Parametric variables were

expressed as mean ± standard deviation, whereas non-parametric variables were presented as median and interquartile range (IQR).

For comparisons between groups, the independent samples t-test was used for normally distributed variables, while the Mann Whitney U test was applied for non-normally distributed variables. Categorical variables were compared using the chi square test. Fisher's Exact test was preferred when expected cell frequencies were low.

Spearman correlation analysis was performed to evaluate relationships between variables. A p value <0.05 was considered statistically significant.

Results

A total of 33 patients with pneumonia admitted to the intensive care unit were evaluated.

The median age of the patients was 74 years (IQR: 60–83). The median ferritin level was 447 (IQR: 249–1144), and the median length of hospital stay was 2 days (IQR: 2–4).

Of the patients, 66.7% (n=22) were male and 33.3% (n=11) were female.

Mortality was observed in 18.2% (n=6) of the patients, while the remaining patients were discharged alive.

Ferritin levels differed significantly according to sex (p=0.001), with higher ferritin levels observed in male patients.

Procalcitonin levels were significantly associated with mortality (p=0.013), being higher in patients who died. Length of hospital stay was also significantly longer in deceased patients (p=0.001). In addition, the age of deceased patients was significantly higher compared to survivors (p=0.035).

Among the 22 male patients, mortality occurred in 6 patients (27.3%), whereas no mortality was observed among female patients (0/11, 0%). Although mortality appeared to be higher in males, this difference did not reach statistical significance according to Fisher's Exact test (p=0.077).

A weak positive correlation was found between age and procalcitonin levels (r=0.372, p=0.033). Similarly, age demonstrated a weak positive correlation with length of hospital stay (r=0.349, p=0.049). Procalcitonin levels showed a moderate positive correlation with length of hospital stay (r=0.646, p=0.001). Furthermore, ferritin levels were moderately positively correlated with length of hospital stay (r=0.521, p=0.002), as shown in Table 1.

Table 1. Correlation Analysis Between Age, PCT, Ferritin, and Hospital Length of Stay

Parameter		n	r	p
Age	PCT	33	0.372	0.033
Age			0.349	0.049
PCT	Hospital Length of Stay		0.646	0.001
Ferritin			0.521	0.002

Discussion

In our study, the median age of the patients was 74 years, which is consistent with the literature reporting that patients with pneumonia requiring intensive care are generally in the advanced age group [3]. This finding may be explained by the increased need for intensive care in elderly patients due to the more severe clinical course of pneumonia resulting from impaired immune responses. In addition, the inclusion of patients over 65 years of age in the high-risk category within the CURB-65 scoring system and the subsequent intensive care admission decisions may also have contributed to this result.

The fact that 66.7% of our patients were male is consistent with the literature indicating male predominance among patients with pneumonia treated in intensive care units. Previous studies have reported that the proportion of male patients in ICU pneumonia cohorts is approximately 60–65% [4]. The higher incidence of pneumonia in males may be associated with factors such as smoking and a greater burden of comorbidities.

In our study, the mortality rate was found to be 18.2%. Previous studies in the literature have reported approximately 23% 30-day mortality and around 28% 90-day mortality rates in pneumonia patients [5]. The relatively lower mortality rate observed in our reanimation intensive care unit may be attributed to the effective implementation of early diagnosis and appropriate treatment strategies.

Ferritin levels were found to be significantly higher in male patients. Previous studies have similarly reported that serum ferritin levels are higher in males than in females. We believe that this difference may be explained by both physiological and pathological mechanisms [6]. Physiologically, males have higher baseline iron stores, and therefore higher ferritin levels during disease states are expected. Pathologically, the more frequent occurrence of smoking and comorbid diseases in males may contribute to a more pronounced inflammatory response, thereby explaining the increased ferritin levels. Accordingly, both processes are thought to contribute to ferritin elevation.

The finding that procalcitonin levels were higher in deceased patients is consistent with studies reporting an association between procalcitonin levels and mortality [7]. Since procalcitonin is a marker reflecting the severity of bacterial infections and systemic inflammation, elevated PCT levels may indicate increased mortality risk.

Length of hospital stay was found to be longer in deceased patients. In addition, deceased patients were older compared to survivors. These findings suggest that increased disease severity necessitates prolonged hospitalization and is associated with mortality. Prolonged hospitalization may reflect an increased burden of complications and worsening clinical course. Furthermore, the association between advanced age and mortality is an expected finding and should be interpreted within this clinical context.

Mortality was observed in 6 male patients (27.3%), whereas no mortality was detected among female patients. Although mortality appeared to be higher in males, this difference was not statistically significant according to Fisher's Exact test. Nevertheless, the observation that mortality occurred only in male patients is noteworthy and may be clinically relevant in terms of possible sex-related differences. However, this finding should be interpreted cautiously due to the limited sample size.

In our study, a weak but significant positive correlation was found between age and procalcitonin levels. Advanced age is characterized by impaired immune function and is associated with the process of inflammaging, which involves chronic low-grade inflammation and dysregulation of immune responses. This immunological remodeling may influence both the host response to infection and procalcitonin release. Therefore, despite the more severe course of infection in elderly patients, the procalcitonin response may not increase to the expected extent. Indeed, it has been reported that increases in procalcitonin levels may be more limited in elderly patients compared to younger individuals due to alterations in inflammatory responses and reduced parenchymal cell reserve. This suggests that procalcitonin levels may not demonstrate a direct linear relationship with age but are more likely influenced by infection severity, etiology, and individual immune response differences [8]. Although procalcitonin levels are known to be associated with inflammatory response, the weak strength of the correlation may be explained by the process of immunosenescence observed in advanced age. In this context, the positive but weak correlation identified in our study may reflect not only the effect of age itself but also disease severity and accompanying clinical factors.

In our study, positive correlations were identified between age, procalcitonin and ferritin levels, and length of hospital stay. Previous studies have similarly demonstrated that increases in inflammatory markers are associated with disease burden and prolonged clinical course. Elevated procalcitonin and ferritin levels may accompany longer hospital stays, as these markers reflect the extent of infection and the degree of systemic inflammatory response [9]. Furthermore, increasing age may contribute to prolonged hospitalization due to greater variability in clinical response and delayed treatment response. Accordingly, the findings obtained in our study may indicate that increased inflammatory activity and individual differences in clinical response contribute to prolongation of the disease course.

Conclusion

In this study, procalcitonin and ferritin levels were shown to be associated with disease severity and clinical course in patients with pneumonia treated in the intensive care unit. In

particular, the association between procalcitonin levels and mortality suggests that this parameter may serve as a prognostic biomarker. The relationship between advanced age, elevated inflammatory markers, and prolonged hospital stay indicates that these parameters may be useful in clinical monitoring. Although sex-related differences did not reach statistical significance, they may still be clinically noteworthy. Further studies with larger sample sizes are needed to support these findings.

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Comparison of Emergency Physicians' Pneumothorax Diagnosis Performance with Artificial Intelligence Models

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Abstract

Objectives: This study aims to compare emergency physicians' performance with that of general-purpose large language models (LLMs), such as ChatGPT and Gemini, for pneumothorax (PTX) detection on chest radiographs (CXRs).

Methods: This single-centre, retrospective study of adults was conducted between January 2015 and February 2025 and included 265 PTX cases and 267 non-PTX controls. Exclusions included diagnoses made only by computed tomography, absence of CXR, initial treatment at another centre or incomplete data. Thirteen emergency physicians independently and blindly reviewed CXRs and recorded a binary decision ChatGPT and Gemini evaluated the same images with a standardised yes/no prompt, with memory cleared between cases to prevent carryover. The primary outcome was LLM diagnostic performance for PTX, while the secondary outcome compared LLMs with physicians.

Results: ChatGPT and Gemini exhibited distinct diagnostic performance profiles for pneumothorax detection on chest radiographs. Gemini demonstrated a sensitivity of 52.5%, whereas ChatGPT demonstrated a sensitivity of 44.5%. Conversely, ChatGPT achieved a specificity of 95.5% and an overall accuracy of 70.1%, while Gemini demonstrated a specificity of 79.0% and an accuracy of 65.8%. Agreement with the reference standard was moderate for ChatGPT, with a kappa value of 0.401, and fair for Gemini, with a kappa value of 0.315. Increasing case difficulty was associated with a reduction in diagnostic accuracy for both models, with correlation coefficients of −0.438 for ChatGPT and −0.274 for Gemini. For contextual clinical comparison, emergency physicians demonstrated a sensitivity of 64.5%, a specificity of 99.6%, and an overall accuracy of 82.1%.

Conclusion: This study demonstrates model-specific differences in pneumothorax detection by general-purpose AI systems, with Gemini showing higher sensitivity and ChatGPT superior specificity and accuracy, both declining with increasing case difficulty. Physician performance remained higher but was secondary for context. Despite their accessibility and low cost, these models should be considered only adjunctive tools until task-specific optimization and clinical validation are achieved.

Keywords: Pneumothorax; Artificial Intelligence; Emergency Physicians.

6086

Implementing the "3S" Approach for Surge Capacity in a Mass Casualty Incident: Lessons from a Bus Crash

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Introduction

Mass casualty incidents are defined as situations in which the existing healthcare system capacity is insufficient and the number of patients exceeds available resources within a short period of time. In such events, the response of the healthcare system requires not only individual patient care but also system-level organization and resource management. In this context, the concept of surge capacity refers to the ability of a healthcare system to respond to a sudden increase in patient volume and encompasses the rapid reorganization of personnel, equipment, physical space, and organizational structures. Surge capacity is regarded as the ability to provide care for a greater number of patients beyond routine capacity within a limited timeframe (1,2).

In mass casualty incidents, surge capacity is classically described using the “3S” approach (staff, stuff, structure). Staff refers to the number, experience, and task distribution of healthcare personnel; stuff refers to medical equipment, medications, and consumable supplies; and structure refers to physical space, bed capacity, and organizational infrastructure. Simultaneous and balanced expansion of these three components is essential for an effective response to a sudden patient surge. Particularly in emergency departments, reorganization of existing spaces, rapid segregation of non-critical patients, and clear role allocation within the team are among the key elements enabling effective use of surge capacity (3).

Traffic accidents, especially those involving vehicles carrying multiple passengers, are among

the most common causes of mass casualty incidents. In such events, emergency departments are required to rapidly increase capacity, reorganize space management, and work in coordination with multidisciplinary teams. In this case report, the emergency department management, triage practices, and surge capacity response following a collision between a school shuttle minibus and a school bus are presented.

Case Presentation

While routine operations in the trauma area were ongoing, the emergency department was placed on alert following a notification from the emergency medical dispatch center. Although information regarding the scene was limited, it was reported that a school shuttle minibus had collided with a school bus and that approximately 40 injured individuals were present at the scene. As our hospital was the closest healthcare facility to the incident site, it was anticipated that the majority of patients would be transported to our institution, and preparations were made accordingly.

Following the alert, the entire trauma team was informed, relevant specialty departments were notified, and additional physicians, nurses, patient transport personnel, and security staff were assigned to the trauma area. Hospital administration and intensive care teams were contacted and informed of the situation, and preparations for possible admissions were initiated. Coordination was established with law enforcement authorities for the management of legal procedures and patient identification. Simultaneously, communication was established with the hospital supply unit to ensure the availability of necessary medical equipment and consumable materials.

To increase emergency department capacity, the ambulatory triage area was informed, green-zone patients were asked to wait, and yellow-zone patients were redirected to appropriate areas. This allowed expansion of the trauma area and facilitated the admission of multiple trauma patients. Functionally, the trauma area was divided into four sections: an initial reception and physician-led triage area, a monitored critical care area, an unmonitored observation area, and an ambulatory patient assessment area. Physician and nurse responsibilities were assigned in advance, and one physician was designated as area coordinator with the sole responsibility of managing organization and workflow. A dedicated nurse was assigned to oversee patient identification, and all patients received identification wristbands upon admission.

The patient admission process lasted approximately 1.5 hours. The first patient arrived unconscious, was categorized as a red-code patient, and was transferred directly to the resuscitation room. Other patients were received in the trauma area and classified according to

their clinical status following rapid primary assessment. Two patients were assessed as critical and transferred to monitored areas. One of these patients was diagnosed with cerebral hemorrhage and the other with splenic laceration; both were admitted to the intensive care unit after a short period of emergency department observation.

Patients with less severe injuries were monitored in a separate observation area, and all patients underwent secondary and tertiary assessments before discharge. Since the initial assessments were intentionally rapid, observation and reassessment periods were deliberately prolonged. All patients were followed simultaneously using a patient list maintained by the area coordinator, and both clinical management and workflow were coordinated through this system.

A total of 24 patients were evaluated, including 19 pediatric and 5 adult patients. The interval between the admission of the first and last patient was approximately 1.5 hours. Workload decreased significantly after the second hour, and routine emergency department operations resumed by the fourth hour. Discharge of the final patient was completed within approximately 8 hours. At the end of the process, two patients were admitted to the intensive care unit (one of whom subsequently died during follow-up), two patients were planned for ward admission, and one patient declined hospitalization.

Discussion

This case report demonstrates that emergency department management during a mass casualty traffic accident involves not only clinical interventions but also system-level organization, surge capacity expansion, and effective workflow management. The arrival of a large number of patients within a short period necessitated a transition from routine emergency department operations to a dynamic crisis management model.

One of the most prominent aspects of this process was the effective implementation of the surge capacity concept. Surge capacity involves not only responding to increased patient volume but also reorganizing existing resources (1). In this context, the 3S approach (staff, stuff, structure) applied in our case enabled rapid reorganization of the emergency department. Personnel reinforcement (staff), provision of necessary equipment and supplies (stuff), and reorganization of physical space (structure) facilitated continuous and efficient patient management. In particular, functional division of the trauma area into designated zones and the appointment of an area coordinator played critical roles in maintaining workflow. Furthermore, postponing the management of green-zone patients and prioritizing the flow of critically ill patients ensured appropriate allocation of resources (3).

Another important aspect of this case report is the emphasis placed on secondary assessment and observation. Rapid initial assessment did not result in shortened observation periods; rather, all patients underwent repeat evaluation before discharge. This approach is particularly important in preventing overlooked injuries among patients with minor trauma. The literature has similarly demonstrated that secondary and tertiary trauma assessments significantly reduce the rate of missed injuries (4).

Finally, team coordination and leadership are among the most decisive factors in the management of such incidents. Active workflow management by the area coordinator and continuous use of the patient tracking list facilitated organization and accelerated decision-making processes in the management of multiple patients.

Conclusion

Emergency department management in mass casualty traffic accidents requires rapid surge capacity expansion, effective space organization, and structured team coordination. This case report demonstrates that dynamic surge management supported by the 3S approach enables the safe and effective evaluation of a large number of patients within a short period of time.

Although standard disaster preparedness protocols are important, the determining factor under real-world conditions is the ability to organize available resources rapidly and appropriately. In particular, functional division of treatment areas, structured patient flow, and repeated clinical assessments emerge as key elements that enhance patient safety.

This experience highlights that, in mass casualty incidents, not only preparedness but also adaptability and organizational flexibility are critical determinants of patient outcomes.

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Can Machine Learning Replace the SYNTAX Score for Predicting Triple-Vessel Disease in Patients with Acute Coronary Syndrome?

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METHODS

Study Design and Population

This study was designed as a retrospective, observational cohort study comprising patients presenting with acute coronary syndrome (ACS) who underwent coronary angiography. The primary objective was to predict the presence of triple-vessel coronary artery disease (CAD), defined as >70% stenosis in three major epicardial vessels, using clinical and laboratory data via machine learning (ML) algorithms.

Data Collection and Variable Selection

Demographic characteristics, vital signs, comorbid conditions, routine biochemical parameters, and inflammatory markers were obtained from the electronic medical record system. In addition to standard laboratory values, derived inflammatory indices such as neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), systemic immune-inflammation index (SII), pan-immune-inflammation value (PIV), white blood cell-to-platelet (WBC/PLT) ratio, and hemoglobin-to-creatinine (Hb/Creatinine) ratio were calculated. Composite clinical variables, including Age/eGFR and Glucose/eGFR ratios, were also included as candidate predictors.

Outcome Definition

The primary outcome was defined as the binary classification (presence/absence) of triple-vessel CAD. Angiographically confirmed >70% stenosis in three or more major coronary vessels was considered as triple-vessel disease.

Data Partitioning and Preprocessing

The dataset was divided into a 70% training set and a 30% testing set using stratified random sampling (random_state = 42). A preprocessing pipeline comprising median imputation for missing values followed by standard scaling (zero mean, unit variance) was applied. The pipeline was fitted exclusively to the training set and subsequently applied to the test set.

Model Development

Considering the imbalanced distribution of the outcome (~27% triple-vessel, ~73% non-triple-vessel), class balancing was implemented across all models. The class_weight="balanced" parameter was used for Logistic Regression, Random Forest, and SVM; whereas the scale_pos_weight parameter was utilized for XGBoost. Classification thresholds were optimized by applying Youden's J statistic (J = Sensitivity + Specificity − 1) to the test set ROC curve.

Five supervised ML classifiers were developed to predict triple-vessel CAD: Logistic Regression (LR), Random Forest (RF), Support Vector Machine (SVM), XGBoost, and a multilayer perceptron artificial neural network (ANN). A SYNTAX score >22 threshold was included in all analyses as a reference comparator. Model performance was evaluated using stratified 5-fold cross-validation on the training set, with AUC-ROC serving as the primary metric.

Performance Metrics and Statistical Analysis

The following metrics were calculated on the test set at the Youden-optimal threshold: AUC-ROC, accuracy, sensitivity, specificity, and F1 score. 95% bootstrap confidence intervals for test AUC values were calculated using 1,000 resampling iterations. The AUC comparison between ML models and the SYNTAX >22 reference was performed using the DeLong method. Continuous variables were compared using the Mann–Whitney U test, while categorical variables were compared using the Chi-square or Fisher's exact test. A p-value of <0.05 was considered statistically significant. All analyses were performed in Python using the scikit-learn, XGBoost, SHAP, and SciPy libraries. To prevent data leakage, cross-validation was strictly applied only to the training set (70% split).

Model Interpretability

SHapley Additive exPlanations (SHAP) analysis was applied to the Random Forest model. SHAP values were calculated on the test set data processed through the fitted preprocessing pipeline. A beeswarm summary plot was generated to visualize the global feature contribution profile, and SHAP dependence plots were produced for the six most influential predictors.

RESULTS

Patient Characteristics

The cohort consisted of ACS patients who underwent coronary angiography. Triple-vessel CAD was detected in approximately 27% of the patients. Baseline demographic and clinical characteristics stratified by triple-vessel disease status are presented in Table 1.

In patients with triple-vessel disease, left ventricular ejection fraction (LVEF) was significantly lower (median 50% vs. 55%, p=0.013) and oxygen saturation (SpO_2; 96.5% vs. 97.0%, p=0.036) was reduced. Peak troponin I levels were markedly elevated in the triple-vessel group (9059.3 vs. 5373.5 ng/L, p=0.016), as were BNP levels (323.9 vs. 132.6 pg/mL, p=0.001). Renal function, assessed by estimated glomerular filtration rate (eGFR), was significantly lower in the triple-vessel group (70 vs. 81 mL/min, p=0.006). STEMI presentation was more frequent in triple-vessel disease (54.5% vs. 40.0%, p=0.017), whereas NSTEMI was less frequent (43.4% vs. 58.5%, p=0.016). Age, BMI, blood pressure, inflammatory indices, and comorbid conditions did not show significant differences between the groups.

Table 1. *Baseline demographic, clinical, and laboratory characteristics of patients with and without triple-vessel CAD. Continuous variables are presented as median (Q1–Q3) and categorical variables as n (%). *p<0.05.*

Variable	Non-Triple-Vessel Median (Q1–Q3) / n(%)	Triple-Vessel Disease Median (Q1–Q3) / n(%)	p-value
Continuous Variables			
Age (years)	60.00 (51.00–71.50)	63.00 (54.00–74.00)	0.076
BMI (kg/m²)	26.85 (24.22–29.18)	27.40 (25.15–29.32)	0.411
LVEF (%)	55.00 (45.00–60.00)	50.00 (40.00–60.00)	**0.013***
Heart Rate (bpm)	85.00 (74.50–99.50)	87.00 (74.00–105.00)	0.545
SBP (mmHg)	158.00 (140.00–175.00)	155.00 (133.00–176.00)	0.612
DBP (mmHg)	87.00 (76.00–97.00)	84.00 (71.00–98.00)	0.285

Variable	Non-Triple-Vessel Median (Q1–Q3) / n(%)	Triple-Vessel Disease Median (Q1–Q3) / n(%)	p-value
SpO_2 (%)	97.00 (96.00–98.00)	96.50 (95.00–98.00)	**0.036***
Killip Score	0.00 (0.00–0.00)	0.00 (0.00–0.00)	0.140
GRACE Score	100.00 (78.00–127.25)	110.00 (91.25–136.00)	0.062
Peak Troponin I (ng/L)	5373.50 (726.88–21870.03)	9059.30 (1946.20–34620.20)	**0.016***
Lactate (mmol/L)	2.33 (1.44–14.22)	1.90 (1.26–4.42)	0.431
CK-MB (µg/L)	5.05 (2.20–19.00)	6.50 (2.20–17.27)	0.476
AST/ALT oranı	26.00 (20.00–40.00)	28.00 (20.00–49.50)	0.130
Albumin (g/L)	21.00 (16.00–36.00)	27.00 (12.50–36.00)	0.944
BNP (pg/mL)	132.65 (55.35–381.68)	323.95 (162.18–587.33)	**0.001***
eGFR (mL/dak)	81.00 (62.00–95.25)	70.00 (48.50–88.00)	**0.006***
Glucose (mg/dL)	135.00 (110.00–188.00)	144.00 (110.00–193.75)	0.483
NLR	2.65 (1.60–4.32)	2.45 (1.60–3.92)	0.291
MLR	0.26 (0.19–0.39)	0.25 (0.19–0.34)	0.532
SII	680.34 (409.05–1095.80)	641.55 (382.67–1043.81)	0.694
Hb/Creatinine ratio	14.76 (12.61–16.98)	13.78 (10.88–17.11)	0.062

Variable	Non-Triple-Vessel Median (Q1–Q3) / n(%)	Triple-Vessel Disease Median (Q1–Q3) / n(%)	p-value
Categorical Variables			
Male gender	209 (%76.0)	75 (%74.3)	0.831
NSTEMI	158 (%58.5)	43 (%43.4)	**0.016***
STEMI	108 (%40.0)	54 (%54.5)	**0.017***
Hypertension	154 (%56.0)	63 (%62.4)	0.321
Diabetes mellitus	92 (%33.5)	40 (%39.6)	0.324
Prior MI	81 (%30.5)	37 (%37.0)	0.285

Machine Learning Model Performance

Performance metrics for all models are summarized in Table 2. In cross-validation (training set only), the Random Forest model achieved the highest mean AUC (0.728 ± 0.077), followed by XGBoost (0.710 ± 0.096), SYNTAX >22 (0.671 ± 0.051), SVM (0.668 ± 0.061), Logistic Regression (0.596 ± 0.084), and Neural Network (0.522 ± 0.076).

In the test set, the Random Forest model exhibited the highest AUC (0.812; 95% CI: 0.743–0.878), followed by XGBoost (AUC = 0.795; 95% CI: 0.722–0.864), Logistic Regression (AUC = 0.745; 95% CI: 0.643–0.838), SYNTAX >22 (AUC = 0.733; 95% CI: 0.651–0.810), and SVM (AUC = 0.726; 95% CI: 0.634–0.817). The Neural Network demonstrated the lowest discriminatory power (AUC = 0.603; 95% CI: 0.476–0.735). At the Youden-optimal threshold, the Random Forest model achieved a sensitivity of 0.933 and an F1 score of 0.523; whereas XGBoost demonstrated the highest sensitivity (0.967, F1 = 0.487). ROC curves for all models are presented in Figure 1.

Model	CV-AUC (mean±SD)	AUC	95% CI	Acc.	Sens.	Spec.	F1
Random Forest	0.728 ± 0.077	0.812	0.743–0.878	0.691	0.933	0.637	0.523
XGBoost	0.710 ± 0.096	0.795	0.722–0.864	0.630	0.967	0.556	0.487
Logistic Regression	0.596 ± 0.084	0.745	0.643–0.838	0.630	0.900	0.570	0.470
SYNTAX >22	0.671 ± 0.051	0.733	0.651–0.810	0.691	0.800	0.667	0.485
SVM	0.668 ± 0.061	0.726	0.634–0.817	0.612	0.933	0.541	0.467
Neural Network	0.522 ± 0.076	0.603	0.476–0.735	0.612	0.733	0.585	0.407

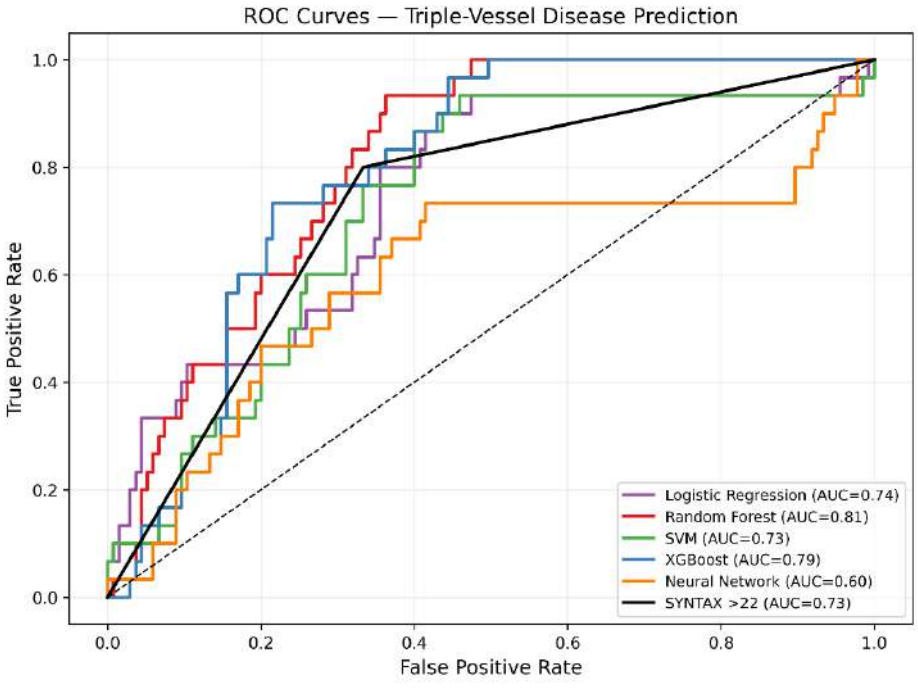

Figure 1. ROC curves of machine learning models and the SYNTAX >22 threshold value for predicting triple-vessel disease.

DeLong AUC Comparison – vs. SYNTAX >22

Statistical comparison of AUC values between each ML model and the SYNTAX >22 reference was performed using the DeLong method (Table 3). Although the Random Forest model exhibited a numerically higher AUC compared to SYNTAX >22 (ΔAUC = +0.079; Z = 1.82), this difference did not reach statistical significance (p = 0.069). Similarly, XGBoost showed a numerical AUC superiority (ΔAUC = +0.061; p = 0.224). None of the pairwise comparisons yielded significance at the p<0.05 level. These findings indicate that the ML models are statistically comparable to the SYNTAX >22 threshold, with Random Forest and XGBoost demonstrating a trend toward superior discriminatory power.

Table 3. DeLong test comparison of the AUC value of each machine learning model against the SYNTAX >22 reference threshold.

Comparison	AUC (Model)	AUC (SYNTAX >22)	ΔAUC	Z	p-value
Random Forest vs. SYNTAX >22	0.812	0.733	+0.079	1.82	0.069
XGBoost vs. SYNTAX >22	0.795	0.733	+0.061	1.22	0.224
Logistic Reg. vs. SYNTAX >22	0.745	0.733	+0.011	0.18	0.857
SVM vs. SYNTAX >22	0.726	0.733	−0.007	−0.13	0.900
Neural Network vs. SYNTAX >22	0.603	0.733	−0.131	−1.60	0.110

Calibration

Calibration curves for the Random Forest and XGBoost models are presented in Figure 2. The Random Forest model demonstrated superior calibration compared to XGBoost, as reflected by a lower Brier score (RF: 0.131 vs. XGBoost: 0.145). Both models exhibited a slight tendency to underestimate event probability in the lower predicted probability ranges, while providing better convergence to the diagonal line at higher probability values.

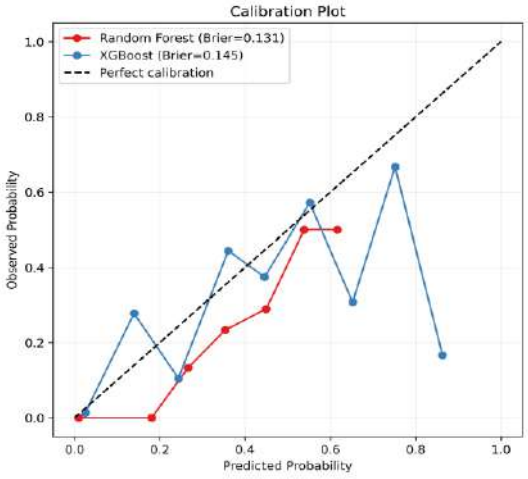

Figure 2. Calibration curves for the Random Forest (Brier = 0.131) and XGBoost (Brier = 0.145) models on the hold-out test set.

Feature Importance Analysis

Variable importance analysis for the Random Forest model identified Hb/Creatinine ratio (importance = 0.064), Peak Troponin I (0.060), WBC/PLT ratio (0.057), diastolic blood pressure (0.056), and heart rate (0.055) as the top five most influential predictors (Figure 3). CK-MB, NLR, SII, AST/ALT ratio, and MLR also contributed significantly to the model decisions.

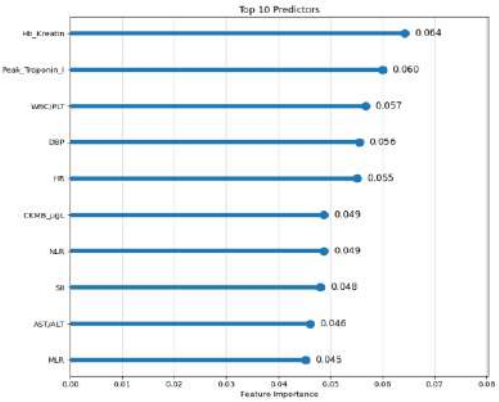

Figure 3. Top 10 predictor variables ranked by Random Forest feature importance scores.

SHAP Analysis

The SHAP beeswarm plot (Figure 4) confirmed that Hb/Creatinine ratio, WBC/PLT, heart rate, diastolic blood pressure, Peak Troponin I, and SpO_2 possessed the greatest impact on model predictions. High values of Hb/Creatinine and WBC/PLT were associated with both positive and negative SHAP contributions, indicating non-linear relationships with triple-vessel disease risk. Higher troponin levels were associated with an increased model output toward triple-vessel disease. STEMI presentation, Age/eGFR ratio, and AST/ALT ratio were also prominent variables in the SHAP ranking.

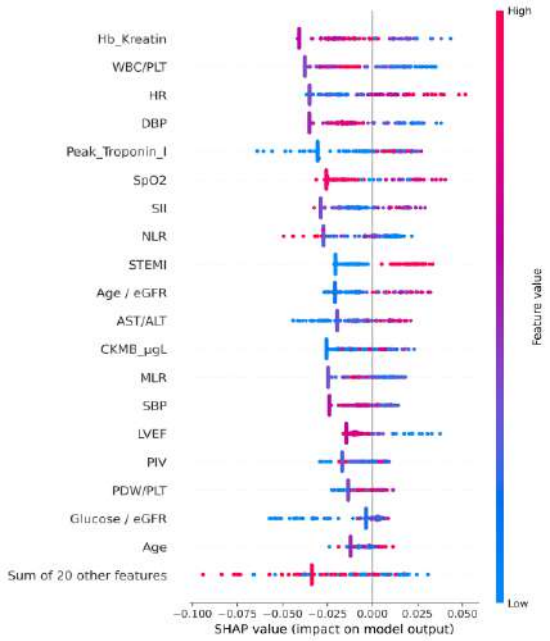

Figure 4. SHAP beeswarm summary plot for the Random Forest model. Features are ranked by mean absolute SHAP value. Color indicates the magnitude of the feature value (red = high, blue = low).

SHAP dependence plots for the six most impactful predictors are presented in Figure 5. The Hb/Creatinine ratio exhibited a non-linear relationship: intermediate values were associated with positive SHAP contributions (increased risk), whereas very low values decreased the model's prediction of triple-vessel disease. The WBC/PLT ratio displayed a monotonically decreasing SHAP pattern; higher values were associated with a lower probability of triple-vessel disease. Heart rate showed a positive monotonic relationship, with higher heart rates linked to increased model output. Diastolic blood pressure exhibited a marked inverse relationship; low DBP values significantly increased the model's triple-vessel prediction. Peak Troponin I followed the expected positive trend, plateauing at higher concentrations. SpO_2 demonstrated an inverse relationship with triple-vessel risk, particularly at values below the median.

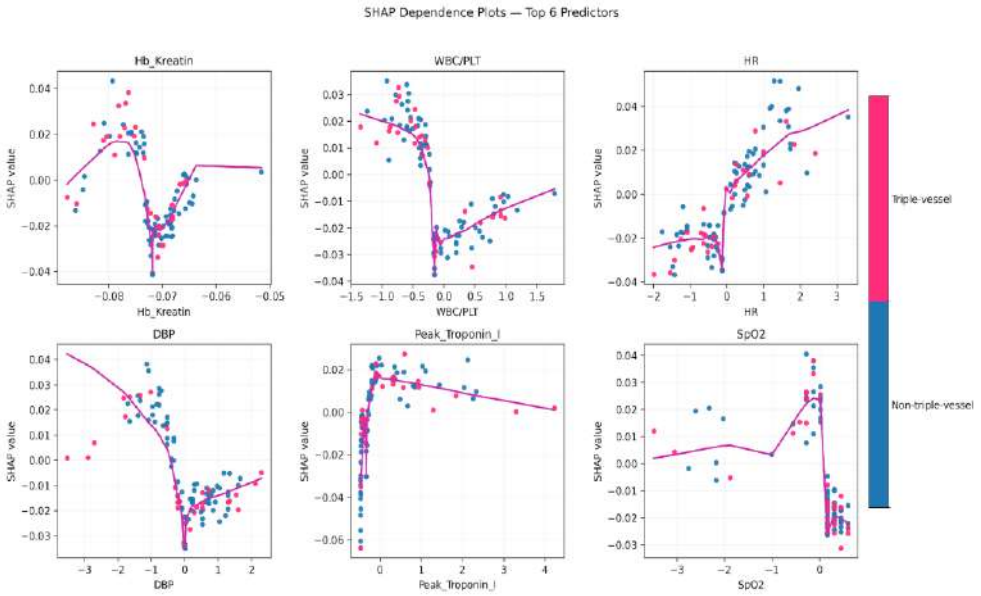

Figure 5. SHAP dependence plots for the six most important predictors. Each dot represents a test-set patient, colored according to their triple-vessel disease status (pink = triple-vessel; blue = non-triple-vessel). The magenta smoothing curve (LOWESS) indicates the overall trend.

CONCLUSION

Machine learning models — particularly the Random Forest algorithm — demonstrated numerically superior discriminatory power compared to the traditional SYNTAX >22 threshold (AUC 0.812 vs. 0.733) in identifying triple-vessel coronary artery disease among ACS patients. However, the DeLong statistical comparison did not reach significance at the conventional level ($p = 0.069$), indicating that the observed difference represents a strong numerical trend rather than a statistically validated superiority. XGBoost exhibited comparable performance with the highest sensitivity (0.967) and a better F1 balance across groups. SHAP analysis identified the Hb/Creatinine ratio, WBC/PLT ratio, peak troponin, heart rate, and diastolic blood pressure as the primary determinants of model predictions; these findings align with established pathophysiological mechanisms in complex CAD. These results suggest that ML-based prediction models utilizing routine clinical and laboratory data may serve as valuable clinical decision support tools for the non-invasive identification of triple-vessel disease in ACS patients, pending prospective validation.

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Extraperitoneal Free Air After Femoral Hemodialysis Catheterization: Bowel Perforation or Benign Complication?

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INTRODUCTION

Femoral hemodialysis catheterization is commonly performed in patients requiring urgent renal replacement therapy, particularly in acute kidney injury superimposed on chronic kidney disease. Although generally safe, mechanical, thrombotic, and infectious complications may occur (1,3). The detection of extraperitoneal free air after femoral vascular access may mimic bowel perforation and pose a diagnostic challenge.

CASE PRESENTATION

A 55-year-old man with diabetes mellitus, hypertension, chronic kidney disease, and coronary artery disease presented with nausea, vomiting, and weakness. Laboratory findings showed glucose 1154 mg/dL, pH 7.15, HCO_3 7.1 mmol/L, potassium 6.79 mmol/L, creatinine 5.93 mg/dL, and hemoglobin 6.7 g/dL, consistent with diabetic ketoacidosis and acute kidney injury on chronic kidney disease.

A temporary right femoral hemodialysis catheter was inserted but replaced with a right internal jugular catheter due to malfunction. Persistent abdominal pain prompted non-contrast abdominal CT, which revealed extraperitoneal free air in the right pelvic region with minimal fluid collection. After surgical consultation, contrast-enhanced CT demonstrated no intraperitoneal perforation or extraluminal contrast leakage. The findings were attributed to the catheterization procedure.

Hemodialysis was initiated, metabolic parameters improved, and the patient was admitted to the intensive care unit. No surgical intervention was required.

DISCUSSION

Complications of femoral hemodialysis catheterization include mechanical (arterial puncture, hematoma, retroperitoneal bleeding, malposition), thrombotic, and infectious events (1,3). Rarely, air may dissect along perivascular tissue planes, leading to extraperitoneal free air without visceral perforation.

True bowel perforation typically presents with peritoneal signs, hemodynamic instability, and contrast extravasation on imaging. In contrast, procedure-related extraperitoneal air is usually seen in stable patients without progressive abdominal findings (4).

In this case, hemodynamic stability, absence of peritoneal irritation, lack of contrast leakage, and stable serial examinations excluded bowel perforation. A multidisciplinary approach prevented unnecessary surgery and enabled conservative management.

In conclusion, extraperitoneal free air after femoral hemodialysis catheter placement does not always indicate bowel perforation. Careful clinical assessment, appropriate imaging, and close monitoring are essential for accurate diagnosis and optimal management.

Keywords: Bowel perforation, Extraperitoneal free air, Femoral hemodialysis catheter

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6164

Hematologic Emergencies in the Emergency Department: Prognostic Role of Physiological Scoring Systems

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Introduction

Hematologic disorders present a heterogeneous patient population in the emergency department (ED), often with nonspecific symptoms and risk of rapid deterioration. Conditions such as anemia, thrombocytopenia, myeloproliferative disorders, and hematologic malignancies may lead to life-threatening complications including infection, bleeding, and

organ failure. Early risk stratification in the ED is therefore critical. Physiological scoring systems are widely used to predict mortality and ICU need (1). Among these, the Modified Early Warning Score (MEWS), Rapid Emergency Medicine Score (REMS), and Shock Index (SI) are commonly applied (2-4). However, their prognostic value in hematologic patients remains limited (5). This study evaluated MEWS, REMS, and SI for predicting short-term mortality and ICU admission in patients presenting to the ED with hematologic conditions.

Methods

A retrospective study was conducted between June 2021 and June 2024 at a tertiary care center, including 400 patients aged ≥18 years with known or newly diagnosed hematologic disorders. Demographics, vital signs, and clinical outcomes were analyzed. MEWS, REMS, and SI were calculated at presentation (3,4). One-month mortality and ICU admission were recorded. Statistical analyses included ROC curves and multivariate logistic regression to identify predictors of poor outcomes.

Results

The mean age was 60.1 ± 17.9 years, and overall mortality was 3.3%. Mortality was highest among patients with chronic leukemia, myelodysplastic syndromes, myeloproliferative disorders, and acute leukemia. Pneumonia or acute hemorrhage significantly increased mortality risk (5). MEWS demonstrated the highest discriminative ability for predicting mortality, while REMS also performed well. SI was not associated with mortality. For ICU admission, MEWS and REMS were strong predictors, whereas SI showed limited utility.

Discussion

Hematologic patients in the ED present heterogeneously, and complications such as infection and bleeding strongly influence outcomes (5). MEWS and REMS were reliable predictors of short-term mortality and ICU need (1,2), with MEWS offering the advantages of simplicity and higher discriminative performance (4). SI alone was insufficient, likely due to the multifactorial nature of deterioration in these patients, including infection, immunosuppression, and organ dysfunction (5). Despite the retrospective, single-center design, these findings support early use of physiological scoring systems to guide risk stratification and clinical decision-making (1). MEWS, in particular, provides a practical tool for early identification of high-risk hematologic patients in ED triage (4).

Keywords: Emergency Department, Hematologic Disorders, ICU, Mortality, MEWS, Prognostic Score, REMS, Shock Index

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6190

Acute Aortic Intramural Hematoma: A Rare Presentation of Acute Aortic Syndrome

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Introduction and Purpose: Acute aortic syndrome (AAS) encompasses life-threatening conditions characterized by acute disruption of the aortic wall integrity, including aortic dissection, intramural hematoma (IMH), and penetrating atherosclerotic ulcer. Intramural hematoma is defined as hemorrhage within the aortic media without an identifiable intimal tear. Type A IMH, involving the ascending aorta, carries a high risk of progression and rupture and often requires urgent surgical intervention. We aimed to present a rare case of Type A IMH diagnosed in the emergency department.

Materials and Methods: A 64-year-old male presented to the emergency department with sudden-onset retrosternal chest pain that began after a meal and persisted at rest. His medical history was significant for hypertension and previous gastric cancer surgery in 2018, with remission since 2017. On admission, he was hemodynamically stable, and physical examination revealed no abnormal findings. Electrocardiography and cardiac biomarkers were not suggestive of acute coronary syndrome. Given persistent chest pain and a history of hypertension, contrast-enhanced thoracic and abdominal computed tomography (CT) angiography was performed to evaluate for AAS.

Image 1: blue arrow intramural hematoma

Image 2: blue arrow intramural hematoma

Results and Conclusion: CT angiography demonstrated circumferential aortic wall thickening extending from the ascending aorta and aortic arch to the distal thoracic aorta without evidence of an intimal flap, consistent with acute Type A IMH. The patient was evaluated by the cardiovascular surgery team and admitted to the intensive care unit for close monitoring and blood pressure control. Due to surgical indication during follow-up, a Bentall procedure was performed.AAS is associated with high mortality and requires rapid diagnosis in the emergency setting. IMH may clinically mimic acute coronary syndrome, particularly in elderly hypertensive patients presenting with chest pain. Contrast-enhanced CT angiography remains the imaging modality of choice due to its high sensitivity and specificity. Early clinical suspicion and prompt imaging are critical to improving outcomes in patients with Type A IMH.

Keywords: Acute aortic syndrome; intramural hematoma; Type A aortic disease

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6209

Life-Saving Role of Emergency Tube Thoracostomy in a Case of Bilateral Pneumothorax Due to Penetrating Thoracic Trauma

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Introduction

Thoracic trauma accounts for approximately 25% of trauma-related deaths (1). Pneumothorax, especially when progressing to tension pneumothorax, can be fatal if not treated promptly (2). Although bilateral pneumothorax is rare, it may present with severe hypoxia and hemodynamic instability (3).

Case Presentation

A 43-year-old male patient was brought to the emergency department hypotensive following penetrating thoracic trauma. On physical examination, breath sounds were absent bilaterally, and bilateral pneumothorax was suspected. Bilateral tube thoracostomy was performed without waiting for imaging (Figure 1, Figure 2, Figure 3). The patient was intubated.

Following tube thoracostomy, the patient's hemodynamic status rapidly improved and stabilization was achieved.

Figure 1. Thoracic CT after tube thoracostomy.

During follow-up, massive hematuria developed.

Figure 2. Left renal laceration.

Figure 3. Splenic hematoma.

The patient was taken to emergency surgery and left nephrectomy was performed.

Discussion

Although bilateral pneumothorax is rare, it is a rapidly progressive and potentially fatal condition if not treated (3). The most critical point in this case was the performance of emergency tube thoracostomy without waiting for imaging. According to ATLS guidelines, intervention should not be delayed in hemodynamically unstable patients (1,2). Tube thoracostomy reduces intrapleural pressure, enables lung expansion, and improves venous return (4). In bilateral pneumothorax cases, this effect is more pronounced and leads to rapid hemodynamic recovery. The literature reports increased mortality in cases with delayed intervention (5). In addition, renal and splenic injuries are common in penetrating trauma and require a multitrauma management approach (6–8).

Conclusion

This case demonstrates that bilateral pneumothorax is a life-threatening condition requiring rapid diagnosis and intervention. In cases of clinical suspicion, tube thoracostomy should be performed without waiting for imaging. Furthermore, associated organ injuries must be carefully evaluated in multitrauma patients. Early and appropriate intervention significantly reduces both mortality and morbidity.

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Keywords: Bilateral pneumothorax, thoracic trauma, tube thoracostomy, nephrectomy, splenic hematoma

6377

The Effects of Fragmented QRS on Mortality and Morbidity in Patients with Chronic Obstructive Pulmonary Disease Presenting to the Emergency Department

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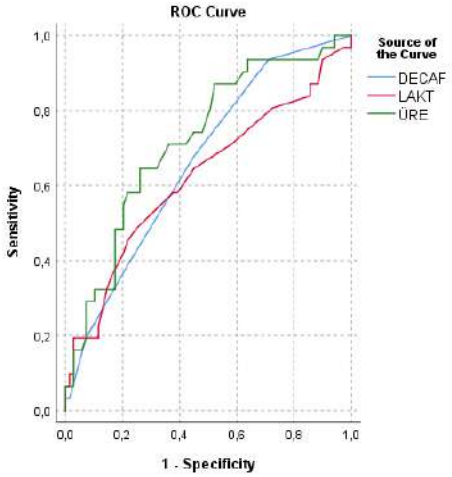
Objective: The aim of this study was to evaluate the prognostic value of clinical, laboratory, and electrocardiographic parameters in predicting clinical outcomes and mortality in patients presenting with acute exacerbation of chronic obstructive pulmonary disease (COPD). In particular, the relationships of arterial blood gas parameters, the DECAF score, and the presence of fragmented QRS (fQRS) with intensive care unit (ICU) requirement and mortality were investigated.

Materials and Methods: This retrospective observational study included patients diagnosed with COPD who were followed between 01.12.2022 and 01.12.2023 at Manisa Celal Bayar University Faculty of Medicine, Hafsa Sultan Hospital. Demographic characteristics, vital signs, arterial blood gas parameters, biochemical and hematological data, electrocardiographic findings, and DECAF scores were recorded. Clinical outcomes were classified as discharge, ward admission, or ICU admission, and 1-year mortality was also evaluated. The discriminative performance of variables for outcomes was analyzed using statistical tests and receiver operating characteristic (ROC) curve analyses.

Results: A total of 100 patients were included in the study. The 1-year mortality rate was 31%, and 20% of patients required ICU admission. Arterial blood gas parameters, particularly pCO_2 and pH, demonstrated strong discriminative ability for predicting ICU admission. The DECAF score showed robust prognostic performance in predicting clinical outcomes and ICU requirement. Patients who died had lower hemoglobin levels and higher urea and lactate levels. In contrast, no significant association was observed between gender, atrial fibrillation, or the presence of fQRS and clinical outcomes or mortality.



Variable	AUC	%95 Confidence Interval	Cut-off	Sensitivity (%)	Specificity (%)	p value
Hemoglobin (g/dL)	0,727	0,615–0,838	≤12,5	80,6	55,1	0,002
			≤10,0	32,3	85,5	
Üre (mg/dL)	0,720	0,613–0,827	≥30,5	90,3	36,2	0,003
DECAF score	0,663	0,546–0,780	≥2	67,7	55,1	0,018
Laktat (mmol/L)	0,623	0,500–0,747	≥1,25	64,5	55,1	0,048

Conclusion: Respiratory physiological deterioration and markers of systemic metabolic stress appear to be the main determinants of clinical course in COPD exacerbations. Combined evaluation of arterial blood gas parameters and the DECAF score provides a practical and effective approach for early prediction of ICU need and mortality risk. The presence of fQRS was found to have limited prognostic value in the acute setting. These findings support the importance of physiology-centered comprehensive risk assessment in the clinical management of COPD exacerbations.

Keywords: Chronic obstructive pulmonary disease, fragmented QRS, electrocardiography, mortality, prognosis

6383

Spontaneous Hemoperitoneum Presenting as Acute Abdomen: A Case Report.

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Introduction:

Spontaneous hemoperitoneum is defined as the presence of blood within the peritoneal cavity in the absence of trauma. It may occur spontaneously or result from the rupture of a known or previously undiagnosed condition and can be serious, potentially leading to rapid death; therefore, early diagnosis is essential for timely treatment (1). Patients often present with nonspecific symptoms such as abdominal pain, nausea, and vomiting, which can mimic more common conditions and delay diagnosis (2). Spontaneous hemoperitoneum most commonly results from the nontraumatic rupture of underlying hepatic, splenic, or abdominal vascular pathologies, particularly in patients with anticoagulation or coagulopathy (3).

We present a case of spontaneous hemoperitoneum in a previously healthy adult male presenting with acute abdominal pain, in whom no definite bleeding source could be identified despite imaging and surgical exploration. This case highlights the diagnostic challenges and the importance of timely evaluation and management in unexplained intraabdominal bleeding.

Case:

A 41-year-old male was brought to the emergency department by emergency medical services with the chief complaint of abdominal pain. The pain had started suddenly two hours prior to admission and radiated to the back. It was associated with nausea and vomiting; the patient reported two episodes of vomiting without hematemesis. The patient stated that he had consumed chicken and rice from outside the previous day, along with three servings of alcoholic beverages. He had one bowel movement, described as loose in consistency, without visible blood. He did not report diarrhea or dysuria during his emergency department stay. There was no history of prior intraabdominal surgery, trauma, pancreatitis, or cholecystitis. He had no history of solid or hematologic malignancy, immunosuppression, or recent international travel. His occupation was a bartender. He had no known chronic medical conditions, no known drug allergies, and was not on any regular medications.

On physical examination, the respiratory system was normal with equal bilateral breath sounds. Cardiovascular examination revealed normal S1 and S2 heart sounds without

murmurs or additional sounds. There was no interarm blood pressure difference, and Homans' sign was negative. The patient was alert, oriented, and cooperative, with a Glasgow Coma Scale (GCS) score of 15. Pupils were isochoric and reactive to light bilaterally. There was no motor deficit, pathological reflex, or abnormality in deep tendon reflexes. Abdominal examination revealed tenderness in both the right and left upper quadrants, with positive guarding and rebound tenderness. Rectal examination was normal. Kehr's sign was negative. Electrocardiography showed normal sinus rhythm, and no changes were observed on serial ECG monitoring. During follow-up, the patient did not develop chest pain or dyspnea. Initial arterial blood gas analysis was within normal limits; however, repeat analysis demonstrated metabolic acidosis with elevated lactate levels. The patient was kept nothing per oral and advised to remain immobilized. A consultation with the general surgery team was requested.

The patient was administered 2 g ceftriaxone, cefuroxime 100 mg twice daily, lactated Ringer's solution, 500 mg intravenous metronidazole, dexketoprofen trometamol 50 mg three times daily, vitamin K 10 mg, tramadol 100 mg, and pantoprazole twice daily. During follow-up, due to the development of hypotension, intravenous fluid resuscitation was initiated.

Initial vital signs were as follows: temperature: 36.1°C, pulse: 77 bpm, oxygen saturation 94% on room air, blood pressure: 94/51mmHg, and blood glucose level: 123 mg/dl. The patient's initial laboratory results were as follows: eGFR: 114, AST:36 U/L, ALT:50 U/L, GGT: 51 U/L, LDH:198 U/L, total bilirubin 0.21mg/dl, direct bilirubin: 0.12 mg/dl, amylase: 80 U/L, Lipase: 28 U/L, CRP <1 mg/L, hemoglobin (HGB): 13.8 g/dl, red blood cells (RBC):4.44 million/ul, white blood cells (WBC): 16 10*3/Ul, platelets (PLT): 268 10*3/Ul, INR:0.87, Anti HBs: 0.96, HBs Ag 0.28 Negative, Anti HIV 0.09 Negative, Anti-HCV 0.08 Negative. Fecal occult blood testing was weakly positive. Stool examination for parasites was negative for Giardia and Entamoeba species. Urinalysis revealed a density of 1.055, trace leukocytes, protein (++), microalbumin positive, and the presence of 4 leukocytes.

Abdominal CT showed a 13 mm parenchymal cyst in segment VII at the dome of the right hepatic lobe. In the upper pole of the spleen, there is a linear hypodense area extending approximately 14 mm in depth, suggestive of a laceration line, possibly secondary to a pseudolesion. Clinical correlation and follow-up are recommended. A 15 mm hyperdense cortical lesion is present in the lower pole of the right kidney, evaluated as consistent with a hemorrhagic cyst. There is widespread high-density (hemorrhagic) free fluid in all peritoneal compartments, including the perihepatic, perisplenic, pelvic regions, and both paracolic gutters. During follow-up, the patient's hemoglobin level decreased from 13.8 g/dL to 11.2 g/dL within the same day. The patient was admitted to the general surgery service.

The patient was taken to the operating room for diagnostic laparoscopy. During exploration, diffuse intra-abdominal blood and clots were seen, so the procedure was converted to open surgery. Blood and clots were found in all four abdominal quadrants and were removed. The

abdomen was washed with warm saline. No active bleeding or injury was found in the liver or spleen. The stomach, small bowel, colon, and rectum were examined, and no bleeding source was identified. Clots in the omentum and mesentery were cleaned, and suspicious areas were coagulated. Since no active bleeding was found, two silicone drains were placed—one toward the pelvis and one toward the splenic area. The abdomen was then closed in layers. The patient was discharged after five days of hospitalization.

Discussion

This case highlights the diagnostic challenges of spontaneous hemoperitoneum presenting as acute abdomen. The patient presented with sudden-onset abdominal pain radiating to the back, accompanied by nausea and vomiting, which initially suggested more common conditions such as acute pancreatitis or gastrointestinal pathology; however, normal laboratory findings, including pancreatic enzymes, made these diagnoses less likely. Imaging revealed diffuse intraabdominal hemorrhagic fluid, raising suspicion for internal bleeding, yet no definitive source was identified. Despite a significant drop in hemoglobin levels and the development of hypotension, surgical exploration failed to demonstrate active bleeding or solid organ injury, suggesting a possible self-limited or low-pressure bleeding source.

Figure 1: Abdominal CT showed a 13 mm parenchymal cyst in segment VII at the dome of the right hepatic lobe. In the upper pole of the spleen, there is a linear hypodense area extending approximately 14 mm in depth, suggestive of a laceration line.

Spontaneous hemoperitoneum is rare and may result from occult vascular injury, minor splenic or mesenteric tears, or rupture of small vessels that may not be detectable intraoperatively. This case emphasizes the importance of close clinical monitoring, repeat laboratory evaluation, and timely surgical intervention in patients with worsening clinical status, and patients may have favorable outcomes with appropriate supportive care and surgical management.

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Crepitation Over Uvula Edema: Diagnostic Pitfalls in Post-Cough Facial Swelling

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INTRODUCTION

Anaphylaxis is a rapidly developing and potentially life-threatening systemic reaction, representing one of the most time-critical clinical conditions encountered in emergency departments. However, certain clinical entities—particularly those presenting with facial and neck swelling—may mimic anaphylaxis, leading to misdiagnosis and inappropriate management.

Pneumothorax is characterized by the accumulation of air within the pleural space, resulting in lung collapse. Secondary spontaneous pneumothorax, which occurs in the presence of underlying lung disease, is associated with a more severe clinical course and higher mortality (1,2).

Pneumomediastinum occurs when air, following alveolar rupture, dissects along the bronchovascular sheaths into the mediastinum. The extension of this air into the cervical fascial planes may lead to subcutaneous emphysema, producing prominent swelling in the face and neck (3,4). This pathophysiological mechanism is defined as the “Macklin effect” (5).

CASE PRESENTATION

A 73-year-old male patient was referred to the emergency department from an outside facility with a preliminary diagnosis of anaphylaxis. His history revealed the sudden onset of left periorbital swelling following two episodes of severe coughing on the same night. Intramuscular adrenaline had been administered at the referring center due to suspected uvular

edema.

His medical history was notable for wedge resection of the left upper and lower lobes performed three months prior, with a pathological diagnosis of lung adenocarcinoma. This condition placed the patient at high risk for secondary spontaneous pneumothorax. Upon admission, oxygen saturation was measured at 87%. Physical examination revealed extensive subcutaneous emphysema over the left hemithorax, neck, and facial regions, with palpable crepitation. Marked swelling was observed in the left periorbital area (Figure 1). Although the oropharyngeal examination demonstrated an edema-like appearance that could initially be interpreted as consistent with anaphylaxis, the presence of crepitation suggested an air-related etiology.

Thoracic computed tomography (Figure 2) demonstrated widespread subcutaneous emphysema, left-sided pneumothorax, and pneumomediastinum. Based on these findings, the patient was consulted with thoracic surgery and admitted to the intensive care unit.

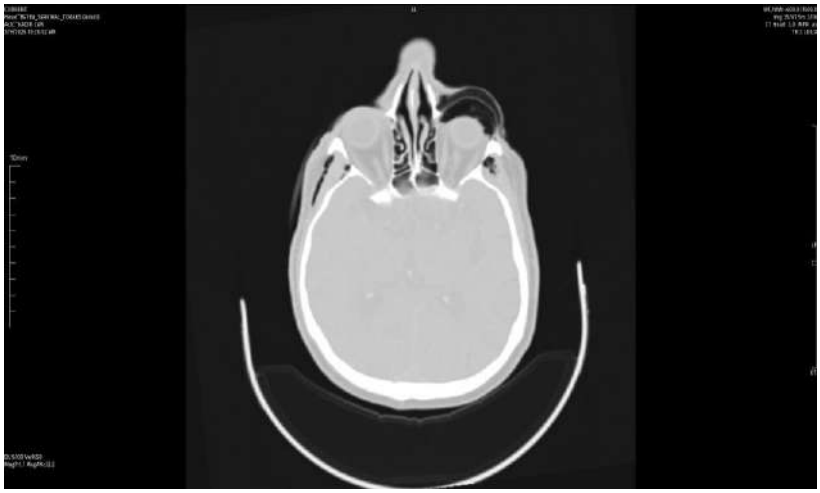
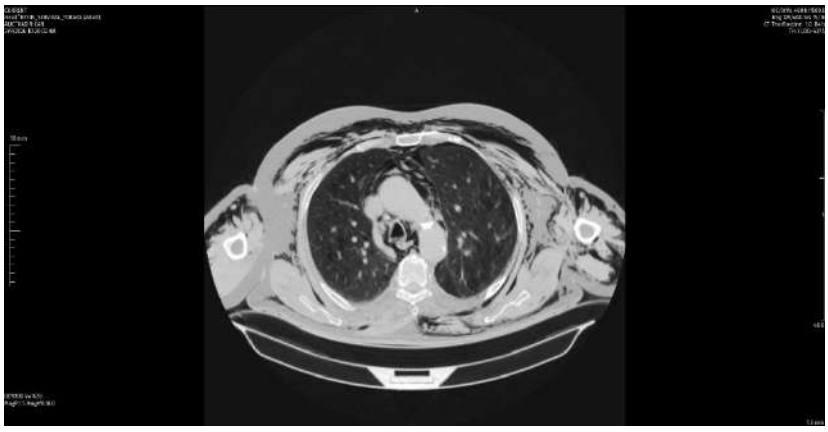


Figure 1 Computed tomography image demonstrating swelling in the left periorbital region



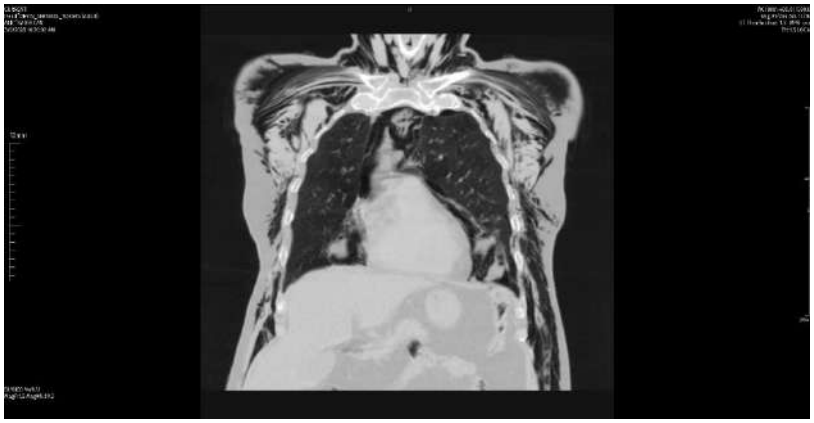


Figure 2 Computed tomography image showing subcutaneous emphysema, left pneumothorax, and pneumomediastinum

DISCUSSION

Secondary spontaneous pneumothorax is more frequently observed in elderly patients and those with malignancy or a history of thoracic surgery, and it typically follows a more severe clinical course (1,2). Current guidelines emphasize lower thresholds for interventional treatment and hospital admission in this patient population (1).

The Macklin effect, the primary mechanism underlying pneumomediastinum, is explained by increased alveolar pressure leading to alveolar rupture and subsequent air dissection along interstitial planes (5). Severe coughing is one of the most important triggering factors in this process.

Subcutaneous emphysema may cause dramatic swelling, particularly in the face and neck, and can be mistaken for anaphylaxis or angioedema. However, unlike anaphylaxis, the presence of crepitation is a critical physical examination finding that aids in differentiation (3,4).

This case illustrates how diagnostic pitfalls during the initial evaluation in the emergency department can significantly impact clinical management. Treatment administered under the preliminary diagnosis of anaphylaxis delayed the recognition of the underlying pathology.

Moreover, current literature highlights that early diagnosis and appropriate management of secondary pneumothorax are crucial determinants of prognosis (2). Therefore, rapid identification of risk factors and meticulous physical examination are of paramount importance in emergency practice.

CONCLUSION

Although anaphylaxis is often the primary consideration in patients presenting with acute facial and neck swelling in the emergency department, air leak syndromes must be included in the differential diagnosis, particularly in individuals with relevant risk factors. The presence of crepitation and a detailed clinical history are critical for accurate diagnosis. Early recognition and appropriate management are essential to prevent unnecessary treatments and to improve patient outcomes.

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Keywords: Pneumothorax, pneumomediastinum, subcutaneous emphysema, anaphylaxis, emergency medicine

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Beyond Venous Thrombosis: A Fatal Case of Trousseau Syndrome Presenting with Multiorgan Arterial Infarction

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Introduction

Hypercoagulability is a condition that causes blood to form clots easier and increases the likelihood of thrombosis. This condition emerges when innate coagulation systems weaken or when fibrinolysis is impaired (1,2). Causes of hypercoagulability can be summarized as; hereditary thrombophilia conditions (Factor V Leiden mutation, prothrombin gene mutation; antithrombin III, protein C and protein S deficiencies) and acquired causes (malignities, pregnancy and postpartum period, oral contraceptive usage, immobilization, surgery/trauma, antiphospholipid syndrome, obesity and cigarette use). Malignity associated hypercoagulability is caused by tumor cells secreting tissue factors and procoagulant substances which dramatically increase clotting. Additionally, thrombocyte activation, fibrinolysis inhibition and releasing of tumor related microparticles also facilitates thrombosis (1,3)

Trousseau syndrome is a paraneoplastic hypercoagulable condition characterized by migratory thrombophlebitis and is most commonly associated with pancreatic cancer. Additionally; stomach, lung, colon and other gastrointestinal malignities can also cause this syndrome (4-6). Clinical presentation of intestinal malignancies usually start with nonspecific symptoms such as stomachaches, weight loss, loss of appetite, anemia and changes in defecation. Intestinal obstructions, bloating and vomiting can be seen in advanced stages; venous thrombosis or recurrent thromboemboli can be the first finding in some patients (4,7).

Vitamin K antagonists, low molecular weight heparin and direct oral anticoagulants are used in the treatment of malignancy associated thrombosis. Low molecular weight heparin is the first choice among these as its half-life is shorter and causes a lower risk of bleeding. Although factor Xa inhibitors are more effective in reducing the risk of venous thromboembolism, they are not preferred as first-line therapy because they increase the risk of bleeding, particularly in gastrointestinal malignancies.

This case report aims to highlight that gastrointestinal malignancies can present with thrombosis and that anticoagulant therapy may be insufficient in certain cases.

Case

Fifty-six-year-old male patient, presented to ER with severe stomach pain that started 2 hours ago. No known chronic diseases but had history of deep vein thrombosis. It was noted that the patient was on regular treatment with antiplatelet agents (cilostazol, acetylsalicylic acid), a venotonic (calcium dobesilate), and a direct oral anticoagulant (apixaban). Vital signs on arrival to ER were evaluated as; blood pressure of 140/80mmHg, pulse of 82/min, body temperature of 36,6 °C, oxygen saturation of %99 on room air and Glasgow Coma Score (GCS) of 15. Physical exam findings on arrival were; diffuse abdominal tenderness with no sign of guarding or rebound tenderness, head and neck examination revealed no abnormalities, the pupils were isochoric and reactive to light, no additional heart sounds, murmurs, rales, or rhonchi were detected on cardiovascular and respiratory examination, no edema or deformity was observed in the extremities, neurological examination was unremarkable, with normal motor and sensory function and no cranial nerve deficits, no skin rash or lesions were observed. Bedside echocardiography revealed no thrombus in the atria or ventricles, while bedside abdominal ultrasonography demonstrated hypoechoic areas in the splenic parenchyma that could be suggestive of infarction, as well as multiple cystic lesions in the liver. During emergency department management, intravenous fluid resuscitation was initiated (a total of 1500 cc of %0.9 NaCl). Despite repeated doses of narcotic analgesics for pain control (4 doses of 50 µg fentanyl and 4 doses of 2 mg morphine), the patient's pain did not subside.

Laboratory investigations revealed inflammation and leukocytosis (WBC 25 × 10^9/L, CRP 81 mg/L). Mild elevations in liver enzymes (AST 42 U/L, ALT 61 U/L) and an increase in LDH (637 U/L) were also observed. D-dimer was 33 µg/mL (cut-off: 0–0.5 µg/mL), and Troponin I was 1.9 ng/mL (cut-off: 0–0.06 ng/mL). Other laboratory values were as follows: hemoglobin 11 g/dL, hematocrit 33.7%, platelet count 109 × 10^9/L, INR 1.19, prothrombin time 14.3 seconds, and lactate 3.83 mmol/L on venous blood gas analysis.

Despite the patient's severe abdominal pain, the absence of guarding or rebound tenderness on examination, along with marked abnormalities in laboratory findings and findings on bedside abdominal ultrasonography, prompted a contrast-enhanced abdominal computed tomography (CT) scan. On CT imaging, diffuse patchy hypodense areas consistent with splenic infarction were identified in the splenic parenchyma (Figure 1).

Figure 5

With these findings, the patient was admitted to the ward with a diagnosis of splenic infarction. During the initial evaluation, considering the possibility of an acute abdomen and the potential need for emergency surgery, no additional anticoagulants were us due to the risk of bleeding, as the patient had already taken apixaban earlier that day.

During follow-up, the patient developed a marked increase in abdominal pain along with clear signs of an acute abdomen. Consequently, the patient was taken for emergency laparotomy; intraoperatively, occlusion of the superior mesenteric artery was identified, and the small bowel loops and spleen appeared ischemic. In addition, multiple firm nodules were observed in the liver, and an approximately 2 cm ulcerative, firm mass was detected in the jejunal region. In the postoperative period, the patient was followed in the intensive care unit under inotropic support and died on postoperative day 3.

Discussion

In our case, despite the patient being on regular direct oral anticoagulant treatment (apixaban), acute splenic infarction and massive mesenteric ischemia was developed. These multiple arterial thromboses of unclear etiology were later found to be caused by an underlying “occult malignancy”, as evidenced by the jejunal mass and multiple hepatic metastatic nodules detected during emergency surgery.

This aggressive and resistant thrombotic state despite anticoagulant therapy can be explained by the pathophysiological effects of tumor cells. Tissue factor and procoagulant substances secreted by cancer cells directly and strongly activate the coagulation cascade, leading to thrombosis that is resistant to standard treatments (1,3). A review of the literature reveals that cases of unexplained arterial thrombosis as the first manifestation of pancreatic, lung, or gastrointestinal malignancies are reported(4,7). In such resistant hypercoagulable states, low-molecular-weight heparin (LMWH) is considered a more effective and reliable treatment option in the management of malignancy-associated thrombosis (8).

Unexplained arterial thromboses occurring despite regular anticoagulant therapy should be regarded as an important clinical warning sign of paraneoplastic hypercoagulability (Trousseau syndrome). It should be kept in mind that this syndrome may present not only with venous thrombosis but also with highly lethal and aggressive multiorgan arterial ischemia as in our case, and malignancy screening should be promptly performed in cases of unexplained, anticoagulant-resistant thrombosis.

Recent studies have shown that although the most common causes of arterial thrombotic events such as splenic infarction and acute mesenteric ischemia are cardioembolic, paraneoplastic conditions should also be considered (4,7,9,10). There are reports of pancreatic and lung cancers presenting with splenic infarction or mesenteric artery occlusion as the first finding, and it has been emphasized that Trousseau syndrome may involve a broad spectrum of manifestations beyond venous thrombosis, including multiorgan arterial infarctions (5). Consistently, large cohort studies have demonstrated a significant increase in arterial thromboembolic events in the months preceding a cancer diagnosis, supporting the need for malignancy screening in unexplained arterial thrombosis (9,10). In this context, the coexistence of splenic infarction and superior mesenteric artery occlusion revealing a newly diagnosed intestinal malignancy is a rare presentation consistent with this paraneoplastic mechanism described in the literature.

Conclusion

Trousseou syndrome should be considered in cases with recurring arterial thrombosis and multiple organ ischemia. Splenic enfarct can be an early finding of this syndrome. Occult malignities should be investigated even in patients without diagnosis of malignancy, low molecule weight heparin should be preferred in treatment.

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The Deadly Progression: Autonomic Instability as a Fatal Complication of Guillain-Barré Syndrome

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INTRODUCTION

Guillain-Barré Syndrome (GBS) is an acute, inflammatory, autoimmune demyelinating polyneuropathy characterized by rapidly progressive, ascending (from lower to upper extremities), symmetrical muscle weakness and areflexia (1,2). Although the etiopathogenesis of the disease is not fully understood, in most cases there is a history of an upper respiratory tract or gastrointestinal infection occurring 1–4 weeks before the onset of symptoms (1,5). During this process, antibodies produced by the body against the infectious agent cross-react with the myelin tissue of peripheral nerves due to molecular mimicry, resulting in nerve damage (1). The annual incidence of GBS ranges from 0.6 to 2.4 per 100,000 population and it can occur in all age groups (3). The clinical presentation typically begins with paresthesia in the fingertips and weakness in the legs; within hours or days, this condition may progress to involve the arms, face, and oropharyngeal muscles (1). One of the most characteristic findings in the diagnostic process is an elevated protein level in cerebrospinal fluid (CSF) analysis without an increase in cell count, a condition defined as "albuminocytologic dissociation"

(1,6). Cranial nerve involvement is observed in 45–75% of patients with GBS, with the facial nerve being the most commonly affected, whereas involvement of the twelfth cranial nerve (hypoglossal nerve) is extremely rare (1). Approximately half of GBS cases follow a severe course and require intensive care monitoring because of life-threatening respiratory failure or autonomic instability (2,4).

CASE PRESENTATION

A 47-year-old male patient with no known comorbidities or history of medication use was brought to the emergency department with complaints of numbness and tingling in the upper extremities that had begun 3 days earlier, followed by progressive weakness in both the upper and lower extremities and shortness of breath. These symptoms were preceded by generalized fatigue and malaise that had started approximately 7 days before presentation. Initial vital signs in the emergency department were as follows: blood pressure 130/80 mmHg, SpO_2 95%, heart rate 90 beats/min, and body temperature 37°C. Central nervous system imaging performed in the emergency department (non-contrast cranial, cervical, thoracic, and lumbar MRI) revealed no acute neuropathology. On initial examination, the patient was conscious, oriented, cooperative, and had normal speech. Neurological examination revealed muscle strength of 2–3/5 proximally and 3–4/5 distally in both upper extremities, 2–3/5 proximally and 3/5 distally in the lower extremities, and deep tendon reflexes (DTRs) were absent. The patient was admitted to the Neurology Department with a preliminary diagnosis of GBS. Following admission, dysphagia developed. Lumbar puncture (LP) demonstrated elevated CSF protein levels, and plasmapheresis treatment was initiated. On the second day of hospitalization, the patient developed diplopia. Brain computed tomography (CT) and diffusion-weighted magnetic resonance imaging (MRI) were repeated; however, no acute pathology was detected. During follow-up, increased secretions and worsening dysphagia were observed. On the fourth day, the patient developed deterioration in consciousness and tachypneic respiration. Arterial blood gas analysis revealed severe respiratory acidosis, and the patient was intubated and connected to mechanical ventilation with low-dose sedation initiated. Vasopressor support was administered following the development of hypotension, and the clinical picture was considered to represent autonomic involvement of GBS. The patient remained intubated in the Neurology Intensive Care Unit for 54 days and ultimately died following cardiac arrest despite all resuscitative interventions.

DISCUSSION

Due to its clinical heterogeneity, Guillain-Barré Syndrome is often difficult to recognize, particularly in its early stages. The literature reports that the diagnosis of GBS may initially be missed or misdiagnosed in up to 25% of cases (6). In our case, the completely normal central nervous system imaging studies obtained at presentation represented a potentially misleading factor that could have led clinicians to exclude the diagnosis radiologically. However, absent

deep tendon reflexes and progressive motor weakness should always prompt consideration of GBS in the differential diagnosis. In the present case, the onset of symptoms with numbness in the upper extremities rather than the typical ascending pattern suggests atypical GBS variants (1). In a similar case reported in the literature, a 34-year-old patient presented with asymmetric upper extremity involvement and the rare finding of twelfth cranial nerve (hypoglossal nerve) palsy (1). The dysphagia and increased secretions observed in our patient also indicate the severity of cranial nerve involvement and the loss of airway protective reflexes. Cranial nerve involvement in GBS is generally associated with a poorer prognosis and more severe disease course (1,4). One of the most critical aspects of our case is the role of elevated CSF protein levels (albuminocytologic dissociation) in confirming the diagnosis. This finding is considered a highly specific indicator for GBS (1,7). However, despite timely initiation of plasmapheresis treatment, progression to autonomic dysfunction demonstrates how aggressive GBS can sometimes be. Autonomic involvement is one of the most feared complications of GBS, as it may result in fatal outcomes such as arrhythmias, blood pressure instability, and cardiac arrest (4,7). Although early diagnosis and prompt initiation of immunotherapies such as plasmapheresis or intravenous immunoglobulin (IVIG) reduce mortality in GBS, the risk of respiratory failure and autonomic paralysis remains in fulminant cases such as ours (4). The prolonged 54-day intensive care course likely reduced the patient's systemic resilience, and death ultimately occurred due to cardiac complications triggered by autonomic involvement.

CONCLUSION

This case emphasizes that GBS should always be considered in patients presenting with progressive muscle weakness and respiratory distress, even when radiological imaging findings are normal. Early CSF analysis is of critical importance for diagnosis. GBS is not merely a motor disorder but also a systemic emergency that may be accompanied by life-threatening autonomic involvement. Therefore, patients diagnosed with GBS should be closely monitored in intensive care settings for autonomic instability and sudden cardiac complications, which is essential for improving prognosis.

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Suspected Recurrent Myocarditis Presenting as Acute Decompensated Heart Failure After Viral Prodrome in a Young Woman: A Case Report

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Abstract

Myocarditis is an inflammatory disease of the myocardium with a broad clinical spectrum ranging from mild symptoms to fulminant heart failure and cardiogenic shock. Viral infections remain among the most common etiological triggers and may induce myocardial injury through both direct cytotoxic effects and immune-mediated inflammatory mechanisms. We report the case of a 28-year-old woman with a previous history of myocarditis who presented to the emergency department with acute onset dyspnea following a recent viral upper respiratory tract infection. On admission, oxygen saturation on room air was 82%, heart rate was 110 beats/min, and blood pressure was 119/70 mmHg. Pulmonary auscultation revealed bilateral fine crackles. Laboratory testing demonstrated markedly elevated troponin levels (11,597 pg/mL), creatinine 2.13 mg/dL, AST 394 U/L, and ALT 442 U/L. Chest radiography demonstrated pulmonary edema. Transthoracic echocardiography revealed severe left ventricular systolic dysfunction with an ejection fraction of 25-30% and segmental wall motion abnormalities. The patient was admitted to the intensive care unit with a preliminary diagnosis of acute decompensated heart failure associated with suspected recurrent or post-viral myocardial inflammatory involvement. This case highlights the importance of

considering myocarditis and myocarditis-related heart failure in the differential diagnosis of acute dyspnea in young patients presenting after a viral prodrome.

Keywords: myocarditis; acute decompensated heart failure; viral prodrome; emergency department; case report

Introduction

Myocarditis is an inflammatory disease of the myocardium characterized by a heterogeneous clinical presentation ranging from asymptomatic biomarker elevation to malignant arrhythmias, acute heart failure, and cardiogenic shock [1-3]. Recent reviews and consensus documents emphasize that myocarditis should be strongly suspected in young patients presenting with unexplained dyspnea, chest pain, elevated troponin levels, and left ventricular systolic dysfunction [1-3]. Viral infections may trigger myocardial inflammation through direct viral cytotoxicity and immune-mediated mechanisms [1,4]. A recent history of upper respiratory tract infection may therefore provide an important clinical clue in patients presenting with acute cardiac deterioration [1,4]. Early recognition in the emergency department is essential for timely initiation of supportive therapy, hemodynamic monitoring, bedside echocardiographic assessment, and appropriate intensive care planning [1-3].

Case Presentation

A 28-year-old woman presented to the emergency department with sudden-onset dyspnea that developed earlier that morning. Her medical history revealed a previous episode of myocarditis diagnosed approximately five months earlier. The patient reported symptoms of upper respiratory tract infection, including fever, nasal discharge, and malaise, during the preceding 10 days. On admission, oxygen saturation on room air was 82%, heart rate was 110 beats/min, blood pressure was 119/70 mmHg, and body temperature was 36.2 C. Electrocardiography demonstrated normal sinus rhythm. Pulmonary auscultation revealed bilateral fine crackles, whereas peripheral edema was absent. The patient appeared diaphoretic.

Laboratory evaluation demonstrated marked leukocytosis and substantial cardiac biomarker elevation: white blood cell count 35.96 x10^3/uL, neutrophils 32.78 x10^3/uL, lymphocytes 1.78 x10^3/uL, glucose 144 mg/dL, creatinine 2.13 mg/dL, urea 42.8 mg/dL, AST 394 U/L, ALT 442 U/L, creatine kinase 1707 U/L, CK-MB 67.7 U/L, LDH 1266 U/L, troponin 11,597 pg/mL, and C-reactive protein 2.36 mg/dL. Arterial blood gas analysis revealed metabolic acidosis with a pH of 7.29, pCO2 of 40.7 mmHg, bicarbonate of 17.9 mmol/L, and oxygen saturation of 80.7%.

Posteroanterior chest radiography demonstrated bilateral, predominantly perihilar and lower-zone opacities compatible with pulmonary edema (Figure 1). Transthoracic echocardiography revealed severe left ventricular systolic dysfunction with an ejection fraction of 25-30%,

accompanied by segmental wall motion abnormalities and no major valvular pathology. Considering the clinical, laboratory, and imaging findings together, acute decompensated heart failure associated with suspected recurrent or post-viral myocardial inflammatory involvement was considered the leading diagnosis. After cardiology consultation, the patient was admitted to the intensive care unit for further management.

Figure 1. Posteroanterior chest radiograph obtained at presentation demonstrating bilateral, predominantly perihilar pulmonary edema.

Discussion

This case is notable for several reasons. First, although primary pulmonary causes are often considered initially in young patients presenting with abrupt dyspnea, the combination of marked troponin elevation, severe left ventricular systolic dysfunction, and radiographic evidence of pulmonary edema shifted the diagnostic focus toward a cardiac etiology. Contemporary reviews and major cardiology documents indicate that myocarditis does not conform to a single clinical pattern and may present with dyspnea, chest pain, palpitations, syncope, heart failure, or cardiogenic shock [1-3]. Accordingly, myocarditis should be considered in the emergency department when unexplained dyspnea is accompanied by elevated cardiac biomarkers, particularly in younger patients [1-3].

Second, the recent viral prodrome in this patient is clinically important. Viral infections may provoke myocardial inflammation through direct myocardial injury, immune activation, and cytokine-mediated pathways [1,4]. In susceptible individuals, this inflammatory process may lead to progressive systolic dysfunction and acute decompensation over days to weeks [1,4]. However, in the absence of cardiac magnetic resonance imaging or endomyocardial biopsy, definitive etiological confirmation was not possible in the present case. The diagnosis therefore relied on an integrated interpretation of the clinical presentation, biomarker profile, and echocardiographic findings [2,3]. For this reason, the most appropriate diagnostic formulation is suspected recurrent myocarditis or post-viral myocardial inflammatory involvement rather than a definitive etiological label.

Third, the differential diagnosis warrants careful consideration. Severe pneumonia, sepsis-associated myocardial dysfunction, decompensated non-inflammatory dilated cardiomyopathy, acute coronary syndrome, and toxic-metabolic disorders may all present with a similar clinical picture. Nevertheless, the patient's young age, prior history of myocarditis, recent viral illness, marked troponin elevation, and severe left ventricular systolic dysfunction together strengthened the likelihood of myocarditis-related acute heart failure. Current guidelines emphasize the central role of echocardiography and cardiac magnetic resonance in the diagnostic algorithm, while recommending endomyocardial biopsy for selected high-risk situations [2,3].

Fourth, time-sensitive management in the emergency department is crucial. In patients with hypoxemia and acute heart failure, prompt correction of oxygenation, initiation of hemodynamic monitoring, concurrent cardiology assessment, and early advanced-care planning may directly influence prognosis. Both ESC and ACC documents emphasize the need for early advanced imaging, rhythm surveillance, and intensive monitoring in suspected myocarditis accompanied by heart failure or hemodynamic compromise [2,3]. In the present case, early echocardiography identified severe systolic dysfunction and facilitated rapid intensive care planning.

Conclusion

In young patients presenting with acute dyspnea after a viral prodrome, particularly those with a previous history of myocarditis, recurrent myocarditis or post-viral myocardial inflammatory involvement should be included in the differential diagnosis. Troponin measurement, chest radiography, bedside echocardiography, and close hemodynamic assessment are key elements for early recognition of cardiac causes of respiratory distress [1-3]. This case underscores the critical importance of rapid evaluation and multidisciplinary management in myocarditis-related acute decompensated heart failure.

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Investigation Of The Prognostic Value Of The ONCALL Score In The Clinical Decision-Making Process For Oncology Patients Presenting To The Emergency Department

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Objective: The continuity of demand for healthcare services and overcrowding in emergency departments necessitate optimizing early and safe discharge processes for low-risk oncology patients or making rapid admission decisions for high-risk patients. The aim of this study is to develop a scoring system to optimize hospitalization decision-making processes for oncology patients presenting to the emergency department.

Methods: This retrospective, single-center study was conducted on patients aged 18 and over diagnosed with malignancy who presented to the Bezmiâlem Vakıf University Emergency Department between January 1, 2021, and December 31, 2023. Demographic data, vital signs, treatment histories, and laboratory parameters were recorded to develop the ONCALL score, aimed at predicting the risk of "hospitalization," which includes hospital admission (ward/intensive care) and mortality. In constructing the scoring system, 13 predetermined potential parameters were evaluated first by univariate and then by multivariate logistic regression analyses. As a result of statistical analyses, the multivariate regression model (Model 3), comprising **O**ncologic diagnosis type (hematologic/solid), **N**eutropenia (ANC <1000/mm³), High **C**reatinine (>1.30 mg/dL), **A**ge (≥65 years), **L**ow Blood Pressure (Hypotension), and **L**ast 48h Chemo (Chemotherapy within the last 48 hours), was identified as the model with the highest discriminatory power (AUC: 0.720). The model's performance and predictive power were tested using ROC analysis, and all analyses were performed within a 95% confidence interval using MedCalc® software.

Results: A total of 285 patients with a mean age of 63.8 ± 13.4 years, 55.8% of whom were male, were included in the study. The most common reasons for presentation were gastrointestinal (41.3%) and respiratory (14.5%) symptoms, while digestive (24.9%), respiratory (22.5%), and hematopoietic (20.4%) system malignancies were prominent among primary diagnoses. A history of metastasis was present in 62% of patients, and additional chronic diseases in 72.5%; hypotension was detected in 19.3% and elevated creatinine in 23.9% at the time of presentation. When clinical outcomes were evaluated, it was determined that the vast majority of patients (73.7%) were hospitalized (ward 48.4%, intensive care 25.3%), only 22.5% were discharged, and the total in-hospital mortality rate was 28.4%.

Conclusion: The ONCALL score, developed with bedside parameters, is a practical tool for identifying the high-risk group requiring hospitalization among emergency oncology patients. Multicenter prospective studies are needed to increase the clinical validity and generalizability of the model.

Keywords: Emergency department, Oncology, Cancer, Risk Stratification, Score, Prognostic

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THE EFFECT OF EMERGENCY DEPARTMENT BOARDING TIME ON INTENSIVE CARE UNIT CLINICAL OUTCOMES: A SINGLE-CENTER RETROSPECTIVE COHORT STUDY

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ABSTRACT

Background/Objectives: Emergency department (ED) boarding — the time critically ill patients wait in the ED before transfer to an intensive care unit (ICU) — is a growing patient safety concern worldwide. This study aimed to evaluate the impact of ED boarding time on

ICU length of stay and in-hospital mortality, and to identify independent predictors of delayed ICU transfer.

Methods: This single-center retrospective cohort study included all adults aged ≥18 years admitted to any ICU (medical, surgical, or resuscitation) from the ED of Ankara Etlik City Hospital between March 2025 and February 2026. Patients transferred directly to the ICU from the operating room, those bypassing the ED, and those with incomplete records were excluded. Patients were stratified into early transfer (<6 hours) and delayed transfer (≥6 hours) groups.

Results: Of 2029 patients screened, 2005 were included in the final analysis. The median ED boarding time was 18.6 hours (IQR: 8.5–34.2). Overall in-hospital mortality was 34.5%. Mortality was significantly higher in the delayed transfer group (36.0% vs. 30.3%; p=0.016), and ICU length of stay was markedly longer (8 vs. 5 days; p<0.001). On multivariable logistic regression, ED boarding ≥6 hours was an independent predictor of mortality (OR: 1.46; 95% CI: 1.18–1.81; p=0.001). ROC analysis identified an optimal cut-off of 11.5 hours (AUC=0.583; sensitivity 64.2%; specificity 51.8%).

Conclusions: Prolonged ED boarding is independently associated with increased in-hospital mortality and longer ICU stay among critically ill patients. These findings from a high-volume tertiary center underscore the urgent need to optimize ED-to-ICU transition processes at the national level.

Keywords: emergency department boarding, ED overcrowding, intensive care unit, mortality, retrospective cohort

1. INTRODUCTION

Emergency department (ED) boarding — defined as the period during which critically ill patients requiring intensive care remain in the ED while awaiting ICU transfer — has emerged as a major global patient safety challenge. Rising ED census volumes, finite ICU capacities, and structural constraints within health systems have collectively led to critically ill patients being held in ED environments lacking adequate monitoring and life support infrastructure [1,2]. The problem is particularly pronounced in high-volume tertiary hospitals, where the gap between critical care demand and available ICU beds is widest.

A robust body of evidence links prolonged ED boarding to adverse clinical outcomes. Chalfin et al., in a landmark multicenter analysis of over 50,000 patients, demonstrated that delayed ICU transfer independently increased in-hospital mortality and ICU length of stay; a boarding duration of 6 hours emerged as the clinically meaningful threshold separating acceptable from harmful delay [3]. Subsequent investigations by Howell et al. and Bing-Hua et al. corroborated these findings, showing a consistent positive correlation between boarding time, mortality, and resource utilization [4,5]. Reznek et al. further identified vulnerable patient

subgroups — including those with sepsis and respiratory failure — in whom the mortality impact of boarding was disproportionately large [7].

In Turkey, the transition to large-capacity city hospital campuses has created high-volume emergency services that may be particularly susceptible to boarding-related harm. Ankara Etlik City Hospital is one of the country's largest tertiary referral centers in terms of both daily ED census and ICU bed capacity. Despite the recognized importance of this issue globally, real-world data from Turkish tertiary centers on the clinical consequences of ED boarding remain scarce.

This study aimed to (1) quantify the prevalence and duration of ED boarding in a high-volume Turkish city hospital, (2) evaluate the independent effects of prolonged boarding on in-hospital mortality and ICU length of stay, and (3) determine the optimal boarding time threshold for predicting mortality.

2. MATERIALS AND METHODS

2.1. Study Design and Ethics

This single-center, retrospective, observational cohort study was conducted at Ankara Etlik City Hospital (Ankara, Turkey). Institutional ethics committee approval is ongoing, and the study was conducted in accordance with the Declaration of Helsinki. Given the retrospective and non-interventional nature of the study, obtaining individual patient consent was not deemed necessary.

2.2. Study Population

All consecutive adult patients (≥18 years) presenting to the ED and subsequently admitted to any ICU — medical ICU, surgical ICU, or resuscitation ICU — between March 2025 and February 2026 were considered for inclusion. Exclusion criteria were: (1) direct ICU transfer from the operating theater without ED passage; (2) ED bypass; and (3) incomplete or implausible data records. After excluding 24 patients on these grounds, 2005 patients constituted the final analytic cohort.

2.3. Variable Definitions

The primary independent variable, ED boarding time, was calculated as the difference between the ICU admission timestamp and the ED registration timestamp, both extracted from the hospital information management system (HIMS). Consistent with the most widely adopted threshold in the literature, patients were dichotomized into an early transfer group (<6 hours) and a delayed transfer group (≥6 hours). Boarding time was additionally analyzed as a four-level ordinal variable (<6 h, 6–12 h, 12–24 h, ≥24 h) to assess for a dose-response relationship with mortality.

The primary outcome was all-cause in-hospital mortality. The secondary outcome was ICU length of stay (days). ICU type (medical, surgical, resuscitation) was incorporated into multivariable models as a potential confounder.

2.4. Statistical Analysis

Continuous variables were compared between groups using the Mann-Whitney U test; categorical variables were compared with the chi-square test. A trend test across the four boarding time strata was performed to assess for monotonic dose-response effects on mortality. Independent predictors of in-hospital mortality were identified through multivariable logistic regression with adjustment for ICU type. Discriminatory performance of boarding time for mortality prediction was assessed by receiver operating characteristic (ROC) analysis; the optimal cut-off was determined by the Youden index. The Delong method was used for AUC confidence intervals. Correlation between boarding time and ICU length of stay was quantified by Spearman's rho. All analyses were performed using IBM SPSS Statistics v.26; statistical significance was set at $p<0.05$.

3. RESULTS

3.1. Patient Characteristics and Group Distribution

Of 2029 patients screened during the study period, 24 were excluded due to incomplete or abnormal records, yielding a final cohort of 2005 patients. Early transfer (<6 h) occurred in 521 patients (26.0%), while 1484 patients (74.0%) experienced delayed transfer. The overall median ED boarding time was 18.6 hours (IQR: 8.5–34.2). Regarding ICU type distribution, most patients were admitted to the surgical ICU (47.0%), followed by the medical ICU (34.5%) and the resuscitation ICU (19.8%).

3.2. Primary and Secondary Outcomes

Overall in-hospital mortality was 34.5% (n=692). Mortality was significantly higher in the delayed transfer group (36.0%) compared with the early transfer group (30.3%; p=0.016). Median ICU length of stay was 8 days (IQR: 3–21) in the delayed transfer group versus 5 days (IQR: 2–14) in the early transfer group (p<0.001). Clinical outcomes by transfer group are summarized in Table 1.

Table 1. Clinical Outcomes by Transfer Group

Parameter	Early Transfer (<6 h) n=521	Delayed Transfer (≥6 h) n=1484	p value
In-hospital mortality, n (%)	158 (30.3%)	534 (36.0%)	**0.016**
Median ICU length of stay,	5 (2–14)	8 (3–21)	**<0.001**

days (IQR)			
Median ED boarding time, hours (IQR)	–	–	18.6 (8.5–34.2)

3.3. Multivariable Logistic Regression

On ICU type-adjusted multivariable logistic regression, ED boarding ≥6 hours was independently associated with in-hospital mortality (OR: 1.46; 95% CI: 1.18–1.81; p=0.001). Resuscitation ICU admission carried higher mortality risk compared with medical ICU admission (OR: 1.56; 95% CI: 1.13–2.16; p=0.007), whereas surgical ICU admission was associated with lower mortality (OR: 0.76; 95% CI: 0.60–0.96; p=0.022). Full regression results are presented in Table 2.

Table 2. Multivariable Logistic Regression Analysis for In-Hospital Mortality

Variable	Odds Ratio (OR)	95% Confidence Interval	p value
ED boarding time ≥6 hours	**1.46**	1.18 – 1.81	**0.001**
ICU length of stay (days)	1.02	1.01 – 1.03	**<0.001**
Surgical ICU (ref: Medical ICU)	0.76	0.60 – 0.96	**0.022**
Resuscitation ICU (ref: Medical ICU)	1.56	1.13 – 2.16	**0.007**

3.4. ROC Analysis, Trend Analysis, and Correlation

ROC analysis demonstrated that ED boarding time predicted in-hospital mortality with an AUC of 0.583 (95% CI: 0.558–0.608; p<0.001). The optimal cut-off was 11.5 hours (sensitivity: 64.2%; specificity: 51.8%). A statistically significant trend toward increasing mortality with longer boarding time was observed across the four strata (trend p=0.028). Spearman correlation analysis revealed a weak but statistically significant positive association between boarding time and ICU length of stay (r=0.27; 95% CI: 0.23–0.31; p<0.001). The distribution of mortality rates across boarding time strata is presented in Table 3.

Table 3. Mortality Rates by Boarding Time Strata

Boarding Time	<6 h	6–12 h	12–24 h	≥24 h	Total
Number of patients (n)	521	468	512	504	**2005**
Mortality (%)	30.3%	35.9%	36.5%	35.5%	**34.5%**

4. DISCUSSION

The central finding of this study is that ED boarding ≥6 hours independently increased the odds of in-hospital mortality by 46% (OR: 1.46) among critically ill patients requiring ICU admission. This result is consistent with, and reinforces, the pivotal multicenter evidence published by Chalfin et al., who first demonstrated this association in a landmark cohort of over 50,000 patients across 120 US hospitals and established 6 hours as the clinically meaningful boarding threshold [3].

The median boarding time of 18.6 hours (IQR: 8.5–34.2) observed in our cohort, with 74% of patients experiencing delays exceeding 6 hours, indicates a substantially higher boarding burden than figures typically reported in Western tertiary centers, where median boarding times frequently range between 4 and 9 hours [3,4]. This discrepancy likely reflects the extreme patient throughput and limited critical care bed availability characteristic of large Turkish city hospitals — a structural challenge that may amplify the clinical consequences of boarding.

The dose-response trend across boarding time strata (<6 h: 30.3%; 6–12 h: 35.9%; 12–24 h: 36.5%; ≥24 h: 35.5%; trend p=0.028) further strengthens the causal interpretation of the boarding-mortality relationship. While the plateau observed at ≥24 hours may reflect survivor selection bias (patients who survive extreme boarding may represent a less immediately ill subgroup), the overall gradient supports the biological plausibility that prolonged ED stays expose critically ill patients to suboptimal monitoring, delayed goal-directed interventions, and accumulated physiological deterioration.

The significantly longer ICU stay in the delayed transfer group (8 vs. 5 days; p<0.001), coupled with a moderate positive correlation between boarding time and ICU length of stay (r=0.27), suggests that the harm of boarding extends beyond acute mortality and generates substantial downstream resource utilization. These findings have direct implications for hospital capacity planning and ICU bed management strategies.

The ICU type-specific mortality differences identified in our regression model — resuscitation ICU conferring the highest risk (OR: 1.56) and surgical ICU the lowest (OR: 0.76) — are physiologically coherent. Resuscitation ICUs serve the most hemodynamically unstable patients requiring mechanical ventilation and multi-organ support, inherently

carrying the highest baseline mortality. The relatively favorable surgical ICU outcomes likely reflect the definitive, procedure-driven management of surgically correctable pathology with strong recovery potential.

The moderate discriminatory performance of boarding time for mortality prediction (AUC=0.583) and the elevated optimal cut-off of 11.5 hours — compared with the widely cited 6-hour policy threshold — underscore that boarding time is one of several interacting determinants of critical illness outcomes. Disease severity, comorbidity burden, the nature of the presenting condition, and the quality of ED care all modulate the magnitude of boarding-attributable harm.

Several limitations of this study warrant acknowledgment. The retrospective, single-center design limits generalizability, and unmeasured confounders — including illness severity scores (e.g., APACHE II), pre-existing comorbidities, presenting diagnosis, and staffing ratios — could not be fully adjusted for. Nevertheless, the large sample size, consecutive enrollment over a full 12-month period, and derivation from a nationally representative high-volume center lend substantial validity to the findings and provide a meaningful contribution to the sparse national literature on this topic.

5. CONCLUSION

Prolonged ED boarding is an independent predictor of increased in-hospital mortality and extended ICU length of stay in critically ill patients. In this high-volume tertiary center, where three in four patients experienced delayed ICU transfer and the median boarding time exceeded 18 hours, the clinical impact of ED crowding on critical care outcomes is clinically significant and quantifiable. These data support urgent efforts to optimize ED-to-ICU transfer pathways at the national level, including capacity expansion, early ICU triage protocols, and real-time bed management systems. Prospective multicenter studies incorporating severity-adjusted outcomes are needed to further characterize the modifiable determinants of boarding-attributable harm in the Turkish health system.

Ethical Statement

The expanded portion of this oral study is ongoing and is undergoing the approval process by the Ankara Etlik City Hospital Institutional Ethics Committee, and has been conducted in accordance with the Helsinki Declaration. Due to the retrospective and non-interventional nature of the study, obtaining individual informed consent was not deemed necessary.

Conflict of Interest: The author declares no conflict of interest.

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Hidden Danger in Blunt Chest Trauma: Traumatic Aortic Dissection with Concomitant Thoracic Injuries

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INTRODUCTION

Traumatic aortic injuries are commonly seen in blunt chest trauma, particularly in motor vehicle accidents and falls from height (1). Mortality reaches 30% within the first 24 hours (2). As it is the second most common cause of death after head trauma, its recognition in the emergency department is of vital importance (3). This report presents a case of traumatic aortic dissection complicated by multiple thoracic injuries.

CASE PRESENTATION

A 67-year-old male patient was admitted to the emergency department following a car accident. He had a history of prostate cancer. On arrival at the emergency department, his GCS was 12, blood pressure 112/70 mmHg, pulse 103 bpm, and oxygen saturation 86% (without O_2). Imaging revealed pericardial effusion, left-sided pneumothorax, haemothorax, sternum fracture and multiple rib fractures.

The patient was placed on monitoring and hydration was initiated; a central line was inserted, and routine blood tests and blood products were requested. Following consultations with thoracic surgery and cardiology, a thoracic CT angiography was performed. Imaging revealed aortic dissection distal to the aortic arch (Figure 1). The patient was taken to the operating theatre for emergency surgery by the cardiac surgery team

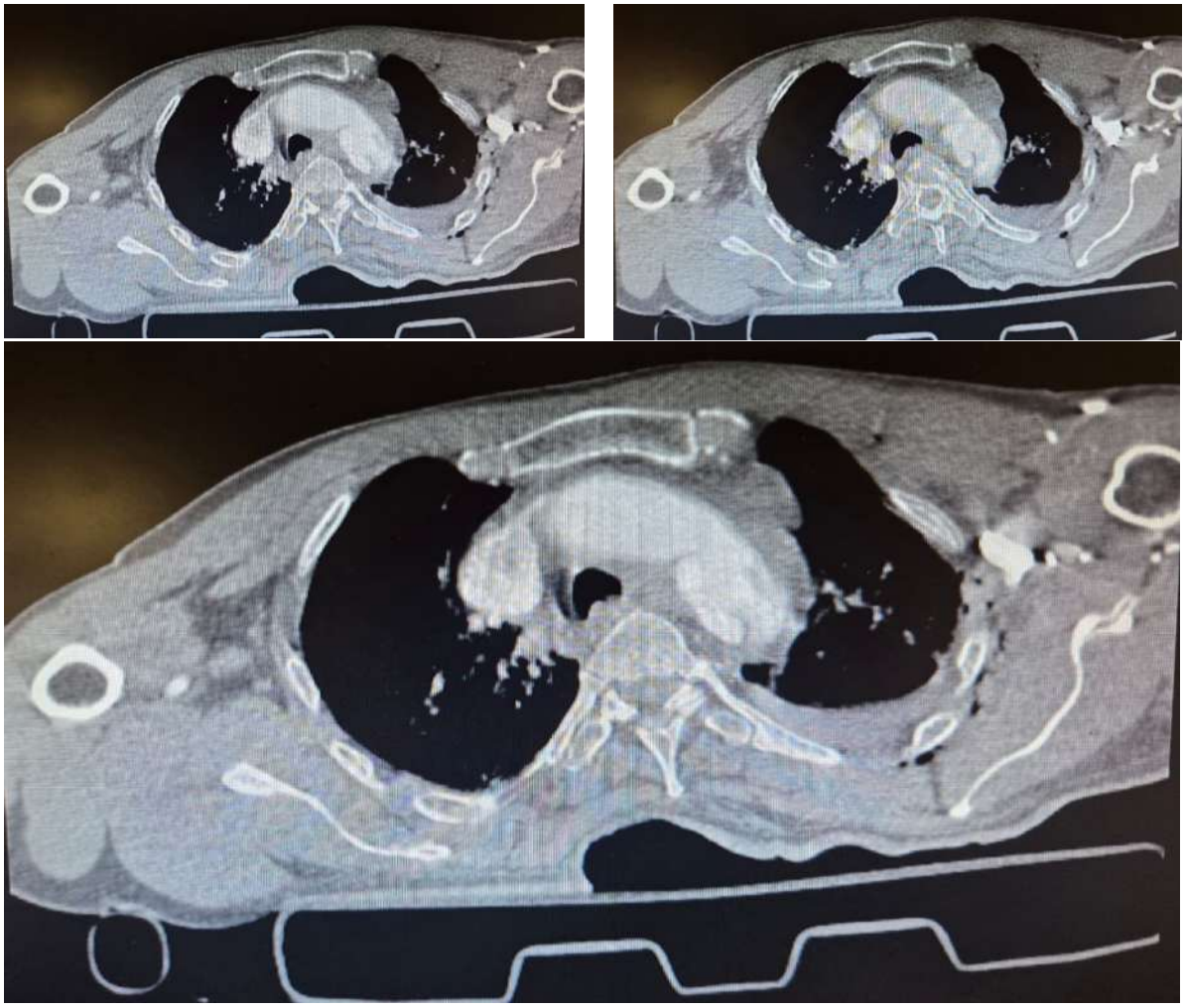

Figure1 Patient's CT Angiography Images

DISCUSSION

Traumatic aortic injuries typically occur in blunt chest trauma, and 80% of patients die at the scene (4). Clinical findings may include chest pain, a difference in blood pressure between the upper and lower extremities, loss of the femoral pulse, and a new murmur (5). However, accompanying pneumothorax, haemothorax, and rib fractures may complicate the diagnosis (6).

CT angiography is the gold standard diagnostic method for traumatic aortic injuries (1,7). In our case, the diagnosis was also made via CT angiography, and the patient was referred for emergency surgery. The literature emphasises that early diagnosis and surgical intervention improve survival (2,3).

CONCLUSION

Traumatic aortic dissection is a common and life-threatening emergency in blunt chest trauma. Although diagnosis is complicated when associated with multiple chest injuries, CT angiography is of critical importance for early diagnosis. Emergency surgical intervention improves the chances of survival.

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Keywords: Traumatic aortic dissection, Blunt chest trauma, Emergency surgery

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Ornidazole-Induced Bullous Fixed Drug Eruption: A Rare Case Report

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Abstract

Background: Fixed drug eruption (FDE) is a localized cutaneous adverse reaction that recurs at the same site upon re-exposure to a specific drug. While NSAIDs and sulfonamides are common triggers, bullous variants induced by nitroimidazoles like ornidazole are less frequently documented. Case Presentation: A 56-year-old woman presented with pruritus and numbness on the left hypothenar eminence six hours after ingesting 500 mg of ornidazole. Within 24 hours, an erythematous, ecchymotic bulla developed at the initial site, followed by ecchymosis on the thenar eminence. There were no systemic symptoms or prior history of similar reactions. Based on the temporal relationship and clinical morphology, a diagnosis of bullous FDE was made. The offending drug was discontinued, and the patient was treated with topical agents. Conclusion: Clinicians should maintain a high index of suspicion for FDE when evaluating localized bullous lesions following nitroimidazole therapy to prevent future recurrence and potential generalization.

Keywords: Drug eruption, ornidazole, bullous lesion.

Introduction

Fixed drug eruption (FDE) is a type IV delayed hypersensitivity reaction, accounting for approximately 20% of all cutaneous drug eruptions(1). Clinically, it presents as well-demarcated erythematous macules or edematous plaques, occasionally progressing to vesicle or bulla formation. Lesions typically appear within hours of drug administration, frequently affecting the face, genitalia, and acral areas. A hallmark of FDE is its recurrence at the exact same anatomical site upon re-exposure to the causative agent(2,3).

Commonly implicated medications include NSAIDs, sulfonamides, and anticonvulsants. However, nitroimidazoles—widely used for anaerobic and protozoal infections—are emerging as significant yet underreported triggers(4). Here, we report a case of bullous FDE specifically induced by ornidazole.

Case Presentation

A 56-year-old female with no significant medical history, known allergies, or prior cutaneous reactions presented with localized pruritus and numbness on the left palm. Symptoms began six hours after her first dose of 500 mg ornidazole, prescribed for a vaginal infection.

Within 24 hours, the patient developed a well-circumscribed, ecchymotic, erythematous bulla on the left hypothenar eminence and an associated ecchymotic patch on the thenar eminence. Physical examination revealed no mucosal involvement or systemic findings such as fever or lymphadenopathy. Laboratory investigations, including a complete blood count, renal and liver function tests, and inflammatory markers (ESR, CRP), were all within normal limits.

A clinical diagnosis of bullous FDE secondary to ornidazole was established. The patient was advised to permanently avoid nitroimidazoles. Treatment consisted of topical corticosteroids and antihistamines for symptomatic relief. The bulla resolved within two weeks, leaving residual post-inflammatory hyperpigmentation.

Figure 1. A well-demarcated, erythematous, ecchymotic bulla on the patient's left hypothenar eminence, consistent with a localized bullous fixed drug eruption.

Discussion

Ornidazole-induced FDE is relatively rare compared to other triggers. A recent systematic review identified only 14 cases, which presented with varied involvement of the oral mucosa, genitals, and extremities(5). Unlike generalized bullous FDE, which can mimic Stevens-

Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN), our patient exhibited a localized bullous variant without systemic compromise.

The clinical progression in this case—starting with sensory symptoms (numbness and itching) followed by rapid bulla formation—highlights the importance of early recognition. While patch testing or controlled oral challenges can confirm the causative agent in polypharmacy cases, the clear temporal correlation and lack of other medications in our patient allowed for a definitive clinical diagnosis.

Conclusion

This case emphasizes that FDE can occur at any age and may be triggered by commonly used antibiotics like ornidazole. Thorough medication history remains the gold standard for diagnosis. Patient education regarding the avoidance of the offending drug is crucial, as subsequent exposures may lead to more severe or generalized eruptions.

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The Role of Lactate, pH, and Creatinine Levels in Predicting Mortality in Patients with Acute Pulmonary Embolism: A Retrospective Single-Center Study

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ABSTRACT

Aim:
Pulmonary embolism (PE) is a serious cardiovascular emergency associated with high mortality. This study aimed to evaluate the relationship between clinical and laboratory parameters and mortality in patients diagnosed with PE.

Materials and Methods:
In this retrospective, cross-sectional, and observational study, patients diagnosed with PE in the Emergency Department of Malatya Training and Research Hospital between September 1, 2025, and December 1, 2025, were analyzed. A total of 41 patients were assessed, and 24 patients who met the inclusion and exclusion criteria were included in the study. Demographic data and laboratory parameters were obtained from hospital records. Statistical analyses were performed using SPSS version 27.0.

Results:
Of the patients, 54.16% were male, and the median age was 68 years. All patients were hospitalized, and the mortality rate was found to be 12.5%. When factors associated with mortality were evaluated, lactate and creatinine levels were significantly higher in patients who died, whereas pH values were significantly lower. No significant relationship was found between other laboratory parameters and mortality. Correlation analysis revealed significant associations among markers of inflammation, acidosis, and renal function.

Conclusion:
In patients with pulmonary embolism, mortality is more closely associated with tissue perfusion and metabolic status rather than inflammatory parameters. Easily accessible biochemical parameters such as lactate, pH, and creatinine may be valuable for early risk stratification and prognosis prediction.

Keywords: pulmonary embolism, mortality, lactate, pH, creatinine, prognosis, emergency department

Introduction

Pulmonary embolism (PE) is a serious cardiovascular emergency associated with high mortality, resulting from the obstruction of the pulmonary arteries by thrombotic material (1). Although it is relatively rare among emergency department admissions, its clinical importance is considerable due to the potential for sudden death when diagnosis is missed or delayed (2). The most common source of pulmonary embolism is deep vein thrombosis of the lower extremities, and these two clinical entities together constitute the spectrum of venous thromboembolism (VTE) (3). The clinical presentation of PE is highly variable, ranging from asymptomatic cases to cardiogenic shock (4).

Risk factors include immobilization, surgical interventions, malignancy, pregnancy, use of oral contraceptives, and genetic thrombophilia (5). However, PE may also occur in some patients without any identifiable risk factors (6).

Clinical probability scores (Wells, Geneva), D-dimer levels, and imaging modalities (particularly computed tomography pulmonary angiography) play a central role in diagnosis (7). Early diagnosis and appropriate anticoagulant therapy significantly reduce mortality (8). Nevertheless, identifying biochemical and clinical parameters that can predict prognosis in patients with PE is of critical importance for patient management and risk stratification (9). In particular, the relationship between parameters such as lactate, acid–base status, and renal function and mortality has been increasingly investigated in recent years (10).

In this study, it was aimed to retrospectively evaluate the clinical and laboratory characteristics of patients diagnosed with pulmonary embolism and to determine factors associated with mortality.

Materials and Methods

Study Design and Patient Population

This study was designed as a retrospective, cross-sectional, and observational study. Patients diagnosed with pulmonary embolism (PE) in the Emergency Department of Malatya Training and Research Hospital between September 1, 2025, and December 1, 2025, were analyzed. A total of 41 patients were evaluated during the study period, and the study was completed with 24 patients who met the predefined inclusion and exclusion criteria.

Inclusion and Exclusion Criteria

Patients aged 18 years and older, with a confirmed diagnosis of acute pulmonary embolism by computed tomography pulmonary angiography and with complete hospital records available, were included in the study. Patients with incomplete clinical or laboratory data, uncertain or suspected diagnoses, those under 18 years of age, and patients diagnosed with chronic pulmonary embolism were excluded.

Data Collection and Variables

Demographic characteristics, clinical data, and laboratory parameters of the patients were retrospectively obtained from the hospital information management system. The laboratory parameters evaluated in the study included C-reactive protein (CRP, mg/L), neutrophils ($\times 10^9$/L), lymphocytes ($\times 10^9$/L), monocytes ($\times 10^9$/L), platelets ($\times 10^9$/L), hemoglobin (g/dL), creatinine (mg/dL), albumin (g/dL), pH, partial pressure of carbon dioxide (pCO_2, mmHg),

and lactate (mmol/L). In addition, patient age (years) and length of hospital stay (days) were also recorded.

Study Endpoint

The primary endpoint of the study was in-hospital mortality. Patients were divided into two groups: those discharged alive and those who died.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 27.0. The distribution of data was assessed using the Kolmogorov–Smirnov test. Normally distributed data were presented as mean ± standard deviation, whereas non-normally distributed data were expressed as median and interquartile range (IQR: 25th–75th percentile). Categorical variables were presented as numbers and percentages.

For comparisons between two groups, the Student's t-test was used for parametric data and the Mann–Whitney U test for nonparametric data. The chi-square test was used for categorical variables. Relationships between variables were evaluated using Spearman correlation analysis. A p-value <0.05 was considered statistically significant.

Results

In our study, 24 patients diagnosed with pulmonary embolism were analyzed. Of these, 13 (54.16%) were male and 11 (45.84%) were female. The age distribution was heterogeneous, with a median age of 68 years. All patients were hospitalized. Three patients (12.5%) died, while 21 patients (87.5%) were discharged alive.

The mean ± standard deviation values of parametric variables were as follows: age 68±12.8 years, albumin 3.1±0.7 g/dL, and length of hospital stay 5±1 days.

The median and IQR values of nonparametric variables were as follows: CRP 1.8 (0.5–7.4), neutrophils 7.7 (5.2–11.5), lymphocytes 1.9 (1.1–2.5), platelets 208 (150–259), hemoglobin 13 (12.1–14.2), monocytes 0.7 (0.5–1), creatinine 1 (0.8–1.4), pH 7.35 (7.27–7.41), pCO_2 42.5 (36.5–50), and lactate 2.3 (1.7–5.9). Detailed data are presented in Table 1.

Table 1. Mean, SD, median, and IQR values of the variables

					IQR	
Parameters	n	mean	SD	median	25%	75%
Age(year)	**24**	68	12.8			
Albumin(g/dL)		3.1	0.7			
HOSPITALTY(day)		5	1			
CRP(mg/L)				1.8	0.5	7.4
Neutrophil(×10³/µL)				7.7	5.2	11.5
Lymphocyte(×10³/µL)				1.9	1.1	2.5

Platelet($\times 10^3/\mu L$)	208	150	259
Hemoglobin(g/dL)	13	12.1	14.2
Monocytes ($\times 10^9/L$)	0.7	0.5	1
Creatinine(mg/dL)	1	0.8	1.4
PH	7.35	7.27	7.41
pCO_2 (mmHg)	42.5	36.5	50
Lactate (mmol/L)	2.3	1.7	5.9

The relationship between categorical variables (gender and mortality) and parametric variables was evaluated using the t-test, and no statistically significant difference was found ($p>0.05$).

The relationship between categorical variables and nonparametric variables was evaluated using the Mann–Whitney U test. When nonparametric variables were compared with mortality, pH, lactate, and creatinine values showed statistically significant associations. Lactate ($p=0.016$) and creatinine ($p=0.038$) levels were higher in patients who died, whereas pH ($p=0.013$) was lower in these patients. No statistically significant relationship was found between mortality and other nonparametric variables (CRP, neutrophils, lymphocytes, platelets, hemoglobin, monocytes, pCO_2) ($p>0.05$).

No statistically significant relationship was found between gender and nonparametric variables ($p>0.05$).

The relationship between gender and mortality was evaluated using the chi-square test. Among the patients who died, two were male and one was female. Although not statistically significant, mortality rates were higher in males ($p>0.05$).

Correlations between variables were evaluated using Spearman correlation analysis. Significant relationships were found among markers of inflammation, acidosis, and renal function. There was a moderate positive correlation between CRP and neutrophils ($r=0.569$; $p=0.004$) and monocytes ($r=0.513$; $p=0.010$), and a moderate negative correlation between CRP and albumin ($r=-0.491$; $p=0.015$). A strong positive correlation was found between neutrophils and monocytes ($r=0.786$; $p<0.001$). A strong negative correlation was observed between creatinine and pH ($r=-0.701$; $p<0.001$), and a moderate negative correlation between lactate and pH ($r=-0.669$; $p<0.001$). Additionally, a moderate positive correlation was found between creatinine and lactate ($r=0.483$; $p=0.017$). A moderate negative correlation was observed between hemoglobin and lactate ($r=-0.417$; $p=0.043$). No statistically significant correlation was found between age, length of hospital stay, and the examined parameters.

Discussion

In our study, the demographic characteristics of 24 patients diagnosed with pulmonary embolism revealed that most patients were in the advanced age group, with a median age of 68 years. Additionally, the fact that all patients were hospitalized and a mortality rate of 12.5% was observed suggests that our study population predominantly represents a clinically

significant, moderate-to-high risk group. In the literature, pulmonary embolism is reported to occur more frequently in older individuals, with mortality risk increasing significantly with age (1,2). Similarly, mortality rates of approximately 10–15% have been reported in hospitalized cases (3). The mortality rate observed in our study appears to be consistent with the literature.

Although the gender distribution was slightly higher in males, it was relatively balanced, supporting that pulmonary embolism affects both sexes at similar rates (4). However, some studies suggest that male sex may be associated with a worse prognosis (5). The lack of a significant difference between sexes in our study may be related to the limited sample size.

Overall, the patient profile in our study is consistent with the typical pulmonary embolism population described in the literature and supports that advanced age and the need for hospitalization are important determinants of mortality.

When factors associated with mortality were evaluated, lactate and creatinine levels were higher, and pH values were lower in patients who died. In contrast, no significant relationship was found between mortality and other parameters such as CRP, neutrophils, lymphocytes, platelets, hemoglobin, monocytes, and pCO_2.

The literature emphasizes that the main determinants of mortality in pulmonary embolism are hemodynamic status and right ventricular function (1,2). This leads to a worse prognosis in patients with tissue hypoperfusion and associated metabolic disturbances. Indeed, Vanni et al. demonstrated that plasma lactate levels are an independent prognostic marker in acute pulmonary embolism and are associated with mortality (10).

Similarly, low pH values reflecting metabolic acidosis are important indicators of impaired tissue perfusion and are associated with poor clinical outcomes in advanced-stage pulmonary embolism (1,9). Elevated creatinine levels may reflect systemic hypoperfusion and organ dysfunction, indicating a higher disease burden and contributing to increased mortality (2,3).

On the other hand, the lack of a significant association between inflammatory and hematological parameters and mortality suggests that these markers may be insufficient to reflect acute hemodynamic deterioration.

Although no statistically significant relationship was found between gender and mortality in our study, higher mortality rates were observed in male patients. There is no clear consensus in the literature regarding the prognostic impact of gender, and pulmonary embolism is generally reported to affect both sexes similarly (5,6).

Overall, our findings support that mortality in pulmonary embolism is more closely related to tissue perfusion and metabolic status, and that parameters such as lactate, pH, and creatinine may be more valuable in early risk stratification.

Correlation analysis in our study demonstrated significant relationships among markers of inflammation, acidosis, and renal function. The positive correlation between CRP and neutrophils and monocytes, and the negative correlation with albumin, suggest that the inflammatory response is a systemic process. The strong positive correlation between neutrophils and monocytes also indicates concurrent activation of inflammatory cellular pathways.

In the literature, the inflammatory response is reported to play an important role in the pathophysiology of pulmonary embolism, with parallel changes observed among acute phase reactants and leukocyte subgroups (3,6). The negative correlation between CRP and albumin is also consistent with the decrease in albumin, a negative acute-phase reactant, during inflammation (11).

The strong negative correlation between creatinine and pH, and the moderate negative correlation between lactate and pH, indicate that renal dysfunction and tissue hypoperfusion are closely associated with metabolic acidosis. The positive correlation between creatinine and lactate further supports this finding. Similarly, the literature emphasizes that elevated lactate levels and acidosis are important indicators of systemic hypoperfusion and are associated with organ dysfunction (2,10).

The negative correlation between hemoglobin and lactate suggests a relationship between reduced oxygen-carrying capacity and tissue hypoxia (12). In contrast, the lack of significant correlations with age and length of hospital stay suggests that biochemical parameters may better reflect the acute pathophysiological process.

Overall, our study supports that inflammation, hypoperfusion, and organ dysfunction are interrelated processes, and that the clinical presentation of pulmonary embolism is shaped by the interaction of these multiple pathophysiological mechanisms.

Limitations

This study has several limitations. First, its single-center and retrospective design limits the generalizability of the results. The relatively small sample size may have reduced the statistical power. Additionally, due to the use of retrospective data, some clinical parameters and imaging findings could not be included in the analysis. The lack of echocardiographic data reflecting hemodynamic status and right ventricular function is another important limitation.

Conclusion

In this study, mortality in patients with pulmonary embolism was shown to be particularly associated with tissue hypoperfusion and metabolic deterioration. Increased lactate and creatinine levels and decreased pH were significantly associated with mortality, whereas no significant relationship was found between mortality and inflammatory or hematological parameters. These findings suggest that easily obtainable biochemical parameters in the emergency department may be clinically valuable for early risk stratification and prognosis prediction.

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Bilateral Toe Necrosis Following Exposure to Extreme Cold: A Case Report with Preserved Major Arterial Flow

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Introduction

Frostbite injuries resulting from exposure to cold are clinical conditions that can lead to significant morbidity, particularly in unprotected and vulnerable populations. Frostbite primarily affects the distal extremities, fingers, and exposed skin areas where blood flow is reduced (such as the nose and ears) and insulation is inadequate. Pathophysiologically, vasoconstriction, microvascular thrombosis, and reperfusion injury play a role. The preservation of major arterial circulation does not rule out distal tissue necrosis. Another classification based on frostbite in the hands and feet has been proposed: 1st degree; no cyanosis in the extremity; no risk of amputation or permanent damage is anticipated. 2nd degree; cyanosis only in the distal phalanx; amputation up to the soft tissue and nail/toe nail sequelae are anticipated.Grade 3: Cyanosis in the middle and proximal phalanges; amputation up to the bone of the finger and functional sequelae are anticipated. Grade 4: Cyanosis in the carpal/tarsal bones; limb amputation and functional sequelae are anticipated. This case presents with bilateral widespread toe necrosis despite normal major vascular flow (1).

Case

The patient presented with complaints of discoloration in both feet. The history revealed exposure to significant cold for 1 day outdoors and subsequently for 3 days indoors approximately 15 days prior. It was learned that the patient had sought care at a public hospital due to pain developing after cold exposure, where he was hospitalized for 5 days and subsequently discharged. The patient is a migrant and subsequently presented to our center in Konya.

On physical examination, complete black discoloration was observed in all toes of the right foot, and approximately 90% black discoloration in all toes of the left foot. The lesions were found to have a sharp border, progressing distally to proximally, and were consistent with dry gangrene (Figure 1).

A computed tomography angiography (CTA) of the lower extremities was performed. Imaging results: Both main external and internal iliac arteries, both deep and superficial femoral arteries, popliteal arteries, the tibio-peroneal trifurcation region, and the anterior tibial, posterior tibial, and peroneal arteries were reported to be patent, with normal filling and caliber, extending down to the ankle level.

These findings indicated that major arterial circulation was preserved. The patient, who had a preliminary diagnosis of a third-degree cold burn during the examination, was referred to the orthopedic and cardiovascular surgery departments. Both departments concluded that there was no indication for acute surgical intervention; instead, outpatient follow-up and elective surgery were recommended.

Figure1 External Physical Examination Image

Discussion

Frostbite injuries are classified as superficial or deep, and in advanced stages, tissue necrosis and gangrene may develop. In this case, the patency of major arterial structures suggests that the pathology developed at the microvascular level rather than due to macrovascular occlusion. Vasospasm, endothelial damage, and microthrombosis developing after exposure to cold can lead to ischemia and necrosis in distal tissues (1).

Bilateral and symmetrical involvement is consistent with systemic cold exposure. Additionally, the patient's socioeconomic status and migrant status may be considered risk factors regarding delayed presentation and difficulties accessing treatment. A previous study demonstrated that 43.75% of patients presenting with frostbite were undocumented migrants (2).

In cases where major vessels remain patent, the treatment approach involves wound care, infection prophylaxis, waiting for the demarcation line to become clearly defined, and

planning surgical debridement or amputation at the appropriate time. Thrombolytic therapy has been shown in numerous studies to improve tissue salvage. The current literature recommends delayed surgery with amputations guided by soft tissue debridement and bone scanning. In these patients, surgical intervention—including debridement, selective blister drainage, fasciotomy, surgical salvage, skin grafts, and flaps—is frequently required to optimize form and function (2,3).The treatment duration for third- and fourth-degree frostbite injuries ranges from 34 to 78 days (3). In our case, since the patient presented 2 weeks after the frostbite occurred, thrombolytic therapy was not administered, and elective surgery was planned for a later stage.

Conclusion

Bilateral toe necrosis following exposure to severe cold can occur even when major arterial circulation is preserved. This case highlights the clinical significance of microvascular damage ( ) in frostbite injuries and the necessity of early intervention in vulnerable populations. A timely and appropriate multidisciplinary approach plays a critical role in reducing morbidity.

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Keywords: Frostbite, frostbite, toe necrosis.

7045

A Rare Cause of Massive Hematemesis: Arterioesophageal Fistula Due to Ruptured Subclavian Artery Pseudoaneurysm Following Foreign Body Ingestion

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Abstract

Introduction and Purpose

Subclavian artery pseudoaneurysms are rare vascular lesions, most commonly associated with penetrating trauma and iatrogenic injuries. This case aims to present a previously unreported cause of subclavian artery pseudoaneurysm developing after ingestion of a sharp foreign body, leading to life-threatening massive hematemesis, and to emphasize the diagnostic challenges associated with this condition.

Case Presentation

A 55-year-old female presented to the emergency department with massive hematemesis. Her history revealed that two weeks earlier, she had presented to another emergency department with a sensation of a foreign body in her throat after consuming a chicken-based meal. A non-contrast computed tomography (CT) scan performed at that time showed no pathological findings, and the patient was discharged. In the following days, she experienced intermittent minor episodes of bloody vomiting (“sentinel bleeding”) and visited multiple healthcare centers; however, no definitive diagnosis was established. Upon presentation to our emergency department with massive hematemesis (approximately 700 cc), an urgent contrast-enhanced CT angiography (CTA) was performed. Imaging revealed esophageal perforation and a ruptured pseudoaneurysm of the left subclavian artery.

Discussion and Conclusion: Artery-esophageal communications carry a mortality rate exceeding 70%. Diagnosis is often delayed because initial non-contrast CT scans have low sensitivity (~56%), whereas contrast-enhanced CTA is the gold standard with nearly 100% sensitivity. Ingested sharp foreign bodies carry a high risk of lethal vascular complications. A high index of clinical suspicion and early use of contrast-enhanced CTA are mandatory for any patient presenting with hematemesis and a history of foreign body ingestion to prevent a fatal outcome.

Keywords: subclavian artery pseudoaneurysm, foreign body ingestion, esophageal perforation

Introduction

Pseudoaneurysms of the subclavian artery are rare vascular entities, most commonly associated with penetrating trauma and iatrogenic injury, and account for a small proportion of civilian vascular lesions (1). Due to their anatomical proximity to vital mediastinal structures, these lesions carry a high risk of life-threatening complications, particularly when associated with rupture or fistulization (2).

Ingestion of sharp foreign bodies is a relatively common clinical scenario and frequently results in esophageal injury or perforation. Although most cases follow a benign course, transmural perforation may rarely lead to severe complications involving adjacent mediastinal structures, including major vascular injury (3,4). Arterioesophageal fistula involving the subclavian artery secondary to foreign body ingestion has been reported in the literature; however, it remains an exceedingly rare and often fatal condition (5–7).

In this report, we aim to present a rare case of subclavian artery pseudoaneurysm secondary to foreign body ingestion complicated by arterioesophageal fistula, and to highlight the diagnostic challenges and clinical pitfalls associated with this catastrophic vascular complication.

Case Report

A 55-year-old female presented to the emergency department with massive hematemesis. Her history revealed accidental ingestion of a toothpick embedded in chicken meat two weeks prior. At that time, she had presented to another emergency department with a foreign body sensation in the throat and dysphagia. Non-contrast cervical and thoracic computed tomography (CT) showed no acute pathology, and she was discharged. Over the following 10 days, the patient presented to multiple healthcare centers with persistent dysphagia, neck pain, and intermittent minor episodes of hematemesis ("sentinel bleeding"); however, no definitive diagnosis was established.

She was eventually brought to our emergency department due to massive hematemesis. On admission, she was hemodynamically unstable, with hypotension (blood pressure: 90/40 mmHg), tachycardia (heart rate: 121 bpm), and oxygen saturation of 91% on room air. Initial management included rapid resuscitation with intravenous fluids and PPI infusion. During observation, the patient developed a sudden episode of massive hematemesis (approximately 700 mL), accompanied by deterioration in consciousness (Glasgow Coma Scale score: 8), necessitating emergent endotracheal intubation for airway protection. Urgent contrast-enhanced CT angiography revealed esophageal perforation and a ruptured pseudoaneurysm of the left subclavian artery (Figures 1 and 2).

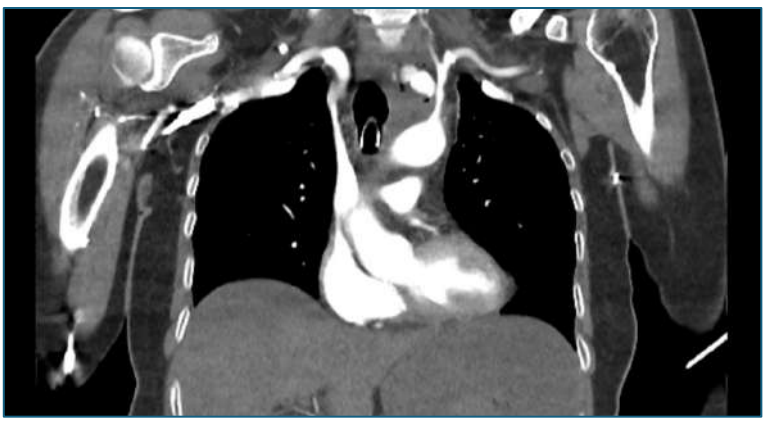

Figure 1. Coronal CT angiography image demonstrating a large pseudoaneurysm arising from the left subclavian artery.

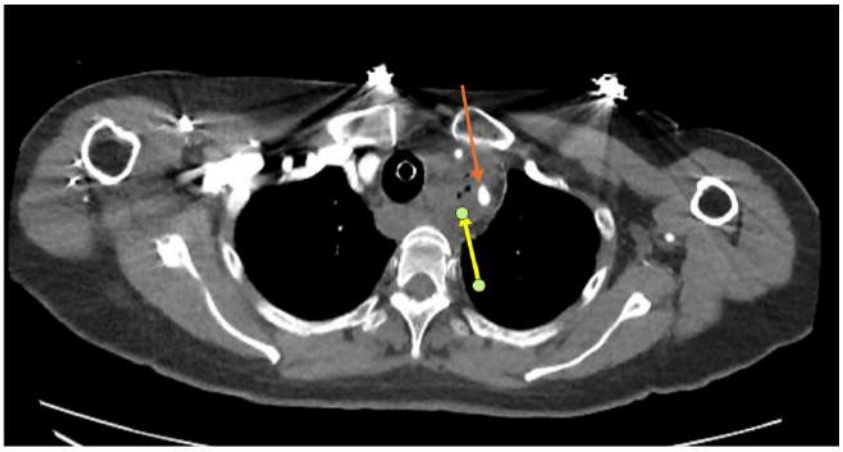

Figure 2. Axial CT angiography image at the level of the upper mediastinum demonstrating a ruptured pseudoaneurysm of the left subclavian artery (arrow), with associated periesophageal gas bubbles.

Following multidisciplinary consultation with thoracic surgery, cardiovascular surgery, and interventional radiology teams, the patient underwent emergent endovascular embolization. Despite initial technical success, hemorrhage control could not be sustained, and the patient died 12 hours after admission.

Discussion

Subclavian artery pseudoaneurysms (SCA-P) are exceedingly rare clinical entities, accounting for less than 2% of all civilian vascular injuries. While most pseudoaneurysms occur in the femoral artery following medical interventions, SCA-P is typically associated with penetrating trauma, iatrogenic injury—particularly during central venous catheterization—or infection. Although ingestion of sharp foreign bodies is a well-recognized cause of esophageal injury, progression to vascular involvement, such as pseudoaneurysm formation or arterioesophageal fistula, remains exceptionally rare and frequently fatal (8).

A key clinical feature in this case was the presence of intermittent, self-limited episodes of hematemesis preceding the final catastrophic hemorrhage, consistent with the phenomenon of "sentinel bleeding." In similar vascular–esophageal pathologies, sentinel bleeding has been reported in approximately 20% of cases and may represent a critical warning sign prior to massive hemorrhage (9,4).

Diagnosis is often challenging due to the nonspecific nature of early symptoms, such as dysphagia and neck pain, which may mimic more common conditions. This case highlights an important diagnostic pitfall: initial non-contrast CT imaging may be falsely reassuring. Current evidence suggests that while plain radiography and non-contrast CT have limited sensitivity, contrast-enhanced CT and CT angiography remain the gold standard modalities, with near-complete sensitivity for detecting vascular complications and guiding management (3,10).

Management of SCA-P is complex due to its anatomical proximity to vital mediastinal structures. Although open surgical repair has traditionally been considered the standard approach, endovascular techniques—including covered stent placement and embolization—have emerged as less invasive and increasingly preferred alternatives, particularly in hemodynamically unstable patients (11).

Importantly, this case underscores that even subtle or transient episodes of hematemesis following foreign body ingestion should not be underestimated, as they may represent an early manifestation of a developing arterioesophageal fistula. A high index of clinical suspicion, coupled with early use of contrast-enhanced imaging, is essential to avoid potentially fatal diagnostic delays in these rare but catastrophic presentations.

Conclusion

This case highlights the catastrophic potential of ingested sharp foreign bodies. Massive hematemesis following foreign body ingestion should raise immediate suspicion of an arterioesophageal communication, even when initial imaging is unremarkable. Sentinel bleeding may represent a critical warning sign requiring urgent evaluation. Early use of

contrast-enhanced CT angiography and a multidisciplinary approach are essential in the management of these rare but life-threatening conditions.

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ACUTE INFEROPOSTEROLATERAL STEMI DURING HAIR TRANSPLANTATION: A CASE REPORT

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Introduction

Hair transplantation is generally considered a minimally invasive cosmetic procedure with a low complication rate. However, although rare, cardiovascular events occurring during the procedure may be life-threatening. In this case report, we present a young male patient who developed acute inferoposterolateral ST-elevation myocardial infarction (STEMI) following sudden-onset chest pain during a hair transplantation procedure.

Case Presentation

A 33-year-old male patient was admitted to the emergency department with sudden-onset chest pain lasting for one hour, described as a squeezing sensation, accompanied by dyspnea and diaphoresis, which developed during a hair transplantation procedure. The patient had no prior history of similar symptoms, chronic illness, regular medication use, smoking, or family history of premature cardiovascular disease.

On admission, vital signs were as follows: blood pressure 110/65 mmHg, and pulse was regular and within normal limits. Cardiac and pulmonary examinations were unremarkable. There was no peripheral edema or jugular venous distention.

Electrocardiography revealed significant ST-segment elevation in leads II, III, aVF, and V5–V6, along with reciprocal changes, consistent with acute inferoposterolateral STEMI.

Initial troponin-I level was 6.111 µg/L, which subsequently increased to 65.325 µg/L. The patient was immediately transferred to the catheterization laboratory for coronary angiography.

Coronary angiography demonstrated 99% critical stenosis in the right coronary artery (RCA) and 80% stenosis in the left anterior descending artery (LAD). Percutaneous coronary intervention (PCI) was successfully performed using a 6F catheter. Coronary flow improved, and no procedural complications occurred.

Post-procedural transthoracic echocardiography revealed hypokinesia in the basal septal and basal inferior segments, without significant segmental wall motion abnormalities overall, and the left ventricular ejection fraction was measured at 50%.

Discussion

Although hair transplantation is a safe cosmetic procedure typically performed under local anesthesia, procedural stress, pain, anxiety, and sympathetic activation may adversely affect the cardiovascular system and trigger acute events. While cases of myocardial infarction during cosmetic procedures have been reported in the literature, most involve older patients or those with known cardiovascular risk factors.

Despite the absence of known risk factors in this patient, the presence of significant stenosis in both the RCA and LAD suggests that subclinical and previously undiagnosed atherosclerotic disease can lead to serious outcomes even in young individuals. Prompt recognition of chest pain and associated autonomic symptoms was crucial in ensuring timely PCI and a favorable clinical outcome.

This case highlights that cardiovascular complications should not be overlooked even during routine cosmetic procedures. Symptoms such as chest pain, dyspnea, palpitations, or syncope during such procedures require immediate evaluation.

Conclusion

Acute myocardial infarction may occur even in young patients with a low-risk profile during cosmetic procedures such as hair transplantation. Early diagnosis, rapid referral, and successful PCI contributed to a favorable outcome in this case. Physicians performing cosmetic procedures should remain vigilant for cardiac symptoms and ensure prompt referral when necessary.

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A RETROSPECTIVE ANALYSIS OF PEDIATRIC VERTEBRAL INJURIES PRESENTING TO A TERTIARY EMERGENCY DEPARTMENT

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ABSTRACT

Objective: The aim of this study was to investigate the effects of injury mechanism and localization in pediatric vertebral fracture cases on biochemical parameters, systemic stress response, and clinical outcomes.

Materials and Methods: Eighteen patients aged 0–18 years who presented to the emergency department between 2021 and 2026 and were diagnosed with vertebral fractures were retrospectively included in the study. The patients' demographic data, injury mechanisms, injury sites, hospital stays, and biochemical and hematological laboratory parameters at the time of presentation were analyzed.

Results: Of the 18 patients included in the study, 9 (50%) were male. The most common injury mechanism was a fall (66.6%). Four patients had cervical, 6 had thoracic, 6 had lumbar, and 2 had sacral injuries. The patients' hospital and intensive care unit (ICU) lengths of stay were found to be 1 day. A statistically significant difference was found between injury location and serum urea levels ($p=0.017$). Urea levels in cases with lumbar fractures were found to be significantly lower compared to those with cervical ($p=0.002$) and thoracic ($p=0.032$) injuries.

Conclusion: Fall is the most common mechanism in pediatric vertebral fractures and is generally associated with a stable clinical course and short hospital stay. As the level of injury increases (cervical and thoracic), rising serum urea levels indicate that higher-level injuries lead to greater metabolic catabolism. In this context, biochemical monitoring in children with high-level spinal cord injuries must be carefully monitored as part of clinical management.

KEYWORDS: Biochemical Markers, Pediatric Trauma, Vertebral Fractures

INTRODUCTION

Although pediatric vertebral injuries are less common than in adults, they hold a critical place in emergency medicine practice due to the high risk of morbidity. Anatomical differences in children, such as ligamentous laxity, the horizontal orientation of the facet joints, and a proportionally larger head size, completely distinguish injury patterns from those in adults (1).

While high-energy motor vehicle crashes are typically the focus in the literature, low-energy falls remain the predominant trauma pattern in the region where our center is located (2). Post-traumatic fluctuations in leukocyte counts and parameters such as the Lymphocyte-to-Albumin Ratio (LAR) serve as important indicators of immune-inflammatory balance and "secondary injury" processes. Additionally, the neuroendocrine response during this process can lead to significant changes in glucose, urea, and renal perfusion (3). Although most studies typically focus on cases with severe neurological deficits, the profile of lower-energy injuries presenting to tertiary care hospitals remains insufficiently elucidated (4). The aim of this study is to present our center's experience, supported by the literature, by examining the effects of injury mechanism and localization on biochemical parameters, systemic stress response, and clinical outcomes in cases of pediatric vertebral fractures.

MATERIALS AND METHODS

This study was conducted by retrospectively analyzing the data of pediatric cases who presented to the Emergency Department of Malatya Turgut Özal University, Malatya Training and Research Hospital following trauma and were diagnosed with vertebral fractures. The study protocol was approved by the Ethics Committee for Scientific Research in Health Sciences at Malatya Turgut Özal University. All procedures were conducted in accordance with the principles of the Declaration of Helsinki.

PATIENT SELECTION

The study included 18 patients who presented to the emergency department between January 1, 2021, and January 1, 2026, were aged 0–18 years, and had at least one level of vertebral fracture confirmed radiologically (X-ray, CT, or MRI).

Inclusion Criteria: History of trauma, radiologically confirmed vertebral fracture, complete demographic and laboratory data.

Exclusion Criteria: Pathological fractures (tumor, osteogenesis imperfecta, etc.), incomplete laboratory data, history of known chronic kidney or liver failure.

Patients were grouped according to the injury site (Cervical, Thoracic, Lumbar, Sacral) and the mechanism of injury (Fall, Traffic Accident).

DATA COLLECTION AND PARAMETERS

The following data were recorded for patients through the hospital automation system:

Demographic Data: Age, gender.

Clinical Data: Mechanism of injury, vertebral level, need for surgical treatment, presence of additional surgical pathology, length of stay in the hospital and the Intensive Care Unit (ICU).

Laboratory Parameters: Complete Blood Count at the time of admission [White Blood Cell (WBC), Hemoglobin (Hb), Hematocrit (Hct), Lymphocyte Count] and Biochemical Analyses [Glucose, Urea, Creatinine, AST, ALT, Albumin].

Indices: The Lymphocyte-to-Albumin Ratio (LAR), calculated by dividing the lymphocyte count by the 564lbümin level, was determined.

RADIOLOGICAL EVALUATION

The location and severity of vertebral injuries were evaluated by specialist radiologists and neurosurgeons using plain radiographs taken as part of the emergency department protocol and, when necessary, computed tomography (CT) scans—considered the gold standard. Fracture distribution was categorized by anatomical region.

STATISTICAL ANALYSIS

Statistical analyses were performed using the SPSS v26.0 program. The normality of the data distribution was assessed using the Shapiro-Wilk test. Continuous variables were presented as mean ± standard deviation for those with a normal distribution, and as median and interquartile range (IQR) for those without a normal distribution. For group comparisons (such as the relationship between injury location and laboratory parameters), a one-way analysis of variance (ANOVA) test was used.

Post-hoc (LSD or Tukey) analyses were performed to determine which group accounted for the differences in statistically significant results. A p-value of <0.05 was considered statistically significant.

FINDINGS:

Based on our inclusion and exclusion criteria, 18 patients who presented to the emergency department over a 5-year period were included in our study. Nine of our patients (50%) were male. Six patients were injured in traffic accidents, while the remaining 12 were injured due to falls. Four patients had cervical injuries, six had thoracic injuries, six had lumbar injuries, and two had sacral injuries. Surgical treatment was performed on six patients. Five of our patients had additional surgical pathologies in addition to vertebral injury.

Table 1. Demographic Characteristics of Pediatric Cases with Vertebral Injury and Distribution of Laboratory Parameters at Presentation

	n	mean	±sd		n	median	IQR(25-75)
wbc	18	12,3	5,9	lymphocyte	18	2,38	1,86-3,17
hb	18	13,7	1,8	lar	18	0,56	0,39-0,7
hct	18	40,9	5,5	glucose	18	102	94,5-130,5
urea	18	30,5	9,5	ast	18	35	22-40
creatinin	18	0,71	0,14	alt	18	17,5	14-22,2
albumin	18	4,4	0,5	age	18	14	9,75-16
				h-los	18	0,5	0-4,25
				icu-los	18	0	0-2

Data are presented as mean ± standard deviation (SD) for normally distributed variables, and as median (interquartile range [IQR]: 25th-75th percentiles) for non-normally distributed variables.

WBC: White Blood Cell count (10^3/µL), Hb: Hemoglobin, Hct: Hematocrit, LAR: Lymphocyte-to-Albumin Ratio, AST: Aspartate Aminotransferase, ALT: Alanine Aminotransferase, ICU-los: Intensive Care Unit length of stay (days), h-los: hospital lenght of stay, n: Number of cases.

The clinical and laboratory characteristics of the 18 pediatric patients included in the study are presented in Table 1.

Demographic Data and Length of Stay:

The median age of the study group was 14 (IQR: 9.75–16). The median hospital length of stay was 1 day (IQR: 0–4.25), while the median intensive care unit (ICU) length of stay was 1 day (IQR: 0–2).

Hematological and Biochemical Parameters:

In complete blood count data, the mean white blood cell (WBC) count was 12.3 ± 5.9 × 10^3/µL, hemoglobin (Hb) was 13.7 ± 1.8 g/dL, and hematocrit (Hct) was 40.9 ± 5.5%. The median lymphocyte count was 2.38 × 10^3/µL (IQR: 1.86–3.17), and the median LAR level was 0.56 (IQR: 0.39–0.7).

In biochemical analyses, the mean urea level was 30.5 ± 9.5 mg/dL, creatinine 0.71 ± 0.14 mg/dL, and albumin 4.4 ± 0.5 g/dL. When examining the non-parametrically distributed data in the right column, the median glucose level was 102 mg/dL (IQR: 94.5–130.5), the median AST level was 35 U/L (IQR: 22–40), and the median ALT was 17.5 U/L (IQR: 14–22.2).

When the relationship between the location of the injured vertebra (cervical, thoracic, lumbar, and sacral) and laboratory parameters was examined; a statistically significant difference was detected between serum urea levels and the injury site (p=0.017).

In the post-hoc analyses (multiple comparison tests) conducted between groups;

Urea levels in patients with lumbar vertebral fractures were found to be significantly lower than those in patients with cervical vertebral fractures (p=0.002).

Similarly, when lumbar vertebral fractures were compared with thoracic vertebral fractures, urea levels were found to be statistically significantly lower in the lumbar injury group (p=0.032).

DISCUSSION

In our study, 50% of the patients were male. Although the literature generally reports a male predominance in pediatric spinal injuries, the gender distribution is found to be similar, particularly in early childhood (0–8 years) (5). The equal distribution in our group may suggest that the injury mechanisms stem from environmental factors affecting both genders in similar proportions.

In our study, the most common injury mechanism was falls (66.6%, n=12), followed by traffic accidents (33.3%, n=6). A similar study also identified falls (73.6%) as the most common type of trauma and confirmed that this represents the predominant injury pattern in the pediatric population (6). The fact that falls are a more common cause of spinal injury than traffic accidents in the pediatric population can be explained by both the anatomical/biomechanical characteristics of children and activity patterns specific to their age groups.

The distribution of vertebral injuries identified in our study (Cervical n=4, Thoracic n=6, Lumbar n=6, Sacral n=2) is consistent with a study conducted abroad, which also reported that injuries to the lumbar and thoracic vertebrae are more common (7). This distribution may be attributed to the unique biomechanical characteristics of the pediatric spine during the growth process, such as a flatter spinal curvature, less pronounced thoracic kyphosis, and an imbalance in the kyphosis/lordosis ratio during growth, which may explain the higher incidence of lumbar and thoracic injuries in pediatric age groups.

In our study, it was determined that 6 of our patients (33.3%) required surgical treatment, and 5 patients (27.7%) had additional surgical pathologies beyond the vertebral injury. Similar findings regarding the need for surgical treatment and the presence of additional surgical pathologies are reported in the literature (8). We believe this is due to the high strength of the vertebral bones, which typically results in these injuries occurring as a consequence of high-energy multiple traumas.

The median age of our study group was 14 (IQR: 9.75–16). Similarly, the average ages of pediatric vertebral injuries in the literature indicate the adolescent period (9). We believe that the increased frequency of vertebral injuries during adolescence is related to an increase in risky behaviors due to age.

In our study, the median hospital stay was 1 day (IQR: 0–4.25), while the median intensive care unit (ICU) stay was 1 day (IQR: 0–2). In the literature, hospital and ICU lengths of stay are significantly longer compared to our findings (10). We believe the primary difference between the reported long hospital and ICU lengths of stay and our findings stems from differences in the mechanism of injury and the case profiles of the centers. The reported longer lengths of stay may be attributed to the primary injury mechanism being high-energy traffic accidents leading to multiple trauma. In contrast, in our series, the predominance of a lower-energy fall mechanism—which reduces the risk of systemic complications—is considered one of the factors shortening the length of stay.

Additionally, since most data in the literature are obtained from specific trauma centers, this leads to a concentration of more severely ill patients—those referred to these centers—in such studies. We believe that the patient profile at our general tertiary care hospital, which reflects

a broader clinical spectrum compared to trauma centers and features a higher proportion of stabilizable cases, is the primary factor explaining this difference.

In complete blood count data, the mean white blood cell (WBC) count was determined to be 12.3 ± 5.9 × 10^3/μL. Similar results are found in the literature (11). Additionally, in the literature, a value of 12.35 × 10^3/μL is considered a cutoff for neurological recovery.

Additionally, a value of 12.35 × 10^3/μL has been identified in the literature as a critical threshold for neurological recovery; we believe that the fact that the mean WBC value observed in our study was below this threshold indicates that the cases in our study generally have a good prognosis (11). Furthermore, we believe that elevated white blood cell counts in pediatric vertebral injuries are due to an acute stress response.

In our study, the mean hematocrit (Hct) values for our cases were 40.9 ± 5.5%, and the mean hemoglobin (Hb) values were 13.7 ± 1.8 g/dL. Lower values for Hb and Hct have been reported in the literature (10). We attribute this difference to the absence of intra-abdominal solid organ injuries in our case series, the absence of additional pathologies that could cause open fractures or significant blood loss, and the fact that our patients were hemodynamically stable.

In our study, the median lymphocyte count was 2.38 × 10^3/μL (IQR: 1.86–3.17), albumin was 4.4 ± 0.5 g/dL, and the median LAR (lymphocyte-to-albumin ratio) was 0.56 (IQR: 0.39–0.7).

Lower mean levels of lymphocyte count, albumin, and LAR (lymphocyte-to-albumin ratio) have been reported in the literature (11). In the relevant study, lower lymphocyte counts, albumin, and LAR (lymphocyte-to-albumin ratio) levels were associated with a poor prognosis; we believe that the generally favorable and moderate course observed in our case group explains this difference.

In our study, biochemical analyses revealed an average urea level of 30.5 ± 9.5 mg/dL and a creatinine level of 0.71 ± 0.14 mg/dL. The literature reports similar average renal function test results within normal limits in pediatric trauma cases (12). These results may be due to the absence of severe renal injuries at the time of initial trauma. Additionally, in such traumas, renal injury may occur as a result of rhabdomyolysis, a secondary injury mechanism; however, since the tests performed at the time of emergency department presentation were examined in our study, the absence of such renal injury—which is a late consequence of trauma—can be explained by the timing of the tests.

In our study, serum glucose levels were found to have a median of 102 mg/dL (IQR: 94.5–130.5) in biochemical analyses. Higher average glucose levels have been reported in the literature (10). We believe this difference can be explained by considering stress-induced hyperglycemia mediated by catecholamines and glucocorticoids within the context of the stress response. That is, traumas causing more severe injuries elicit a higher stress response in

the body; however, in our study, a better prognosis was observed compared to similar studies in the literature, which may not have triggered a stress-induced hyperglycemic response.

In our study, biochemical analyses revealed a median AST level of 35 U/L (IQR: 22–40) and a median ALT level of 17.5 U/L (IQR: 14–22.2); these values fall within the normal range for the pediatric age group. Similar results have been reported in the literature (12). Considering that values above 200 U/L for AST and 125 U/L for ALT have been identified as critical thresholds for predicting liver injury in the literature, we believe that these results falling within normal limits can be explained by the absence of liver injury accompanying vertebral injuries or the absence of widespread skeletal muscle breakdown.

A significant finding of our study is the statistical association between injury location and serum urea levels (p=0.017). Notably, urea levels in cases with lumbar fractures were found to be significantly lower compared to both cervical (p=0.002) and thoracic (p=0.032) injuries. The literature emphasizes that upper-level spinal injuries are associated with more severe sympathetic activation and protein catabolism compared to lower-level injuries (3). This indicates that metabolic stress intensifies as the level of trauma increases. We believe our findings demonstrate that the systemic metabolic burden caused by cervical and thoracic injuries is more pronounced than that caused by lumbar injuries. This finding supports the need for a more aggressive approach to the biochemical monitoring of pediatric cases with severe injuries.

CONCLUSION

This study demonstrates that the most common mechanism of injury in pediatric vertebral fractures is falls (66.6%) and that this condition is associated with a more stable clinical course and shorter hospital stays compared to traffic accident-dominated series in the literature. The fact that laboratory parameters such as WBC, LAR, and glucose remained below critical threshold values in our cases demonstrates that the systemic inflammatory response and metabolic stress remain at limited levels in low-energy trauma. The most distinctive finding of our study is that as the injury level increases (cervical and thoracic), serum urea levels rise significantly compared to the lumbar region, highlighting the decisive effect of anatomical location on metabolic catabolism. Hemoglobin and liver enzyme levels within normal ranges indicate that hemodynamic stability and organ function are successfully maintained in cases of isolated pediatric vertebral injuries. In conclusion, given the increased metabolic stress burden in pediatric cases with high-level spinal injuries, it is considered essential that biochemical monitoring be followed as closely as radiological findings in clinical management.

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A RARE EFFECT OF CALENDULA OFFICINALIS TEA: OBSTRUCTIVE JAUNDICE

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INTRODUCTION

This case represents an important example demonstrating the potential risks of herbal products that may require medical intervention. The patient's dyspeptic complaints—abdominal pain and distension without jaundice, thought to be secondary to biliary obstruction—progressed to toxic hepatitis following the use of Calendula officinalis tea. This highlights the necessity of using herbal products only under medical supervision and at appropriate doses. The hepatotoxic effects of herbal teas and other herbal products are well documented; excessive use may result in severe liver injury. Symptoms of toxic hepatitis include jaundice (yellowing of the skin and sclera), dark urine, pale stools, abdominal pain, nausea, and vomiting. Diagnosis is supported by abnormalities in liver function tests and imaging studies (e.g., liver ultrasonography). Treatment generally includes liver support, symptomatic management, and supportive care; in severe cases, liver transplantation may be required [1,2].

This case report aims to inform emergency physicians and medical students about the adverse effects of herbal products. Awareness of the potential risks of Calendula officinalis is crucial for patient safety and effective management.

CASE PRESENTATION

A 62-year-old female patient was brought to our emergency department by the 112 emergency medical services from Karaman State Hospital under the decision of the Provincial Emergency Health Services Coordination Commission (ASKOM) with a complaint of jaundice. Further history revealed that she had undergone deep brain stimulation surgery four months earlier for Parkinson's disease and had been experiencing abdominal distension and persistent abdominal pain for the past two months. Despite multiple hospital visits and treatments, no relief was achieved. Upon recommendation from relatives, the patient began consuming Calendula officinalis tea nine days prior to admission. Jaundice developed five days after starting the herbal tea. She initially presented to Karaman State Hospital

Emergency Department and was referred to our center with a preliminary diagnosis of obstructive jaundice and toxic hepatitis.

On physical examination, gross icterus was observed. Abdominal examination revealed tenderness in the right upper quadrant and epigastric region. There was no guarding, rebound tenderness, or Murphy's sign.

The patient's only known comorbidity was Parkinson's disease. Surgical history included deep brain stimulation surgery four months earlier and total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO). Current medications included Dopadex SR 50 mg/200 mg (1×1) and Domirpa 100 mg/25 mg tablet (4×1). Vital signs were as follows: pulse 102 beats/min, blood pressure 139/94 mmHg, and oxygen saturation 91% on room air. Oral intake was discontinued immediately, and intravenous fluid support was initiated.

Laboratory tests performed at Karaman State Hospital revealed total bilirubin 2.5 mg/dL, alkaline phosphatase 300 U/L, AST 150 U/L, and GGT 200 U/L. At our hospital, results were similar: total bilirubin 2.7 mg/dL, alkaline phosphatase 280 U/L, AST 160 U/L, and GGT 220 U/L. Hepatobiliary ultrasonography was requested, and consultations with Internal Medicine (Gastroenterology) and General Surgery were initiated immediately. Computed tomography performed at Karaman State Hospital reported:"The gallbladder is in normal localization and size, with edematous wall. Intrahepatic bile ducts are diffusely dilated. The common bile duct could not be clearly visualized; MRCP is recommended."

Ultrasonography at our hospital reported: "Dilated intrahepatic bile ducts and main hepatic duct (main hepatic duct diameter 12 mm). The common bile duct could not be visualized sonographically. MRCP is recommended for differentiation between cholangiocarcinoma and stone. The gallbladder is contracted and filled with stones and sludge." Based on laboratory and radiological findings, General Surgery recommended Gastroenterology consultation for suspected toxic hepatitis and obstructive jaundice. The Gastroenterology Department accepted the patient for admission to the Gastroenterology Intensive Care Unit.

DISCUSSION

A review of the literature suggests that this may be the first reported case of herbal tea–associated obstructive jaundice. A similar case described acute cholestatic hepatitis following Artemisia annua tea consumption used as malaria chemoprophylaxis in a patient returning from Ethiopia [3]. Both patients were of advanced age (the Ethiopian case involved a 51-year-old patient). Gross icterus and abdominal pain were prominent in both cases. Laboratory findings in both cases showed elevated transaminases and cholestatic parameters consistent with obstructive jaundice.

The limited number of reported cases indicates that further scientific investigation in this field is needed. This remains an important issue requiring attention from clinicians.

CONCLUSION

This case provides important lessons regarding the use of herbal products. Patients must use herbal products strictly under physician supervision and must not exceed recommended doses. It should be remembered that certain herbs, such as Calendula officinalis, may have unexpected adverse effects and may lead to severe liver injury. The perception that herbal products are inherently safe because they are natural may lead to underestimation of potential risks. Interactions between herbal products and prescribed medications should also be considered, as some herbs may reduce drug efficacy or increase adverse effects. Patients should inform their physicians about all medications and supplements they are using. Furthermore, patients carrying medical devices (such as a deep brain stimulation controller) should be properly informed and prepared for emergencies.

In conclusion, awareness of the potential risks of herbal products and appropriate medical supervision are essential for patient safety. This case serves as an important warning for both healthcare professionals and patients

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Keywords: Obstructive Jaundice, Toxic Hepatitis, Herbal Tea, Calendula officinalis flower

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Ending the "Cardiopulmonary Ping-Pong": A Case of HFpEF-Induced Acute Right Heart Failure

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Introduction

Taking care of older people who come to the emergency room with shortness of breath is one of the hardest and most complicated parts of emergency medicine [1]. Dyspnea can be caused by a lot of different things, including problems with the heart or lungs, or by problems in both at the same time. It is hard to figure out what caused the main problem, especially when there is acute heart failure (AHF) and diseases of the respiratory system at the same time. Patients can get stuck between cardiology and pulmonology offices because of this lack of clarity. This is known as "cardiopulmonary ping-pong" in the medical world.

Heart failure with preserved left ventricular ejection fraction (HFpEF) is becoming more common, especially in older women and women with high blood pressure [2]. In people with HFpEF, diastolic dysfunction can cause the left ventricle to fill with more blood, which can cause pulmonary venous congestion, secondary pulmonary hypertension, and eventually right heart failure. A substance called BNP and another called NT-proBNP are released when the myocardium contracts. They are very good at finding people who have heart failure [3]. This report talks about a very serious case of acute right heart failure that was left in limbo during consultations between departments because of its unusual presentation and stable LVEF value. However, the emergency expert was able to handle it with his clinical synthesis.

Case

A woman who was 79 years old and had a history of high blood pressure went to the emergency room because her legs were swollen and she was short of breath. The person was awake and aware when they were admitted, with a Glasgow Coma Scale (GCS) score of 15. The vital signs showed that the heart rate was 85 beats per minute, the arterial blood pressure was 170/100 mmHg, and the oxygen saturation in room air was 60%. With oxygen support, the saturation rose to 95%. At the physical exam, there was widespread pretibial edema (+/+) on both sides and widespread rales could be heard in both lungs.

Radiological tests did not show a pulmonary embolism, but they did show that both lungs had a lot of congestion. The lab tests showed that the NT-proBNP level was 5500 pg/mL, the WBC count was 15,000/µL, the creatinine level was 0.96 mg/dL, the ALT level was 2000 U/L, the AST level was 2000 U/L, the Troponin I level was 44 ng/L, the CRP level was 71 mg/L, and the pH of the venous blood gas was 7.43. Cardiology did a bedside echocardiogram (ECHO) and saw that the right chambers were much bigger than the left ones. There was Stage 2 tricuspid regurgitation (TR), the pulmonary artery pressure (PAP) was 96 mmHg, and the left ventricular ejection fraction (LVEF) was 60%. Because the patient was having trouble breathing and their ejection fraction stayed the same, they were stuck between heart and chest diseases. The patient was taken directly to the Coronary Intensive Care Unit after the emergency medicine expert looked at all the information and came up with a comprehensive plan.

Discussion

In this case, the akut dekompanse kalp yetmezliği and kardiyopulmoner yetmezlik spectrum is a very important part of the study. This is a hypothesis about the heart's basal volume, artmış kan basıncı, yaygın raller ve pretibial ödemi; hem sistemik hem de pulmoner venöz sistemde masif konjesyonun varlığını doğrulamaktadır.

LVEF'sin %60 olarak saptanmasıdır. Klinik pratikte hekimleri en çok ikilemde bırakan durum. LVEF'nin korunmuş olması, sıklıkla solunum kaynaklı primer bir akciğer patolojisi algısı yaratsa da; NT-proBNP düzeyinin 5500 pg/mL gibi belirgin yüksek bir seviyede olması tablonun ön planda kardiyovasküler kaynaklı bir basınç/hacim yüklenmesi (HFpEF) olduğunu kanıtlamaktadır [2,3].

Hastanın laboratuvar ve ekokardiyografi bulguları birlikte incelendiğinde tablonun şiddeti netleşmektedir. Ekokardiyografideki masif sağ kalp yüklenmesi ve 96 mmHg olarak ölçülen sPAB, ciddi sekonder pulmoner hipertansiyonu göstermektedir [4]. This long-term systemic venöz system has an analytical base that doesn't show anything, and the laboratory sonuçlarında ALT ve AST değerlerinin 2000 U/L seviyelerine ulaşmasına yol açmıştır. Hepatitis C ("şok karaciğeri" veya iskemik hepatit) tipik ve ölümcül olabilir bulunan akut sağ kalp yetmezliğine bağlı gelişen bir göstergesidir [5].

Beyaz küre ve CRP yüksekliği altta yatan tetikleyici bir inflamatuar sürece işaret edebileceği gibi masif venöz stazından bir sonucudur. When troponin levels are high, the first thing that happens is that the acoustic cortex sendroman ziyade, and when the acoustic cortex sağ kalp yetmezliğindeki miyokardiyal gerilime bağlı tip 2 iskemi ve troponin sızıntısı ile uyumludur [6].

Conclusion

Even if the left ventricular ejection fraction is within normal limits, there is still a chance of massive pulmonary hypertension, which can cause a lot of death, serious right heart decompensation, and damage to other organs. When an older person is admitted to the hospital right away, heart failure on the right side, along with high liver enzymes and cardiac biomarkers, is a medical emergency that needs prompt care. It is very important for the emergency medicine expert to be able to settle disagreements between specialists and make sure that the patient is sent to the right intensive care unit based on the results of the lab tests, physical exam, and imaging.

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Keywords: Akut kalp yetmezliği, HFpEF, kor pulmonale, şok karaciğeri, acil tıp

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Cerebral Malperfusion and Extremity Ischemia Resulting from Type 1 Aortic Dissection: A Case Report

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Introduction

Acute aortic dissection (AD) occurs when a tear in the tunica media layer of the aortic wall creates a separation between a true and a false lumen. According to the Stanford classification, cases involving the ascending aorta are categorized as Type A (encompassing DeBakey Types 1 and 2) and necessitate immediate surgical intervention (1). While the classic presentation involves sudden, "tearing" pain, a significant portion of patients present with neurological or vascular findings due to malperfusion syndromes (2). Neurological symptoms result from the occlusion of the carotid or cerebral arteries by the dissection flap, which can mimic a stroke and potentially mask the primary diagnosis (3). This case presents an AD diagnosis reached via pulse examination in a patient presenting with syncope and neurological deficits.

Case Presentation

A 72-year-old female with a history of hypertension (HT) experienced a sudden onset of severe chest and back pain at approximately 14:00. Following the pain, the patient experienced syncope; upon regaining consciousness, a speech disorder was noted. A brain CT performed at an external center was reported as normal; however, she was referred to our facility for further evaluation due to a 4/5 motor strength loss in the left arm.

During the initial examination in our emergency department, her blood pressure was 110/70 mmHg, heart rate was 53/min (bradycardic), and SpO2 was 97%. The ECG showed sinus bradycardia. Neurological assessment confirmed dysarthria and 4/5 strength in the left arm. A systematic physical examination revealed that bilateral femoral pulses were manually impalpable. Within the stroke protocol, a brain MRI was performed, revealing acute infarcts in the cerebellum and occipital lobes (Figure 1).

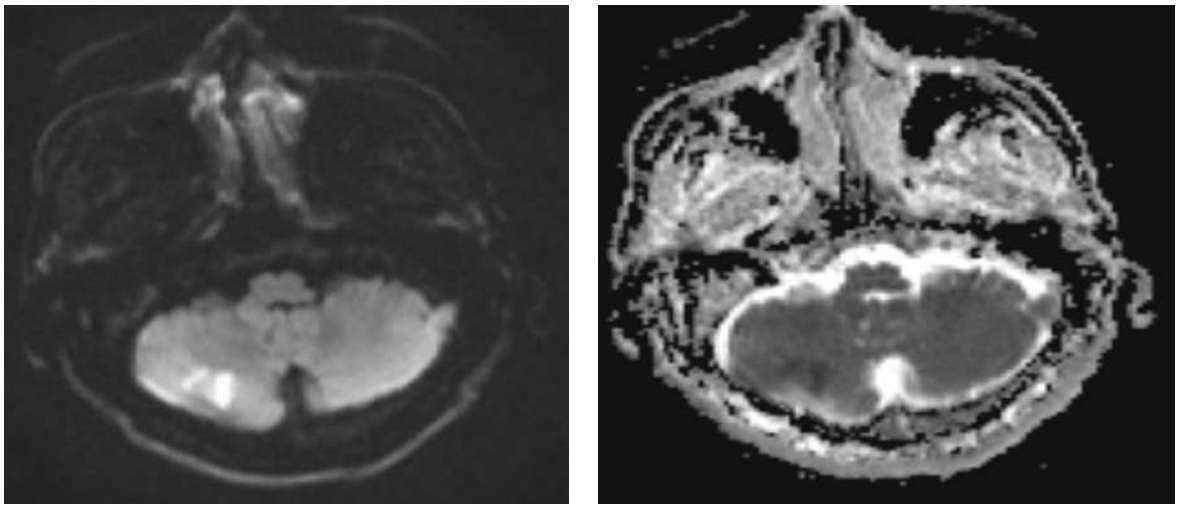

Figure 6 The Patient's Brain MRI Scan

Given the combination of femoral pulse loss and back pain, an urgent CT angiography was requested under the suspicion of AD. The imaging revealed a Type 1 AD extending from the ascending aorta to the iliac arteries, involving the carotid artery origin, which was completely occluded (Figure 2). Interventional radiology did not plan a procedure for the carotid occlusion, and the patient was transferred to Cardiovascular Surgery (CVS) for an emergency operation due to hemodynamic instability and signs of malperfusion.

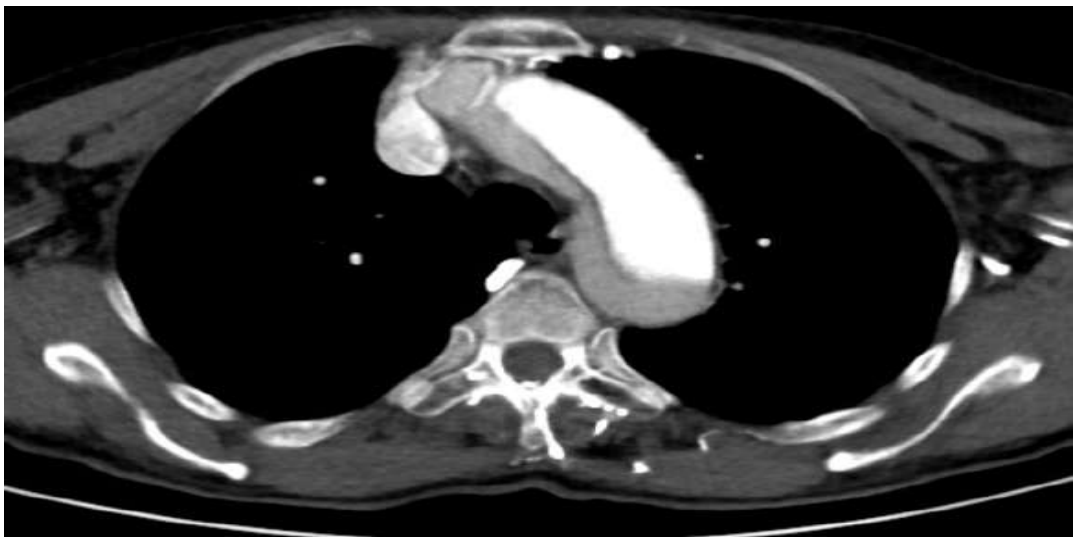

Figure 7 The Patient's Thoracic CT Angiography

Discussion

Acute Type A aortic dissection has a mortality rate that increases by 1-2% per hour during the first 48 hours if left untreated (4). Known in the literature as "the great masquerader," AD can present as a stroke or syncope due to the disruption of cerebral blood flow (3). The presence of neurological deficits in cases with carotid artery involvement may lead clinicians toward primary cerebrovascular events; this carries the risk of administering thrombolytic therapies, which are contraindicated in AD management (1,2).

In our case, although an external facility followed a stroke-oriented approach, the inability to palpate femoral pulses in our emergency department served as the critical diagnostic clue. A femoral pulse deficit is a significant indicator of vascular malperfusion, suggesting that the dissection has extended distally to the iliac arteries (4,5). The bradycardic course (pulse: 53) and history of syncope should serve as warning signs for the extension of the dissection into the aortic root and coronary arteries, or the development of a pericardial effusion (tamponade) (1). The complete occlusion of the carotid artery, combined with the posterior circulation infarct, proved that the AD affected all major branches of the aortic arch.

Conclusion

Bilateral pulse control must be an integral part of the physical examination for elderly and hypertensive patients presenting to the emergency department with syncope or neurological deficits. Neurological findings occurring after chest pain must be considered aortic dissection until proven otherwise. Early diagnosis and rapid coordination with CVS are the cornerstones of managing this high-mortality condition.

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Keywords: Aortic dissection, Syncope, Ischemic stroke, Malperfusion syndrome

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Temporal Changes and Clinical Significance of Monocyte Levels in Geriatric Patients with Urosepsis

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ABSTRACT

Objective:
Urosepsis is a serious clinical condition associated with high mortality and morbidity, particularly in geriatric patients. Although various biomarkers are used to evaluate the inflammatory response during sepsis, the temporal changes in monocyte levels have not been sufficiently elucidated. This study aimed to evaluate the changes in monocyte levels over time in geriatric patients with urosepsis.

MATERIALS AND METHODS:

This retrospective observational study included 63 patients aged 65 years and older who were diagnosed with urosepsis in the emergency department or intensive care unit within the last year. Demographic data and laboratory parameters were obtained from the hospital information management system. Monocyte levels were measured at two different time points and recorded in $\times 10^9$/L. Statistical analyses were performed using IBM SPSS Statistics version 27.0. Data distribution was assessed using the Kolmogorov–Smirnov test, and the relationship between the two measurements was evaluated using Pearson or Spearman correlation analysis.

RESULTS:

Of the included patients, 50.8% were female, and the mean age was 74 ± 3 years. The mean monocyte level was 0.4832 ± 0.2949 $\times 10^9$/L at the first measurement and 0.6268 ± 0.3710 $\times 10^9$/L at the second measurement. These findings indicate an increasing trend in monocyte levels during the follow-up period. Correlation analysis demonstrated a statistically significant positive relationship between the two measurements ($r = 0.316$, $p < 0.001$).

CONCLUSION:

Monocyte levels increase over time in geriatric patients with urosepsis. Dynamic monitoring of monocyte levels may serve as a potential biomarker for evaluating the inflammatory response and supporting clinical follow-up.

Keywords:
Urosepsis, Monocyte, Geriatric patient, Sepsis, Inflammation

INTRODUCTION

Urosepsis is an important clinical condition associated with high mortality and morbidity, particularly among elderly individuals, and is currently defined as a life-threatening organ dysfunction caused by a dysregulated host response to infection. Age-related alterations in the immune system contribute to differences in infection response and may worsen the clinical course in geriatric patients.

Inflammatory response assessment in sepsis commonly involves neutrophil-, lymphocyte-, and platelet-based parameters. However, monocytes are key components of the innate immune system and play active roles in cytokine production, phagocytosis, and antigen presentation.

Changes in monocyte function during sepsis have been shown to be critical in the immunopathogenesis of the disease and in the regulation of immune responses. Recent studies have suggested that monocyte activation and monocyte-related biomarkers may be associated with sepsis prognosis and could be useful in predicting clinical outcomes. Nevertheless, data regarding the temporal changes in monocyte levels among geriatric patients with urosepsis remain limited.

The aim of this study was to evaluate the changes in monocyte levels over time in geriatric patients admitted through the emergency department and subsequently followed in the intensive care unit due to urosepsis, and to investigate the potential clinical significance of these changes.

MATERIALS AND METHODS

This retrospective observational study was designed to evaluate changes in monocyte levels among geriatric patients hospitalized in the intensive care unit with a diagnosis of urosepsis. A total of 63 patients aged 65 years and older who were diagnosed with urosepsis within the previous year in the emergency department or intensive care unit were included.

The diagnosis of urosepsis was established based on clinical findings, laboratory parameters, and, when necessary, imaging studies demonstrating a urinary tract source of infection. Inclusion criteria were age ≥65 years, diagnosis of urosepsis, and complete laboratory records. Patients with hematological malignancies, those receiving immunosuppressive therapy, and patients with incomplete data were excluded.

Demographic data and laboratory parameters were retrospectively collected from the hospital information management system. Monocyte levels were evaluated at two different time points and recorded in $\times 10^9$/L.

STATISTICAL ANALYSIS

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 27.0. The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Normally distributed data were expressed as mean ± standard deviation. The relationship between the two measurements was evaluated using Pearson or Spearman correlation analysis, and a p-value <0.05 was considered statistically significant.

RESULTS

A total of 63 geriatric patients diagnosed with urosepsis were included in the study. Of these patients, 32 (50.8%) were female and 31 (49.2%) were male. The mean age was 74 ± 3 years. Evaluation of temporal changes in monocyte levels revealed that the mean monocyte count

increased from 0.4832 ± 0.2949 ×10⁹/L at the first measurement to 0.6268 ± 0.3710 ×10⁹/L at the second measurement, indicating an upward trend during the follow-up period. Correlation analysis demonstrated a statistically significant positive association between the two monocyte measurements ($r = 0.316$, $p < 0.001$).

DISCUSSION

In the present study, we demonstrated that monocyte levels increased over time in geriatric patients with urosepsis and that a significant positive relationship existed between the two measurements. Sepsis is a complex immune response syndrome involving both pro-inflammatory and anti-inflammatory mechanisms.

Monocytes occupy a central role in this process through cytokine production, phagocytosis, and antigen presentation. Alterations in monocyte function during sepsis have been associated with disease severity and clinical outcomes.

In geriatric patients, immunosenescence leads to alterations in immune responses, resulting in a more dysregulated response to infections. Therefore, the dynamics of monocyte levels may differ in elderly populations.

Although the prognostic value of inflammatory indices such as the neutrophil-to-lymphocyte ratio has been extensively investigated, studies focusing specifically on temporal changes in monocyte levels remain limited. Our findings suggest that monocyte levels are not static parameters during intensive care follow-up and may dynamically reflect ongoing inflammatory processes in sepsis.

These results indicate that monitoring monocyte levels may provide additional information regarding the persistence of the inflammatory response and may serve as a useful adjunct biomarker in clinical follow-up.

CONCLUSION

Monocyte levels increase over time in geriatric patients with urosepsis. Dynamic monitoring of monocyte levels may assist in the evaluation of inflammatory responses and support clinical management during follow-up.

LIMITATIONS

This study has several limitations. First, it was a single-center retrospective study with a relatively limited sample size. Second, the relationship between monocyte levels and clinical outcomes such as mortality was not evaluated.

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7331

DETECTION OF OVARIAN TORSION WITH COMPUTED TOMOGRAPHY IN A PATIENT WITH A PRELIMINARY DIAGNOSIS OF ACUTE ABDOMEN

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INTRODUCTION

Ovarian torsion is a time-sensitive gynecological emergency caused by partial or complete rotation of the ovary and/or adnexa around the vascular pedicle, resulting initially in venous and lymphatic obstruction and, if untreated, arterial compromise, ischemia, hemorrhagic infarction, and potential loss of ovarian function [1,2]. Although it may occur at any age, recognition is particularly challenging in children and adolescents because symptoms often overlap with gastrointestinal, urinary, and surgical causes of acute abdomen. Sudden-onset lower abdominal or pelvic pain accompanied by nausea and vomiting should raise clinical suspicion; however, physical examination and laboratory findings may be non-specific or even normal, as seen in the present case [1].

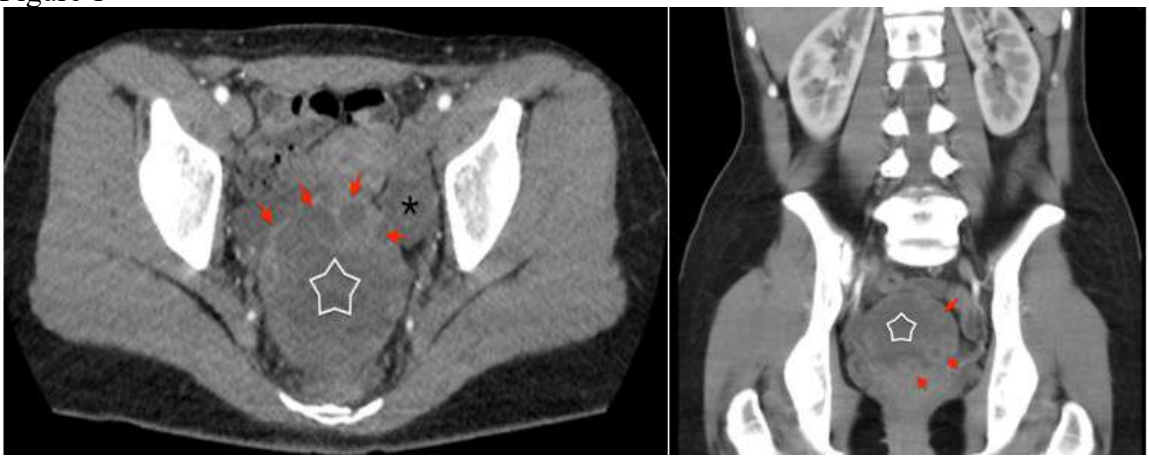
Ultrasonography (USG) with Doppler evaluation is generally accepted as the first-line imaging modality for suspected ovarian torsion, especially in pediatric and adolescent patients. Nevertheless, Doppler findings must be interpreted cautiously, because preserved or peripheral blood flow does not exclude torsion due to the dual ovarian blood supply and the possibility of intermittent or incomplete torsion [1,3]. In emergency practice, computed tomography (CT) is frequently performed before pelvic ultrasonography when the initial clinical picture suggests appendicitis, intestinal obstruction, renal colic, or another cause of acute abdomen. Therefore, radiologists and emergency physicians should be familiar with CT findings that may suggest ovarian torsion. The most relevant findings include unilateral

ovarian enlargement, stromal edema, peripheral displacement of follicles, an associated cyst or mass, deviation of the uterus toward the affected side, pelvic free fluid, abnormal enhancement, and twisting or malrotation of the vascular pedicle [2-4]. This case highlights the diagnostic value of carefully evaluating the ovaries on CT in an adolescent patient who presented with non-specific acute abdominal symptoms.

CASE

A 14-year-old female presented with sudden, severe right lower quadrant pain, vomiting, and lack of bowel output. Symptoms were unrelated to her menstrual cycle. Physical examination revealed a soft abdomen without defence and rebound. Laboratory results were normal. Following an unresponsive enema for colonic gas distension seen on radiography, contrast-enhanced CT was performed to exclude acute pathologies. In the contrast-enhanced CT, an increase in the size of the right ovary and peripheral displacement of the follicles were observed (Figure 1). Due to suspected ovarian torsion, pelvic Doppler USG was performed. On USG, the right ovarian parenchyma appeared edematous, containing a central cystic area 55 mm in diameter, with the follicles displaced to the periphery. On Doppler ultrasound, flow was only detected at the periphery and was high-resistant. The patient, diagnosed with ovarian torsion, was consulted with the Department of gynecology for emergency surgical planning.

Figure 1



Axial (left) and coronal (right) CT images. Enlarged right ovary (white star). Peripherally displaced follicles in the right ovary (red arrows). Normal left ovary (black asterisk)

DISCUSSION

The present case demonstrates how ovarian torsion may initially mimic a non-gynecological acute abdominal condition. The patient presented with severe right lower quadrant pain, vomiting, and reduced bowel output, while physical examination and laboratory tests did not reveal specific inflammatory or surgical findings. Under these circumstances, contrast-enhanced CT was performed to exclude common acute abdominal pathologies. The decisive CT clues were enlargement of the right ovary and peripheral displacement of follicles, both of which are characteristic imaging findings of ovarian torsion [2,3]. Recognition of these

findings prompted targeted pelvic Doppler ultrasonography and urgent gynecological consultation, thereby shortening the diagnostic pathway.

A key teaching point is that ovarian torsion should not be excluded solely on the basis of Doppler flow. In this case, flow was detected only at the periphery and showed a high-resistance pattern, supporting impaired vascularity; however, the literature emphasizes that normal or partially preserved flow may still be encountered in surgically proven torsion [1]. Thus, imaging interpretation should integrate ovarian size, follicular distribution, stromal edema, vascular configuration, and the clinical scenario rather than relying on a single parameter. CT is not the preferred first-line modality for suspected torsion because of radiation exposure, particularly in adolescents, but it may become diagnostically valuable when obtained for acute abdomen of uncertain origin [2,4].

Early diagnosis is clinically important because timely surgical evaluation increases the likelihood of detorsion and preservation of ovarian tissue. Current recommendations emphasize that adnexal torsion is ultimately a surgical diagnosis and that ovarian-sparing management should be pursued whenever feasible, even when the ovary appears ischemic intraoperatively [1]. From a practical perspective, this case underlines the need for systematic inspection of the adnexa on abdominal CT examinations performed in female patients with acute lower abdominal pain. In adolescents, the combination of acute pain, vomiting, unilateral ovarian enlargement, and peripheral follicles should immediately raise suspicion for torsion and trigger urgent multidisciplinary communication.

Keywords: Acute abdomen, CT, ovarian torsion

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Sore throat and critical multi-vessel disease masked by supraventricular tachycardia: A case report

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Abstract

In this case report, we describe the management of a rare presentation in which a patient with symptoms suggestive of an upper respiratory tract infection and supraventricular tachycardia was ultimately found to have critical multivessel coronary artery disease. A 59-year-old man with no known comorbidities and a 17-year history of non-smoking presented to the emergency department with a three-day history of sore throat, fatigue, cough, and generalised body aches. On initial examination, the patient had no active chest pain or fever; however, his vital signs revealed a heart rate of 180 beats per minute. Electrocardiography demonstrated supraventricular tachycardia. Following intravenous administration of diltiazem, the heart rate decreased to 83 beats per minute and normal sinus rhythm was restored. Laboratory investigations revealed that the initial troponin I level of 24.40 ng/L increased markedly to 1653.31 ng/L during follow-up. Additional laboratory results included a creatinine level of 1.5 mg/dL, lactate dehydrogenase of 631 IU/L, creatine kinase of 293 U/L, and C-reactive protein of 65.01 mg/L. Posterior–anterior chest radiography demonstrated a non-homogeneous increase in density in the lower lung zones. Due to the significant elevation in cardiac enzymes, the patient was referred for coronary angiography with a diagnosis of non-ST-segment elevation myocardial infarction (NSTEMI). Coronary angiography revealed 95% stenosis of the proximal left anterior descending artery and sequential 90% stenoses in the proximal right coronary artery. The patient was subsequently referred for emergency coronary artery bypass graft surgery. In patients presenting to the emergency department with isolated sore throat, fatigue, or tachycardia, acute coronary syndromes should be considered in the differential diagnosis, particularly in the presence of cardiovascular risk factors. Atypical symptoms may create a diagnostic trap in high-risk patients and lead to delayed recognition of myocardial injury.

Keywords: Acute coronary syndrome, Sore throat, Supraventricular tachycardia.

Introduction

Typical retrosternal chest pain is the most common presenting symptom of acute coronary syndromes; however, a considerable proportion of patients present with atypical symptoms such as sore throat, fatigue, or dyspnoea (1). Arrhythmias such as supraventricular tachycardia may unmask previously stable coronary artery disease or directly precipitate myocardial ischaemia by increasing myocardial oxygen demand (2).

Previous studies have reported that cardiac pain may occasionally be perceived in the throat region as referred pain, which may contribute to diagnostic delays (3). In this case report, we describe the management of a rare presentation in which a patient with symptoms suggestive of an upper respiratory tract infection and supraventricular tachycardia was ultimately found to have critical multivessel coronary artery disease.

Case Presentation

A 59-year-old man with no known comorbidities and a 17-year history of non-smoking presented to the emergency department with a three-day history of sore throat, fatigue, cough, and generalised body aches. On initial examination, the patient had no active chest pain or fever; however, his vital signs revealed a heart rate of 180 beats per minute. Electrocardiography demonstrated supraventricular tachycardia. Following intravenous administration of diltiazem, the heart rate decreased to 83 beats per minute and normal sinus rhythm was restored. Laboratory investigations revealed that the initial troponin I level of 24.40 ng/L increased markedly to 1653.31 ng/L during follow-up. Additional laboratory results included a creatinine level of 1.5 mg/dL, lactate dehydrogenase of 631 IU/L, creatine kinase of 293 U/L, and C-reactive protein of 65.01 mg/L. Posterior–anterior chest radiography demonstrated a non-homogeneous increase in density in the lower lung zones. Due to the significant elevation in cardiac enzymes, the patient was referred for coronary angiography with a diagnosis of non-ST-segment elevation myocardial infarction (NSTEMI). Coronary angiography revealed 95% stenosis of the proximal left anterior descending artery and sequential 90% stenoses in the proximal right coronary artery. The patient was subsequently referred for emergency coronary artery bypass graft surgery.

Discussion

In emergency medicine practices, the diagnostic challenges posed by atypical presentations and the critical importance of maintaining clinical suspicion have been emphasised in several studies (3). Our case supports these observations.

It has been reported that a substantial proportion of patients with acute myocardial infarction may present with atypical symptoms such as isolated sore throat or jaw pain in the absence of classical chest pain (4). In the present case, the patient's complaint of sore throat initially created a misleading clinical picture; however, the concomitant presence of supraventricular tachycardia raised suspicion of an underlying ischaemic process.

From a pathophysiological perspective, supraventricular tachycardia significantly shortens the duration of diastole in the cardiac cycle, thereby reducing coronary perfusion time while simultaneously increasing myocardial oxygen demand. This supply–demand imbalance has been reported to cause myocardial injury in both normal coronary arteries and vessels with significant stenosis (2).

The detection of near-total stenosis in both the left anterior descending and right coronary arteries in our patient supports the view that supraventricular tachycardia may represent not merely an arrhythmia but also a clinical manifestation of a critical coronary event (5).

Conclusion

In patients presenting to the emergency department with isolated sore throat, fatigue, or tachycardia, acute coronary syndromes should be considered in the differential diagnosis, particularly in the presence of cardiovascular risk factors. Atypical symptoms may create a diagnostic trap in high-risk patients and lead to delayed recognition of myocardial injury.

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7333

A Rare Cause of Acute Dyspnea: Pneumomediastinum in a Patient with a History of Nasal Surgery

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INTRODUCTION

Pneumomediastinum is a rare condition defined by the presence of free air within the mediastinum and may lead to serious complications if not properly evaluated (1). It is classified as spontaneous or secondary.

The most accepted mechanism in spontaneous pneumomediastinum is the “Macklin effect,” in which alveolar rupture allows air to dissect along bronchovascular sheaths into the mediastinum (2,3). This process is typically triggered by increased intrathoracic pressure due to coughing, asthma exacerbation, or Valsalva maneuvers.

Secondary pneumomediastinum may occur after trauma, mechanical ventilation, esophageal perforation, or surgical procedures (2,4). Although rare, cases following head and neck surgery have been reported, possibly due to air spreading along fascial planes into the mediastinum (4,5).

Here, we present a case of pneumomediastinum in a young patient with a history of nasal surgery.

Case Presentation

A 24-year-old man presented to the emergency department with sudden-onset dyspnea. He reported intermittent dyspnea and productive cough over the previous months, partially relieved by bronchodilators. His history included septorhinoplasty three months earlier and a six pack-year smoking history.

On admission, SpO_2 was 85% on room air, heart rate 130 beats/min, blood pressure 115/45 mmHg, and temperature 36.5°C. Lung examination revealed bilateral rhonchi and wheezing. Arterial blood gas analysis showed mild respiratory alkalosis and hypoxemia (pH 7.46, pO_2 50 mmHg). C-reactive protein was 53 mg/L, and electrocardiography demonstrated normal sinus rhythm.

Chest radiography was unremarkable. Thoracic CT angiography, performed to exclude pulmonary embolism, revealed no embolism but demonstrated air densities consistent with pneumomediastinum surrounding the left subclavian artery and aortic arch.

The patient was evaluated by pulmonology and thoracic surgery and admitted for observation. Oxygen therapy, bronchodilators, and systemic corticosteroids were initiated.

Discussion

Pneumomediastinum is more common in young males and usually has a benign course; however, serious conditions such as esophageal perforation and pulmonary embolism must be excluded (2,5). Chest radiography may be normal, whereas thoracic CT is more sensitive (2,3,5).In this case, the three-month interval after surgery and the presence of airway

symptoms suggest spontaneous pneumomediastinum related to airway hyperreactivity rather than a direct surgical complication.
Pneumomediastinum should be considered in young patients presenting with sudden dyspnea, even when chest radiography is normal.

Keywords: Macklin effect; Pneumomediastinum; Septorhinoplasty; Spontaneous pneumomediastinum; Thoracic CT

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SURGICAL STABILIZATION OF RIB FRACTURES: A 3-CASE SERIES

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ABSTRACT

Introduction and Purpose: Blunt thoracic trauma frequently results in rib fractures, which, particularly in cases of multiple fractures or flail chest, are associated with severe respiratory failure, prolonged mechanical ventilation, and increased mortality. While traditional treatment relies on conservative approaches, Surgical Stabilization of Rib Fractures (SSRF) is increasingly recommended to reduce ventilation duration, intensive care unit stays, and pulmonary complications. This study aims to present the clinical characteristics, surgical indications, and early outcomes of patients who underwent chest wall stabilization due to rib fractures resulting from blunt thoracic trauma.

Materials and Methods: This case series evaluates three male patients (aged 42, 45, and 55 years) who presented to the emergency department following blunt thoracic trauma, such as falls and workplace injuries. Clinical and radiological evaluations revealed localized tenderness, dyspnea, multiple displaced rib fractures, significant chest wall deformity, and flail chest characterized by paradoxical respiratory movements. Due to deteriorating

respiratory function and chest wall instability, surgical stabilization was indicated. Under general anesthesia, anatomic reduction was achieved, and the fractured rib segments were fixed using titanium plate-and-screw systems at the targeted levels.

Results and Conclusion: Postoperatively, all patients experienced restored chest wall stability and significant improvements in their respiratory parameters. Effective and rapid pain control was achieved, highlighted by a notable reduction in pain scores. All three patients had an uneventful clinical course with no reported complications and were subsequently discharged in stable and good condition.

In cases of multiple rib fractures and flail chest resulting from blunt thoracic trauma, SSRF is a safe and effective treatment modality when appropriate patient selection is applied. In the presence of chest wall instability and displaced fractures, surgical stabilization serves as a superior alternative to conservative management by ensuring rapid early pain control, significantly improving respiratory function, enhancing patient comfort, and accelerating functional recovery without complications.

Keywords: Rib Fractures, Flail Chest, Pain Management

INTRODUCTION

Blunt thoracic trauma accounts for a significant portion of all trauma cases and is one of the leading causes of trauma-related mortality and morbidity (1). Rib fractures are among the most common injuries in these patients, occurring in approximately 10–40% of thoracic traumas (2). Rib fractures, particularly in the presence of multiple fractures or the development of flail chest, are associated with severe respiratory failure, prolonged mechanical ventilation requirements, and increased mortality (1,3).

Although the treatment of these patients traditionally relies on conservative approaches - including analgesia, respiratory support, and physiotherapy- interest in surgical stabilization methods has been steadily increasing in recent years (4). Surgical Stabilization of Rib Fractures (SSRF) is recommended, especially in flail chest cases, due to its ability to shorten ventilation duration, reduce intensive care unit (ICU) and hospital lengths of stay, and lower the incidence of pulmonary complications (3,4). Randomized controlled trials and meta-analyses have demonstrated that patients undergoing surgical stabilization experience a reduced incidence of pneumonia, more effective pain management, and accelerated functional recovery (4,5). Furthermore, recent guidelines support the early application of SSRF in appropriately selected patients (6).

This study aims to present the clinical characteristics, surgical indications, and early outcomes of four patients who underwent chest wall stabilization due to rib fractures resulting from blunt thoracic trauma, in conjunction with a review of the literature.

CASE 1

A 45-year-old male patient presented to the emergency department following a fall from height, complaining of severe pain in the left hemithorax and dyspnea. Physical examination revealed tenderness in the left chest wall and notable paradoxical chest wall movement during respiration.

Radiological imaging identified multiple fractures involving the left 4th, 5th, 6th, 7th, 8th, and 9th ribs. Based on the clinical presentation and imaging, a diagnosis of Flail Chest was established. Due to deteriorating respiratory function and chest wall instability, surgical stabilization was indicated.

Under general anesthesia, the fractured rib segments were exposed, and fixation was achieved using titanium plates and screws at the targeted levels. Postoperatively, a significant improvement in the patient's respiratory status was observed. Effective pain control was achieved, and no complications were reported. Following a stable clinical course, the patient was discharged in good condition (Figures 1, 2).

CASE 2

A 42-year-old male patient was evaluated following blunt thoracic trauma, presenting with pain in the left hemithorax and worsening discomfort during respiration. Physical examination revealed localized tenderness and a palpable deformity of the left chest wall.

Radiological imaging identified fractures of the left distal ribs with significant displacement of the segments, leading to visible chest wall deformity. Surgical stabilization was indicated due to the resulting chest wall instability and deformity.

Under general anesthesia, the displaced rib segments were exposed, and anatomic reduction was successfully achieved. Stabilization was performed using titanium plate-and-screw systems. Postoperatively, the patient experienced a significant reduction in pain scores, and chest wall stability was restored. With improved respiratory function and an uneventful clinical course without complications, the patient was discharged in stable condition (Figures 3, 4).

CASE 3

A 55-year-old male patient presented to the emergency department following blunt thoracic trauma sustained at his workplace. Physical examination revealed tenderness, crepitus, and paradoxical respiratory movements at the level of the 3rd, 4th, 5th, and 6th costochondral junctions on the left anterior chest wall, leading to a diagnosis of flail chest.

Thoracic computed tomography (CT) confirmed the flail segment, identifying displaced fractures at the corresponding levels. In addition to pain management and respiratory support, the patient underwent chest wall stabilization (surgical fixation).

In the postoperative period, a significant improvement in the patient's respiratory parameters was observed, and he was discharged without any complications (Figure 5).

CONCLUSION

In cases of multiple rib fractures -particularly those involving flail chest- resulting from blunt thoracic trauma, Surgical Stabilization of Rib Fractures (SSRF) is a safe and effective treatment modality when appropriate patient selection is applied. In this case series, patients undergoing surgical stabilization achieved rapid early pain control, significant improvement in respiratory function, and clinical recovery without complications.

These findings suggest that in the presence of chest wall instability and displaced fractures, surgical stabilization serves as a superior alternative to conservative management by enhancing patient comfort and accelerating functional recovery.

Figure 1

Figure 2

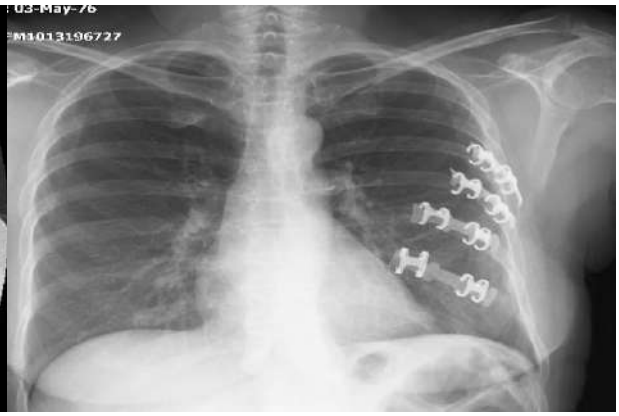


Figure 3

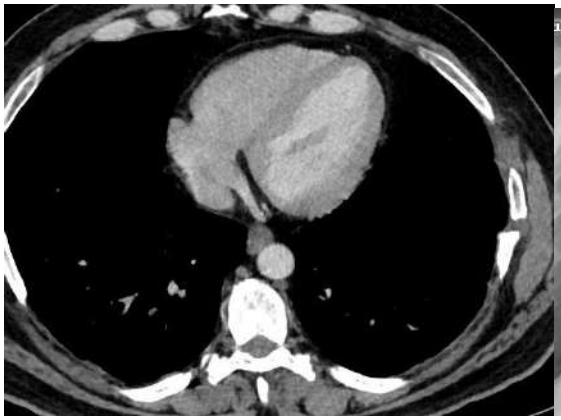
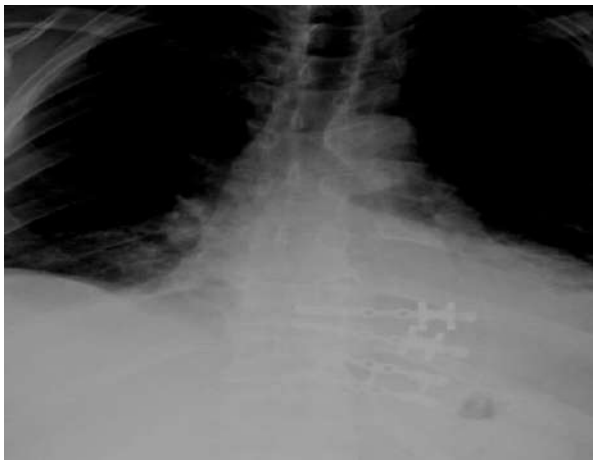


Figure 4

Figure 5



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Evaluation of Demographic and Haematological Parameters and the Role of Systemic Inflammatory Response Index (SIRI) in Patients with Acute Pancreatitis

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Abstract

Introduction: Acute pancreatitis is a disease characterised by the sudden onset of inflammation of the pancreas, with a clinical course ranging from mild inflammation to severe systemic complications. In recent years, haematological parameters obtained from complete blood counts have garnered attention as potential biomarkers for evaluating inflammation.

Objective: This study aimed to evaluate the demographic characteristics and laboratory parameters of patients diagnosed with acute pancreatitis, to examine the relationships between these parameters and gender, and to investigate the possible role of the Systemic Inflammatory Response Index (SIRI), calculated using neutrophil, monocyte, and lymphocyte values, in the inflammatory process.

Method: This retrospective study examined patients who presented to the emergency department and were diagnosed with acute pancreatitis. Demographic characteristics and laboratory parameters of the patients were evaluated, and comparisons were made between genders. Additionally, SIRI was calculated using the values of neutrophils, monocytes, and lymphocytes.

Findings: No significant differences were found between genders in most haematological and biochemical parameters. However, haemoglobin levels were found to be significantly lower in female patients compared to male patients. Conclusion: Haematological parameters obtained from routine laboratory tests in patients with acute pancreatitis can contribute to the evaluation of the inflammatory process. While SIRI is considered a potential marker of inflammation, further studies in larger patient groups are needed to more clearly establish its relationship with disease severity and prognosis.

Keywords: Acute pancreatitis, Systemic inflammatory response index (SIRI), Haematological parameters, gender

INTRODUCTION

Acute pancreatitis is a disease characterised by the sudden inflammation of the pancreas. Its clinical course can range from mild inflammation to severe systemic complications (1). The most common causes are gallstones and alcohol use. In addition, hypertriglyceridemia, drugs,

and infections can also play a role in the aetiology (2). The severity of inflammation in acute pancreatitis is an important factor determining the prognosis of the disease. Therefore, early assessment of the disease is important for clinical management (3). Currently, various scoring systems are used to determine the severity of the disease. However, the application of these systems is not always easy (4).
In recent years, complete blood count parameters have attracted attention as important biomarkers in the evaluation of inflammation. In particular, haematological parameters such as neutrophil, platelet, and haemoglobin have been shown to reflect the inflammatory response and to be associated with the clinical course in many diseases, especially acute pancreatitis (5).

Objective: The aim of this study is to evaluate the demographic characteristics and laboratory parameters in patients diagnosed with acute pancreatitis and to examine the relationships between these parameters and gender. Additionally, the study aimed to determine the Systemic Inflammatory Response Index (SIRI) values, which are used with neutrophil, monocyte, and lymphocyte values obtained from complete blood counts, and to ascertain whether it is a biomarker related to the inflammatory process.

Materials and Methods

This study retrospectively examined patients diagnosed with acute pancreatitis who presented to our acute emergency department within the last month. Patients under 18 years of age and those whose data were incomplete were excluded from the study.

Statistical Analysis

Statistical analysis of the data obtained in this study was performed using IBM SPSS 27 Statistics (Version 27; IBM Corp., Armonk, NY, USA) program. The distribution of continuous variables was evaluated using the Shapiro-Wilk test. Qualitative data were expressed as frequency and percentage. For quantitative data, parametric distributions were presented as mean and standard deviation, while parameters with nonparametric distributions were presented as IQR or minimum-maximum values. The t-test was applied to data with parametric distributions, and the Mann-Whitney U test was applied to data with nonparametric distributions. $P<0.05$ was considered statistically significant.

Findings

A total of 18 patients with acute pancreatitis were included in the study. The mean age of the patients was 58.28 ± 24.58 years, with an age range of (26-85) years. When laboratory parameters were examined, the mean CRP level was found to be 23.53 ± 28.05 mg/L (median: 13.9, min–max: 1.2–121.1). The mean albumin level was 37.00 ± 5.63 g/L (median: 38.2, min–max: 27.6–43.8).

When complete blood count parameters were evaluated, the mean neutrophil count was 9.03 ± 5.66, the lymphocyte count was 1.35 ± 0.84, and the monocyte count was 0.76 ± 0.38. The mean platelet count was 268.50 ± 83.28 $\times 10^3/\mu L$. The patients' hemoglobin level was determined to be 12.71 ± 2.37 g/dL on average.
When pancreatic enzymes were examined, the average amylase level was found to be 1316.56 ± 1140.51 U/L (median: 1098.5, min–max: 63–3300). The average lipase level was determined to be 482.67 ± 205.49 U/L (median: 600, min–max: 59–600).
The patients' average hospital stay was 8.00 ± 2.99 days, and the length of stay was min-max(2 – 12) days.
The Systemic Inflammatory Response Index (SIRI), calculated from neutrophil, monocyte and lymphocyte values, was found to be 8.82 ± 11.33 on average. The median SIRI value is 4.09, and the IQR is (0.87 - 41.23).

Table 1. Distribution of age and blood parameters.

Variable	Mean ± SD	Median	Min–Max
Age (years)	58.28 ± 24.58	65.5	6 – 85
CRP	23.53 ± 28.05	13.9	1.2 – 121.1
Albumin	37.00 ± 5.63	38.2	27.6 – 43.8
Neutrophil	9.03 ± 5.66	8.82	2.23 – 19.78
Amylase	1316.56 ± 1140.51	1098.5	63 – 3300
Lipase	482.67 ± 205.49	600	59 – 600
Lymphocyte	1.35 ± 0.84	1.30	0.33 – 3.38
Platelet	268.50 ± 83.28	267	148 – 444
Hemoglobin	12.71 ± 2.37	13.15	5.9 – 15.2
Monocyte	0.76 ± 0.38	0.65	0.27 – 1.54
Hospital stay (days)	8.00 ± 2.99	8.5	2 – 12

A significant relationship was found between female and male genders, with lower hemoglobin levels in women (p=0.017). No significant relationship was found between female and male genders regarding CRP, albumin, neutrophil, amylase, lipase, lymphocyte, platelet, and monocyte levels (p=0.778, p=0.408, p=0.925, p=0.443, p=0.863, p=0.881, p=0.060, p=0.633). (Table 2)

Table 2: Relationship between gender and blood parameters.

Parameter	Female(mean)	Male (mean)	p value
CRP	21.62	26.53	0.778
Albumin	36.09	38.43	0.408
Neutrophil	9.12	8.89	0.925
Amylase	1472.09	1072.14	0.443
Lipase	489.82	471.43	0.863
Lymphocyte	1.33	1.39	0.881
Platelet	296.45	224.57	0.060
Hemoglobin	**11.79**	**14.14**	**0.017**
Monocyte	0.79	0.71	0.633

DISCUSSION

Acute pancreatitis is characterised by the sudden inflammation of the pancreas, and its clinical course can range from mild and self-limiting conditions to severe systemic complications (6). Early assessment of inflammation and prediction of prognosis are important for clinical management. Therefore, evaluation of routine laboratory parameters can help understand the course of the disease (7).

In our study, the mean age of the patients was found to be 58.28 ± 24.58 years. It has been reported in the literature that acute pancreatitis is more common, especially in adults and the elderly age group (8).

Gallstone formation is more frequent due to changes in cholesterol metabolism and decreased gallbladder motility as age increases. We believe that gallstones obstruct the pancreatic duct, leading to activation of pancreatic enzymes within the pancreas and inflammation.

In our study, 50% of the patients were female. The gender distribution in acute pancreatitis may vary depending on the aetiology. Literature reports that biliary pancreatitis is more common in women, while alcohol-related pancreatitis is more common in men (8). We believe the main reason for this is that the risk factors leading to pancreatitis are distributed differently between genders. In our study, a comparison between gender groups revealed that haemoglobin levels were significantly lower in female patients than in male patients (p=0.017). This finding is consistent with the literature. It is known that haemoglobin levels are lower in women than in men in the general population (9). We believe that the main reasons for this include chronic blood loss associated with menstruation, the erythropoiesis-enhancing effect of androgen hormones in men, and lower iron stores in women.

CONCLUSION

In conclusion, this study evaluated demographic characteristics and laboratory parameters in patients diagnosed with acute pancreatitis. In our study, no significant difference was found

between genders in terms of most hematological and biochemical parameters. Only haemoglobin levels showed a significant difference. Haemoglobin levels were found to be lower in female patients compared to male patients. This is thought to be related to physiological gender differences. In addition, SIRI values calculated using neutrophil, monocyte, and lymphocyte values obtained from complete blood count appear to be a potential biomarker in evaluating the inflammatory process. However, due to the limited number of patients, further studies in larger patient groups are needed to more clearly reveal the relationship of these parameters with the clinical course and prognosis of acute pancreatitis.

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A Rare Case: Postmenopausal Ovarian Torsion

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Introduction:

Ovarian torsion is a process that occurs when the ovary twists over the ligaments that support it in the adnexa (1). Most ovarian torsion occurs in the reproductive age group, and it is less common in premenarchal girls and postmenopausal women (2). Additionally, adnexal torsion develops in approximately 2–15% of women with a history of prior surgical intervention for adnexal masses or cysts (3).

This report presents a rare case of adnexal torsion in a perimenopausal woman, emphasizing that torsion can occur at any age and that the presence of an ovarian cyst constitutes a significant predisposing factor regardless of age.

Case:

A 48-year-old female patient presented to our emergency department with an acute onset of pain in the lower abdominal quadrant of one-day duration, which was gradually progressive. She was diagnosed with an ovarian cyst three years ago. She was gravida 6, para 4, abortus 2. Her menstrual cycles were regular. Initial vitals of the patient revealed as follows: temperature: 36.4°C, pulse: 66 bpm, oxygen saturation 99% on room air, blood pressure: 110/70 mmHg. The patient's overall condition is alert, oriented, and cooperative, with a Glasgow Coma Scale (GCS) score of 15. Physical examination findings in the abdomen are tenderness in the lower left quadrant without rebound tenderness or guarding. The patient's inital laboratory results were as follows: eGFR: 90, AST: 15 U/L, ALT: 20 U/L, GGT: 12 U/L, LDH: 171 U/L, total bilirubin 0.62 mg/dL, direct bilirubin: 0.18 mg/dl, amylase: 55 U/L, lipase: 21 U/L, CRP 3.4 mg/L, hemoglobin (HGB): 13.8 g/dL, red blood cells (RBC): 5.09 million/uL, white blood cells (WBC): 10,11 10*3/uL, platelets (PLT): 284 10*3/uL.

Abdominal CT showed a 70×62 mm cyst originating from the left adnexa and located anterior to the uterus. Left ovarian stromal edema was present, with a mild increase in size and anterior displacement. The CT scan results were insufficient to explain the patient's pain. Therefore, symptomatic treatment was administered and the patient was placed under observation. Since the patient's pain did not improve with symptomatic treatment, a pelvic Doppler ultrasound was scheduled.

Pelvic Doppler ultrasonography showed venous blood flow could not be detected in the left ovary, and its echogenicity was decreased. On abdominal MRI, stromal edema and decreased contrast enhancement were present in the left ovary, and the cyst was displaced anteriorly toward the midline. These findings were evaluated as ovarian torsion. The patient was referred to the gynecology and obstetrics clinic. Elective laparoscopic hysterectomy and salpingo-oophorectomy procedures were performed. As the patient had no bleeding or active symptoms, she was discharged in good condition.

Discussion and Conclusion:

Adnexial and ovarian torsions are rare gynecological emergencies that can happen at any age. Although they are more common in reproductive age than perimenopausal age, they should be considered in the differential diagnosis of acute lower abdominal pain in women, particularly in the presence of predisposing factors. This case report emphasizes that ovarian torsion can happen at all ages, and the existence of an ovarian cyst is and predisposing factor at all ages.

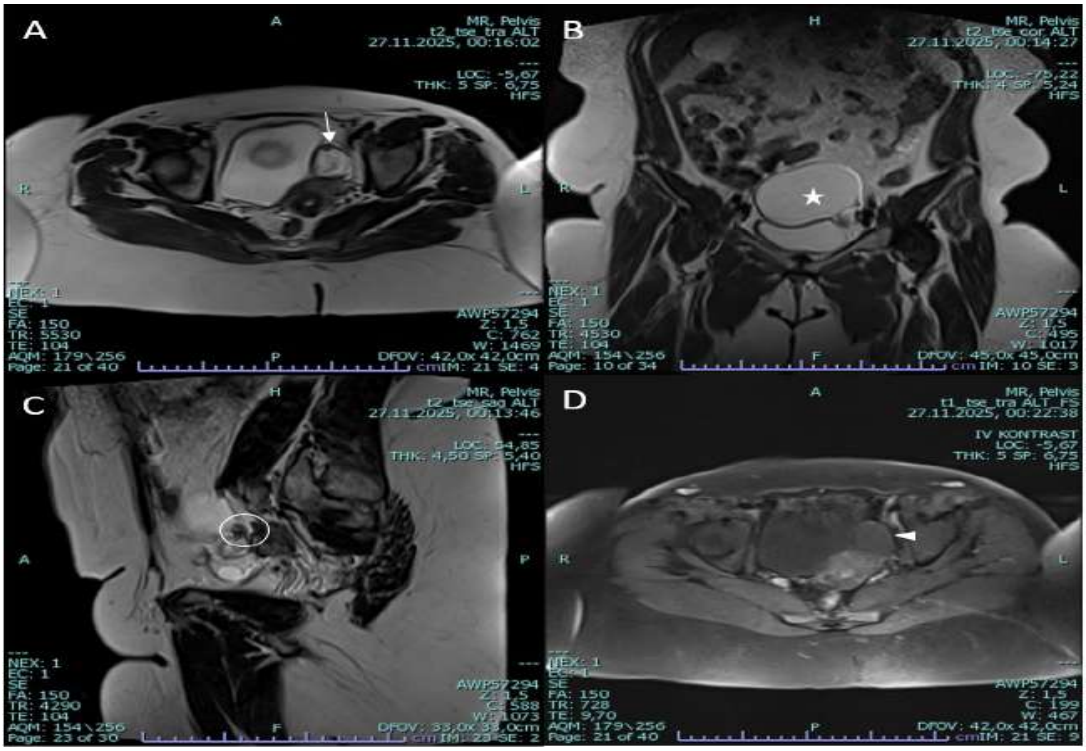

Figure 1: A. In T2-weighted axial MRI images, increased signal intensity consistent with stromal edema and an increase in size (arrow) are observed in the left ovary. B. In T2-weighted coronal MRI images, a 70 × 62 mm cyst (star) originating from the left ovary is observed. C. In sagittal T2-weighted images, a whirlpool sign (circle) is seen in the left fallopian tube. D. In axial contrast-enhanced T1-weighted images, reduced contrast enhancement of the left ovary (arrowhead) is noted.

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Spontaneous Pneumothorax Due To Hydatid Cyst's Pleural Enucleation: A Case Report

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Abstract

In this case report, we reported; 34 years old male person who experienced, spontaneous pneumothorax due to hydatid cyst's pleural enucleation. A 34-year-old man presented to our emergency department with one week history of dyspnea. Has no medical history. Physical examination of the patient who had no complaints other than dyspnea revealed the following: auscultation revealed that breath sounds in the right lung were less frequent than in the left. Other system examinations were normal. A plain chest X-ray revealed a pneumothorax on the right side. And consulted to thoracic surgery. Tube thoracostomy was performed in the emergency department with local anesthesia. Thoracic

computed tomography scan of the patient revealed hydatid cysts in both the lung and liver. The patient, diagnosed with spontaneous pneumothorax developing from hydatid cysts, underwent surgery. During the operation, it was determined that the lesion had spontaneously enucleated into the pleural space, causing pneumothorax. The patient was discharged on the 10th day after the operation. In secondary spontaneous pneumothorax, which is more common than primary spontaneous pneumothorax, identifying the underlying cause plays a crucial role in the early diagnosis and treatment of the patient. It should be kept in mind that pneumothorax due to hydatid cyst can cause secondary spontaneous pneumothorax.

Keywords: Emergency, Hydatid Cyst, Pneumothorax.

Introducion

Dyspnea, is one of the most frequent emergency department visits (1). Spontaneous pneumothorax (SP) is one of the critical diagnosis in this patient group (2). In this case, we discussed a patient who presented with dyspnea and was diagnosed SP in the emergency medicine. We emphasized the importance of differentiating between primary and secondary SP and the multidisciplinary approach. The rare condition of hydatid cyst enucleation causing SP and its management are presentation in this case report.

Case Presentation

A 34-year-old man presented to our emergency department with one week history of dyspnea. His vitals were: 125/85 mmHg blood pressure, 77/min hearth rate, temperature at 36.6°C, respiratory rate at 22/min, and oxygen saturation in room air at 93%. Has no medical history. Only smokes a pack of cigarettes per day. Physical examination of the patient who

had no complaints other than dyspnea revealed the following: auscultation revealed that breath sounds in the right lung were less frequent than in the left. Other system examinations were normal. ECG was normal sinus rhytm. A plain chest X-ray revealed a pneumothorax on the right side. And consulted to thoracic surgery (Figure 1). Tube thoracostomy was performed in the emergency department with local anesthesia. Following successful tube thoracostomy, chest X-ray revealed a suspicious area in the lower zone of the right lung, suggesting a possible cystic lesion (Figure 2). A thoracic computed tomography (CT) scan was then performed (Figure 3). Thoracic CT scan of the patient revealed hydatid cysts in both the lung and liver. The patient, diagnosed with SP developing from hydatid cysts, underwent surgery. Thoracic magnetic resonance imaging (MRI) was performed as a pre-operative complementary examination (Figure 4). During the operation, it was determined that the lesion had spontaneously enucleated into the pleural space, causing pneumothorax (Figure 5). The patient was discharged on the 10th day after the operation (Figure 6).

Discussion

Whether SPs diagnosed in the emergency department are primary SPs or secondary SPs affects the course of the disease (3,4). Generally, SP cases without a known underlying lung disease are considered primary SP. However, secondary SP occurs in the presence of an underlying lung disease and is a serious condition requiring urgent medical attention. Diseases involved in the etiology of secondary SP are often chronic, and the patients' history helps in differentiating between secondary SP and primary SP. However, patients with hydatid cysts are usually diagnosed late (5). This situation may cause the clinician to mistakenly diagnose

primary SP. Misdiagnosed secondary SP cases can progress to serious clinical conditions, such as septic shock or tension pneumothorax, and threats patients life (6). Hamdah et al. presented a case of secondary SP that developed on the basis of previously misdiagnosed hydatid cyst (7).

Shameem et al. in their case of a 22-year-old male patient who presented to the emergency department with respiratory distress and signs of shock. Initially the patient wasd diagnosed with primary SP. They performed tube thoracostomy. But upon seeing a cavitary lesion with air-fluid level on control chest X-ray, they detected a ruptured hydatid cyst on thoracic CT and were only able to operate on the patient on the 21st day (8).

In the etiology of secondary SP, hydatid cyst should be kept in mind in endemic regions even if hydatid cyst were not among the first, (9,10). In our case, the patient was diagnosed with secondary SP upon presentation to the emergency department, and the operation was performed in a shorter time. In addition, in our case, a very rare event occurred: spontaneous enucleation of the hydatid cyst into the pleural space, causing pneumothorax.

Conclusion

In secondary SP, which is more common than primary SP, identifying the underlying cause plays a crucial role in the early diagnosis and treatment of the patient. It should be kept in mind that pneumothorax due to hydatid cyst can cause secondary SP.

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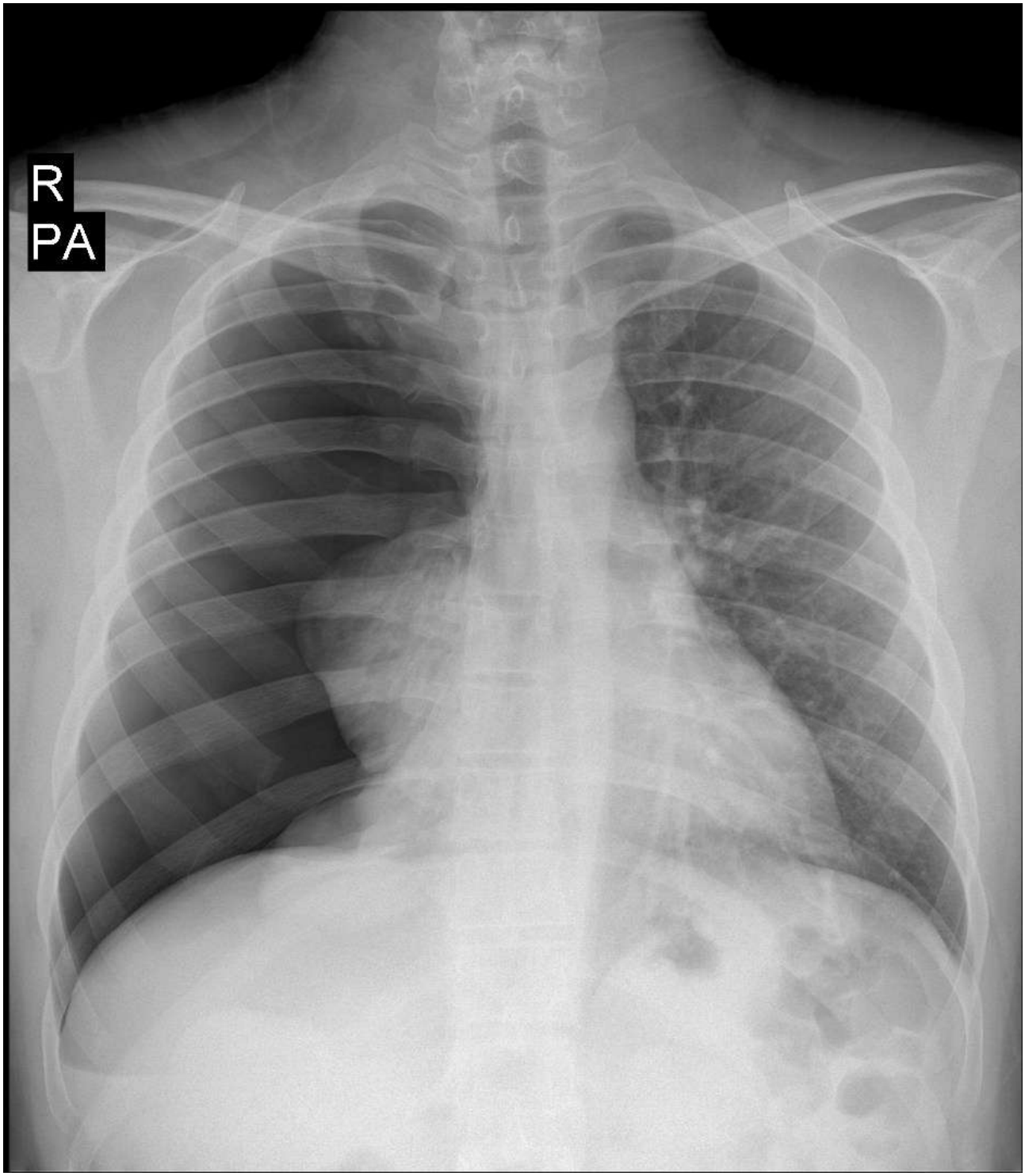


Figure 1. Chest X-ray at the time of admission to the emergency department.

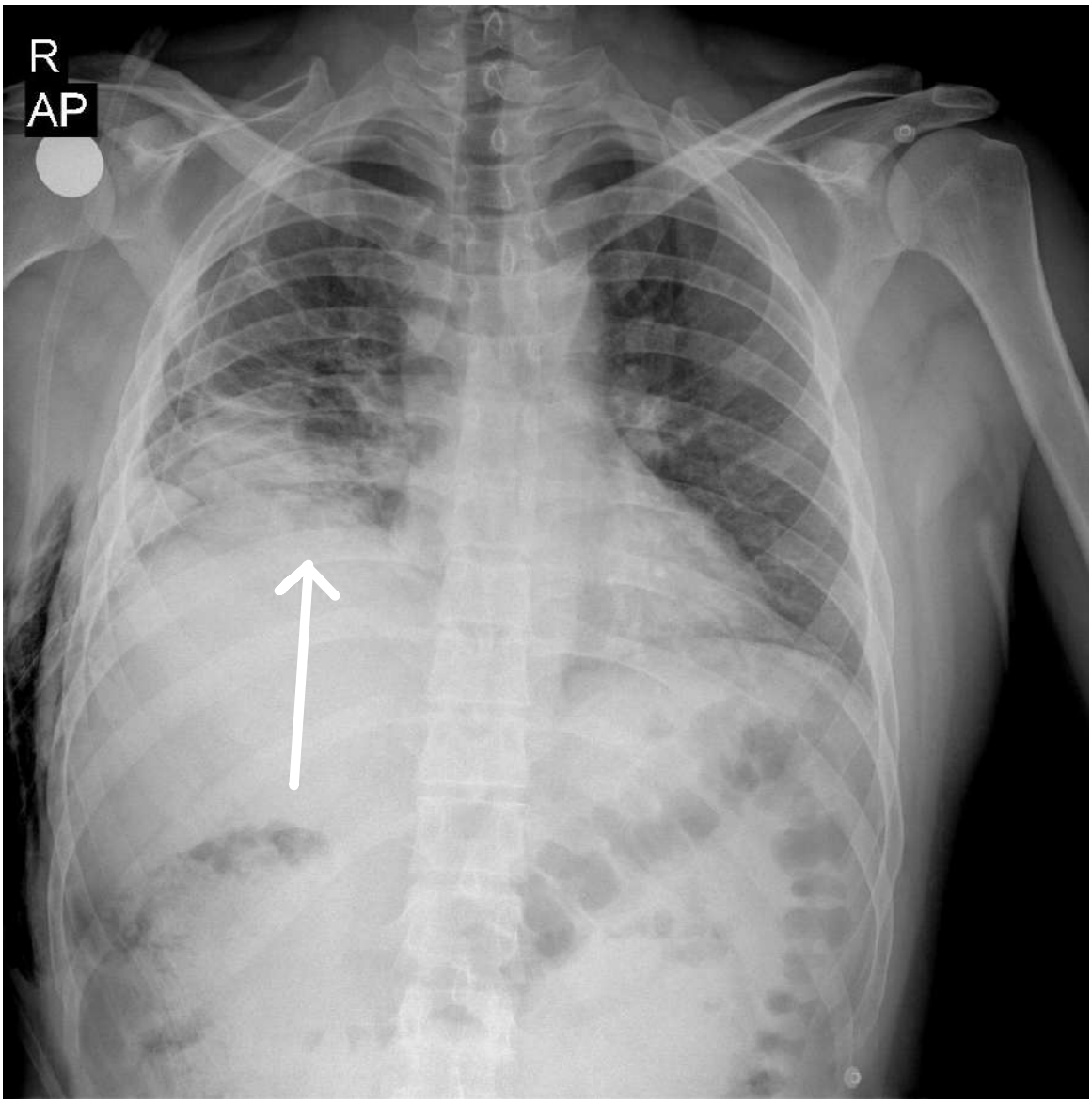
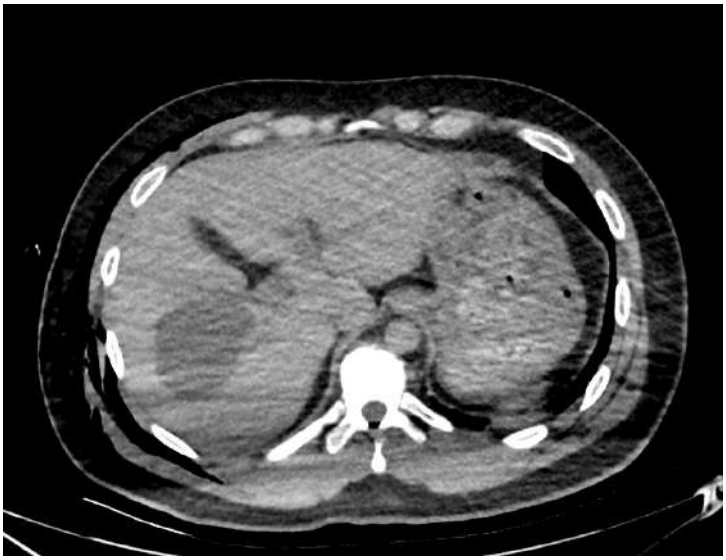


Figure 2. Chest X-ray taken after tube thoracostomy.



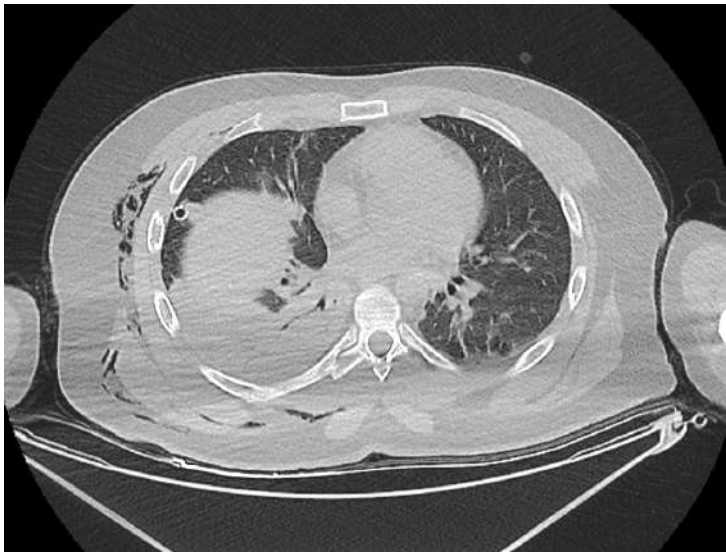


Figure 3. Non-contrast thoracic computed tomography scan.

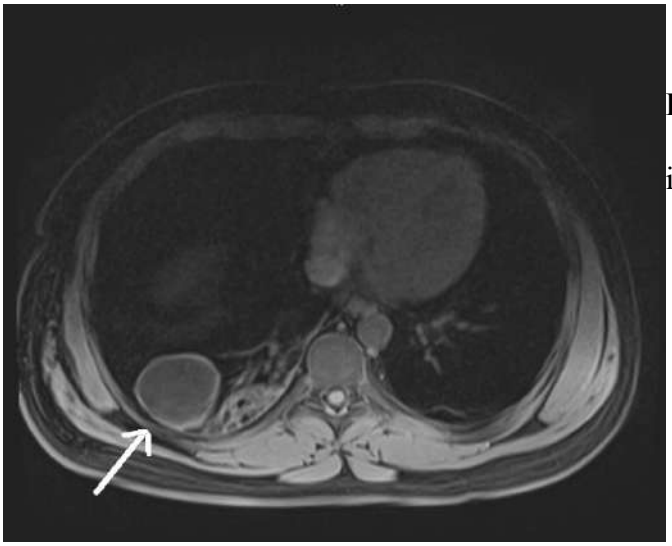


Figure 4. Thoracic magnetic resonance imaging.

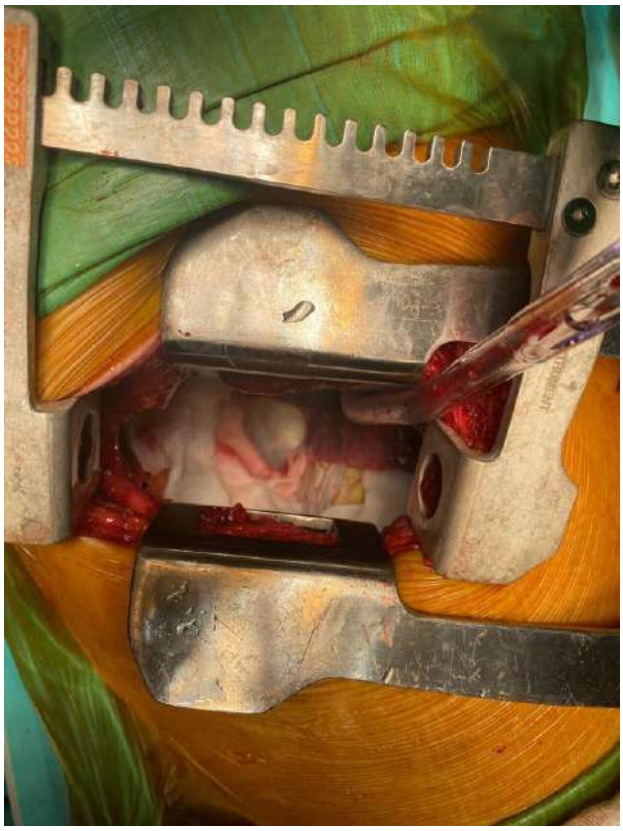


Figure 5. Hydatid cyst lesion causing pneumothorax with enucleation into the pleural space.

Figure 6. Postoperative chest X-ray.

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Optimal In-Hospital Location of Hyperbaric Oxygen Units Considering Disaster and Emergency Indications

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Introduction and Objective: Hyperbaric oxygen therapy (HBOT) is a time-critical treatment modality for several emergency conditions, including carbon monoxide poisoning, air embolism, decompression sickness, crush injuries, necrotizing infections, and acute traumatic ischemia. These patients are typically first evaluated and stabilized in emergency departments (EDs), where rapid access to definitive therapy is essential. In Türkiye, 57 HBOT centers operate across 28 provinces; however, the intra-hospital placement of these units has not been systematically planned according to emergency workflow and disaster response principles. This study aims to evaluate the current in-hospital location of HBOT units and discuss optimal placement strategies considering disaster and emergency indications.

Materials and Methods: A national review was conducted based on publicly available institutional data regarding the number, provincial distribution, and hospital placement characteristics of HBOT units in Türkiye. Particular attention was given to physical proximity to emergency departments and potential implications for time-to-treatment in critical conditions.

Results and Conclusion: Although 57 HBOT centers operate nationwide, available information suggests that only one unit is physically integrated within or directly adjacent to an emergency department. The majority of units are located in separate hospital blocks or independent facilities, requiring interdepartmental transfer after initial stabilization. In time-critical conditions and disaster scenarios, such separation may contribute to treatment delays and increase logistical complexity, particularly for intubated or hemodynamically unstable patients. Considering the emergency-driven nature of many HBOT indications, hospital infrastructure planning should prioritize proximity to emergency departments to facilitate rapid therapy initiation, improve patient safety during transfer, and enhance multidisciplinary coordination.

Keywords: hyperbaric oxygen therapy, emergency department, hospital planning, disaster medicine, time-critical care

Introduction

Hyperbaric oxygen therapy (HBOT) is a time-sensitive treatment modality for several emergency conditions, including carbon monoxide poisoning, arterial air embolism, decompression sickness, crush injuries, necrotizing soft tissue infections, and acute traumatic ischemia (1–3). In most clinical scenarios, these patients are first evaluated and stabilized in emergency departments (EDs). Early initiation of HBOT has been associated with improved neurological outcomes in carbon monoxide poisoning and enhanced limb salvage in crush-related ischemia (1,2,4).

Despite the emergency-driven nature of many HBOT indications, intra-hospital placement of hyperbaric units has rarely been evaluated from a disaster preparedness and emergency workflow perspective. Hospital planning traditionally emphasizes chamber safety requirements, oxygen systems, and structural considerations, while proximity to emergency care pathways receives limited attention.

Materials and Methods: A national descriptive review was conducted to evaluate the number, provincial distribution, and intra-hospital placement characteristics of hyperbaric oxygen therapy (HBOT) units in Türkiye. Data were obtained from publicly accessible institutional sources, including the official list of licensed HBOT centers published by the Republic of Türkiye Ministry of Health, institutional websites of public and private hospitals hosting HBOT units, and publicly available information from national hyperbaric medicine organizations where applicable. Hospital campus maps and satellite imagery (Google Maps) were used to assess the physical proximity of HBOT units to emergency departments. When publicly available information was insufficient to determine precise intra-hospital location, telephone confirmation was performed with hospital administrative staff or HBOT unit personnel to verify whether the unit was physically integrated within, directly adjacent to, or located separately from the emergency department. The study utilized exclusively publicly available institutional data and did not involve patient-level information; therefore, ethical approval was not required.

Results:

National HBOT Capacity in Türkiye

Currently, 57 HBOT centers operate across 28 provinces in Türkiye (Figure 1), reflecting substantial national capacity. However, available institutional information suggests that only one HBOT unit is physically integrated within or directly adjacent to an emergency department. The majority of units are located in separate hospital blocks or independent facilities within hospital campuses.

Figure 1. Geographic distribution of the 57 hyperbaric oxygen therapy (HBOT) centers across 28 cities in Türkiye included in the study. Additional HBOT facilities in Batman, Samsun,Aydın, Ordu, and Zonguldak are currently in the planning phase and are therefore not included in the present analysis.

While geographic distribution is important at the national level, intra-hospital positioning may significantly influence time-to-treatment once patients arrive at the hospital. Physical separation between EDs and HBOT units necessitates interdepartmental transfer following initial stabilization.

Emergency and Disaster Workflow Considerations

A substantial proportion of HBOT indications originate in the ED. Conditions such as carbon monoxide poisoning, arterial air embolism, decompression illness, crush-related compartment syndrome, and necrotizing infections require rapid decision-making and timely therapy initiation (1–3).

Although intra-hospital transport times may appear minimal under routine circumstances, even short logistical delays can become clinically relevant in time-critical settings. Transfer of intubated or hemodynamically unstable patients requires portable ventilators, infusion pumps, monitoring systems, and trained personnel, increasing complexity and risk exposure.

Disaster scenarios further amplify these challenges. Experiences from recent large-scale earthquakes in Türkiye have demonstrated that hospital functionality may be partially compromised due to elevator shutdowns, structural safety precautions, restricted inter-block access, surge patient volume, and temporary staff shortages. During the 2023 Kahramanmaraş earthquakes, reports highlighted disruptions in intra-hospital transportation systems and

logistical bottlenecks affecting critical care pathways (5). In such circumstances, the physical separation of HBOT units from emergency departments may further prolong transfer time, particularly for intubated, mechanically ventilated, or hemodynamically unstable patients requiring continuous monitoring. Therefore, intra-hospital adjacency between EDs and HBOT units should be considered not only a workflow optimization strategy but also a disaster resilience measure.

Discussion

Hospital infrastructure planning for HBOT has traditionally prioritized technical and safety requirements, while integration into emergency clinical workflow has received limited emphasis. This may represent a structural gap in disaster-readiness planning.

From a systems-based perspective, intra-hospital location should align with the origin of clinical demand. Since many HBOT indications are emergency-driven, ED-adjacent placement may facilitate:

- Reduced time-to-treatment initiation
- Improved safety during patient transfer
- Enhanced multidisciplinary coordination
- More efficient disaster response logistics

While Türkiye possesses strong national HBOT capacity, optimization of intra-hospital positioning represents an underrecognized opportunity for healthcare system improvement. The current model where most units are physically separated from emergency departments may not fully reflect the acute and time-sensitive nature of many HBOT indications.

Importantly, future hospital planning should incorporate emergency workflow considerations into architectural design. Newly established HBOT centers should, whenever feasible, be located in close proximity to or directly integrated with emergency departments. Incorporating ED adjacency as a structural planning principle may enhance rapid therapy initiation, reduce interdepartmental transfer risks, and strengthen disaster-response capability.

In addition to structural considerations, awareness and institutional integration of hyperbaric oxygen therapy within hospital systems remain variable. Previous research conducted in Türkiye has demonstrated gaps in knowledge and attitudes toward HBOT among family physicians, suggesting that organizational awareness and interdisciplinary coordination may not be fully optimized (6). These findings further support the need to align physical infrastructure planning with clinical workflow and emergency care pathways to improve system-level responsiveness.

Conclusion

Türkiye hosts 57 HBOT centers across 28 provinces, demonstrating considerable national treatment capacity. However, most units are not located adjacent to emergency departments, despite the emergency-driven characteristics of many HBOT indications. Considering both routine emergency care and large-scale disaster scenarios, intra-hospital proximity to emergency departments should be recognized as a strategic factor in future infrastructure planning. Aligning HBOT unit placement with emergency care pathways may enhance rapid intervention capacity, improve patient safety, and reinforce disaster preparedness.

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Clinical and Laboratory Findings of Carbon Monoxide Poisoning in Pediatric Patients: Evaluation of a Single-Center Retrospective Study

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Abstract

Objective :

Carbon monoxide (CO) poisoning is a common and potentially life-threatening condition in the pediatric age group. This study aimed to evaluate the demographic, clinical, and laboratory characteristics of pediatric patients diagnosed with CO poisoning and to examine the relationships between these parameters.

Materials and Methods:

This retrospective single-center study included patients under 18 years of age who were diagnosed with CO2 poisoning between December 1, 2025, and March 1, 2026. Of the 23 patients evaluated, 19 patients meeting the inclusion criteria were analyzed. Demographic data, laboratory parameters, and clinical outcomes were recorded. Continuous variables were presented as mean ± standard deviation or median (IQR). Intergroup comparisons were performed using Student t-test and Mann–Whitney U test. Correlation analyses were evaluated using Spearman correlation test.

Findings:

Of the 19 patients included in the study, 68.4% were male. The mean age was determined as 11±5.23 years. All patients were discharged alive. The median COHb level was 16.3% (1.9–20.4), and the median length of hospital stay was 1.3 (1–2.7) days. There was no need for

intensive care. A moderate positive correlation was found between COHb level and length of hospital stay (r=0.533;

p=0.019). A moderate negative correlation was found between albumin and COHb (r=-0.514;

p=0.024). In addition, a moderate negative correlation was found between age and lymphocyte count (r=-0.475; p=0.040).

Conclusion:

Although pediatric carbon monoxide poisoning generally has a good prognosis, some laboratory parameters can provide additional information about the clinical course. It is thought that COHb levels alone may not be sufficient, and parameters such as albumin may contribute to the clinical evaluation.

Keywords:

Carbon monoxide poisoning, Pediatrics, Carboxyhemoglobin, Albumin, Emergency department

Introduction:

Carbon monoxide (CO) poisoning is a common and potentially fatal environmental toxic exposure in childhood. CO binds to hemoglobin with an affinity approximately 200–250 times higher than oxygen, leading to the formation of carboxyhemoglobin (COHb) and ultimately resulting in tissue hypoxia (1,2). Children are more susceptible to CO poisoning than adults due to their higher metabolic rates and greater sensitivity to hypoxia (3). In pediatric patients, symptoms are often non-specific and manifest as headache, nausea, vomiting, weakness, and altered consciousness (4). This can lead to delays in diagnosis. Pulse oximetry is unreliable in carbon monoxide poisoning, and oxygen saturation may appear normal. Therefore, clinical suspicion and measurement of COHb levels by co-oximetry are important in diagnosis (5). It is reported in the literature that COHb levels do not always parallel clinical severity and that prognosis cannot be predicted by this parameter alone (6). However, parameters such as lactate level, neurological findings and clinical course are used in the assessment of disease severity (2,6). While prognosis is generally good with early diagnosis and appropriate oxygen therapy in pediatric patients, delayed neurological sequelae may develop in some cases (7,8). In this study, we aimed to examine the demographic, clinical and laboratory characteristics of pediatric patients diagnosed with carbon monoxide poisoning between December 1, 2025 and March 1, 2026 and to evaluate the relationships between variables

Materials and Methods

Study design and patient selection

This study is a single-center, retrospective study that retrospectively examined patients under the age of 18 who were diagnosed with carbon monoxide poisoning in the emergency department between December 1, 2025, and March 1, 2026. A total of 23 patients were evaluated during this period, and 19 patients who met the inclusion/exclusion criteria were included in the study.

Data collection:

Patients' demographic data, length of hospital stay, and laboratory parameters were obtained from the hospital information management system. The parameters and units evaluated were:

. PLT ($\times 10^3/\mu L$)

. Albumin (g/L)

. CRP (mg/L)

. Neutrophil ($\times 10^3/\mu L$)

. Lymphocyte ($\times 10^3/\mu L$)

. Monocyte ($\times 10^3/\mu L$)

. Hemoglobin (g/dL)

. Lactate (mmol/L)

. COHb (%)

. pH

.pCO_2 (mmHg)

. pO_2 (mmHg)

.Saturation (%)

Statistical analysis:

Data showing a normal distribution were presented as mean ± standard deviation, while data not showing a normal distribution were presented as median (IQR, 25–75th percentile). The relationship between qualitative variables and parametric data was evaluated using the Student t-test, and the relationship between non-parametric data was evaluated using the Mann–Whitney U test.

Correlation analyses were performed using the Spearman correlation test. Correlation strength was interpreted according to the Mukaka classification: <0.30 negligible, 0.30–0.49 weak, 0.50–0.69 moderate, 0.70–

0.89 strong, ≥0.90 very strong (9).

The statistical significance level was accepted as $p<0.05$.

Result:

A total of 19 cases of carbon monoxide poisoning were included in the study. 68.4% of the cases were male (n=13) and 31.6% were female (n=6). The average age of the patients included in our study was determined to be 11. All patients were discharged alive. The mean and SD values of our parametric data were as follows: age: 11±5.23, plt: 282.47 ±79.68,

albumin: 44.13±2.89, pH: 7.39±0.031 (Table 1).

Table 1. Mean and SD values of our parametric data.

Variables	n	Mean	SD
Yaş (yıl)	19	11	5,23
Plt (10^3/uL)		282,47	79,68
Albülim(g/L)		44,13	2,89
Ph		7,39	0,031

The median and IQR values of our non-parametric data were: CRP: 0 (0-3.1), neutrophil: 4.34 (3.18-6.36),

lactate: 1.8 (1.2-4.9), lymphocyte: 3.3 (2.13-4.62), HGB: 12.9 (12.2-14.2), monocyte: 0.6 (0.5-0.81),

COHB: 16.3 (1.9-20.4), pCO2: 41.5 (38.6-44.4), PO2: 19.4 (7.9-48.6), saturation: 62.8 (43.1-88.5),

hospital stay: 1.3 (1-2.7), IB stay: 0 (0-0) (Table 2).

Table 2. Median and IQR values of our non-parametric data.

			IQR	
Variables	n	Median	25%	75%

Crp (mg/L)		0	0.Oca	3.Oca	
Neu (10^3/uL)		4,34	3,18	6,36	
Laktat		1,8	1,2	4,9	
Lym (10^3/uL)	19	3,3	2,13	4,62	
Hgb		12,9	12,2	14,2	
Monosit		0,6	0,5		0,81
Cohb		16,3	1,9	20,4	
Pco2		41,5	38,6	44,4	
Po2		19,4	7,9	48,6	
Sat		62,8	43,1	88,5	
Hospital stay		0	0	0	
Stay in intensive care		0	0	0	

We compared the relationship between our qualitative and parametric data using a T-test and found no significant result. We compared the relationship between our qualitative and non-parametric data using the Mann-Whitney U test. It did not show a statistically significant difference. We evaluated the correlation between our data using Spearman correlation analysis. We found a moderately significant negative correlation between age and lymphocytes (r=-0.475, p=0.04), a moderately significant positive correlation between CoHb and length of hospital stay (r=0.533, p=0.019). We found a moderately significant negative correlation between albumin and CoHb (r=-0.514, p=0.024), and a moderately significant positive correlation between saturation and CRP (r=0.535, p=0.018).

Dıscussıon:

In our study, the fact that all patients in pediatric carbon monoxide poisoning cases were discharged alive and did not require intensive care reflects a generally mild-to-moderate patient group. It is also reported in the literature that the prognosis is mostly good and mortality is low with early diagnosis and appropriate oxygen therapy in pediatric patients (7,8,10). This situation supports the decisive role of early intervention on clinical outcomes in the pediatric population.

In our study, the moderate positive correlation found between COHb and hospital stay duration suggests that the clinical follow-up period may be longer as exposure increases. However, the literature emphasizes that COHb levels do not always accurately reflect clinical severity, that COHb levels may be misleading, especially in delayed admissions, and that it should not be used as a reliable prognostic marker alone (6,11). Therefore, we believe that COHb should be interpreted together with clinical evaluation and other biochemical parameters. Another noteworthy finding is the moderate negative significance of the correlation between albumin and COHb. Considering that albumin is a negative acute phase reactant and can decrease in hypoxic conditions, this finding may be one of the systemic manifestations of tissue hypoxia and oxidative stress that develop in carbon monoxide poisoning. In the literature, it has been shown that inflammatory and oxidative mechanisms play a role in CO poisoning, and (2), our findings support this pathophysiological process. From this point of view, albumin may be a helpful biomarker that can reflect the disease burden in pediatric CO poisoning. In addition, the negative relationship found between age and lymphocyte count can be explained by the physiological hematological variability specific to the pediatric age group. On the other hand, the positive correlation between saturation and CRP is not a clinically expected relationship and may be due to factors such as small sample size, measurement timing, or accompanying subclinical inflammatory processes. Overall, our study shows that mortality is low and short-term clinical outcomes are good in pediatric carbon monoxide poisoning. However, the relationships between COHb and albumin levels, in particular, stand out as potential parameters that can provide additional information about disease severity and clinical course. More large sample sizes and prospective studies are needed to confirm these findings.

Conclusion:

Although the clinical course is generally good in pediatric carbon monoxide poisoning cases,

a positive correlation has been found between COHb levels and length of hospital stay, and a negative correlation has been found between albumin and COHb. These findings suggest that COHb alone may not be sufficient and that parameters such as albumin may contribute to clinical evaluation.

Limitations:

The main limitations of the study are its retrospective and single-center nature, limited sample size, and lack of long-term neurological follow-up data.

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7643

Heart Attack or Broken Heart?

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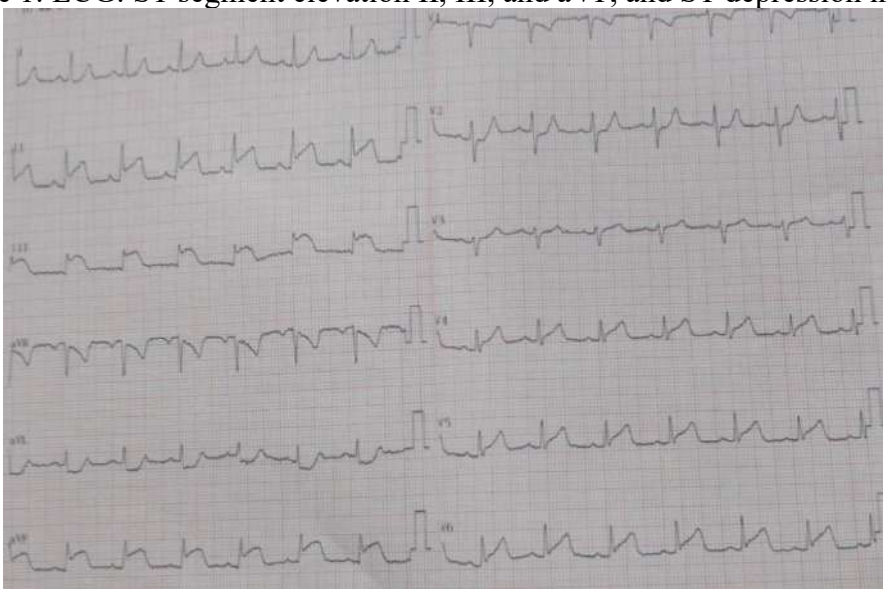
Introduction and Purpose: Introduction Takotsubo cardiomyopathy (TCM) is a transient left ventricular dysfunction triggered by emotional or physical stress. Patients present with chest pain. Acute coronary syndrome (ACS) is diagnosed based on electrocardiographic (ECG) ST segment changes and mild troponin elevation. This presentation describes a rare case of TCM in a patient who presented with typical chest pain and ST segment elevation in the postpartum period.

Materials and Methods: A 28-year-old female patient presented with severe, constricting chest pain radiating to her left arm and back, which had started one hour prior. She had a history of gestational hypertension and had undergone cesarean section (C/S) a few days prior. Vital signs: Blood pressure: 110/ 70 mmHg, Heart rate: 100 beats/minute, Respiratory rate: 18 breaths/minute, SpO2: 98 %. Cardiac and physical examination were normal. ECG showed ST segment elevation of more than 2 mm in derivations II, III, and aVF, and ST depression in aVL. Troponin T level was 13.9 ng/mL. The patient was referred to cardiology for emergency coronary angiography with a preliminary diagnosis of acute inferior myocardial infarction. Coronary angiography revealed normal coronary arteries with no obstructions.

Figure 1: ECG: ST segment elevation II, III, and aVF, and ST depression in aVL



Results and Conclusion: TCM is frequently seen in postmenopausal women; however, it can also occur in younger patients in situations involving intense post-operative stress factors, such as the postpartum period. In this case, the diagnosis of TCM was made due to the patient's postpartum condition and the absence of coronary artery occlusion on angiography.Although patients presenting to the emergency department with chest pain and ST elevation on ECG are primarily evaluated for acute myocardial infarction, TCM should be considered in patients under physical and emotional stress with normal angiographic results. With early supportive treatment and follow-up of complications, the prognosis for these patients is generally good.

Keywords: broken heart syndrome, pseudo-infarction, takotsubo cardiomyopathy

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7662

Performance of ChatGPT and Gemini Compared with Emergency Physicians in NSTEMI Cases: A Prospective Cross-sectional Study

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Background:
Acute coronary syndromes remain a major cause of morbidity and mortality worldwide, and non-ST elevation myocardial infarction (NSTEMI) accounts for a substantial proportion of these cases (1). Diagnosing NSTEMI in emergency departments is challenging because of high patient volume, time pressure, and variable clinical presentation. Artificial intelligence (AI) systems based on large language models (LLMs), such as ChatGPT and Gemini, have recently emerged as potential clinical decision support tools (2). These systems can rapidly process medical information and generate guideline-based responses. This study aimed to evaluate the performance of ChatGPT and Gemini in NSTEMI-related clinical scenarios by comparing their responses with those of emergency physicians.

Methods:
This prospective cross-sectional study was conducted using an online survey between February and April 2025. A total of 1,106 emergency physicians in Turkey participated voluntarily. The questionnaire included demographic questions and ten multiple-choice items related to NSTEMI diagnosis and management based on the 2023 European Society of Cardiology guidelines (1). The same questions were presented to ChatGPT-4.0 and Gemini-2.5 using identical standardized prompts with temperature set to zero and web access

disabled. Physicians' responses were analyzed using descriptive statistics and subgroup comparisons according to professional title, experience, and workplace.

Results:
The mean number of correct answers among physicians was 7.62 ± 1.32 out of 10. Both ChatGPT and Gemini answered nine questions correctly (90% accuracy). General practitioners scored 7.48 ± 1.3, residents 7.98 ± 1.1, and specialists 8.19 ± 1.3, showing improved performance with increasing experience. Physicians working in training and research hospitals achieved higher scores than those in state hospitals. Questions related to basic knowledge, such as diagnosis and biomarkers, had high correct response rates (>90%), whereas questions requiring more complex clinical reasoning showed lower accuracy among physicians (3,4).

Conclusion:
ChatGPT and Gemini demonstrated higher accuracy than emergency physicians in NSTEMI-related guideline-based questions. These findings suggest that AI may support medical education and clinical decision-making. However, performance in knowledge-based questions does not necessarily reflect real clinical diagnostic ability. Human clinical judgment remains essential, particularly in complex clinical situations.

Keywords:
Artificial intelligence; Emergency medicine; Clinical decision support; Large language models; Non-ST elevation myocardial infarction

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7695

More Than a Medical Visit: Homelessness as a Social Emergency in the Emergency Department

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Introduction

Homelessness is an increasingly critical public health and social service concern globally and within our nation, stemming from contemporary urbanization and socioeconomic disparities. Homeless individuals frequently utilize emergency services as a sanctuary or initial point of contact due to their inability to fulfill fundamental human necessities, including shelter, nutrition, and basic hygiene. This cohort of patients, identified in the literature as "frequent flyers/users," utilizes emergency services not for acute medical crises but for chronic pain, substance dependency, psychiatric disorders, or merely in pursuit of a hospitable atmosphere [3]. Research indicates that the frequency of emergency room utilization among the homeless significantly exceeds that of the general populace [4]. For these patients, social deprivation and insufficient psychological support typically outweigh any underlying biological disease [5]. Despite typical outcomes in their medical assessments, the absence of a secure living situation following discharge presents emergency response professionals with social and ethical challenges in addition to medical ones [1,2]. The execution of "case management" processes in hospitals and the proactive involvement of social service specialists (SSS) are essential for the societal integration of patients and the prevention of recurrent admissions [6]. This case presentation details the management of a 49-year-old male patient who frequently visited the emergency department for extensive body pain, despite normal clinical and laboratory findings. He remained in the emergency observation unit for several days due to housing complications.

Case

A forty-nine-year-old male patient, devoid of known comorbidities or regular medication, appeared to the emergency department with complaints of generalized body discomfort and weakness. The patient was assessed in the emergency department triage as being in stable general condition, alert, oriented, and cooperative (Glasgow Coma Scale Score: 15). The initial vital signs indicated a blood pressure of 120/75 mmHg, a pulse of 78 bpm, a body temperature of 36.6 °C, and an oxygen saturation of 98% in room air, all within normal norms. The physical examination revealed insufficient self-care upon evaluation of the patient. No acute pathological abnormalities, impairments, or symptoms of trauma were noted throughout the systemic examination, which included cardiovascular, respiratory, gastrointestinal, and neurological assessments. Upon further investigation of the patient's past, it was revealed that he had no family, had been homeless for an extended period, and lacked a stable income or social security. A retrospective analysis using the hospital's automation system revealed that the patient had recurrently presented to the emergency room with analogous complaints (diffuse pain, chills, weariness) over the last six months. No abnormal values were identified in the comprehensive blood parameters asked during the medical examination in the emergency room, which included a complete blood count, kidney and liver function tests, electrolytes, cardiac markers, and acute phase reactants. The concurrent radiological scan was deemed normal. The patient, lacking any medical justification for acute hospitalization, was scheduled for discharge from the emergency room. Consequently, because the patient was homeless and without a secure location for post-discharge, the emergency department physicians notified the hospital's Social Services Department. The patient was interrogated by social service specialists, who subsequently contacted the appropriate institutions. The arrangement of an appropriate refuge for the patient was extended due to insufficient availability of accommodations. Throughout this interim, given the potential risk of hypothermia or injury outdoors, the patient was observed in our emergency room for 3 to 4 days. The emergency department staff addressed fundamental necessities. Upon locating an appropriate shelter center, the patient was discharged with the assistance of social service officers and thereafter transferred to the designated facility.

Discussion

Homeless patients represent a particularly demanding and commonly encountered demographic in emergency services. Işıkhan's study on the social service requirements of the homeless indicates that, due to their inability to fulfill basic demands for feeding, heating, and secure housing, they perceive healthcare institutions as a refuge [1]. The patient's recurrent visits with vague complaints, including generalized body discomfort, correspond precisely with the notion of "social indication visits" as documented in the global literature [3,5]. In medical literature, persons who frequent the emergency room four or more times annually are typically termed "frequent users," with homelessness identified as a significant independent risk factor for this phenomenon [4]. The reality that our

Conclusion

People who are homeless and go to emergency rooms often go back more than once, not because they have an urgent medical problem, but because they need help with housing, food, and safety. No matter how normal the lab and clinical results are, it is morally and humanely wrong to leave people on the street. Social service methods (case management) should be used in addition to medical help, and it is very important to set up a quick and reliable network for emergency services and local/public aid groups to work together.

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7747

WHEN STROKE IS NOT STROKE: CENTRAL NERVOUS SYSTEM INVOLVEMENT OF LANGERHANS CELL HISTIOCYTOSIS IN AN ELDERLY PATIENT

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Introduction

Histiocytoses are rare disorders characterized by the accumulation of cells belonging to the mononuclear phagocyte system in various tissues and organs in both children and adults.

Langerhans Cell Histiocytosis (LCH) is defined by organ infiltration of histiocytes that share features with epidermal dendritic Langerhans cells but arise from the aberrant differentiation of myeloid dendritic cell precursors. Although bone involvement is the most common manifestation, the lungs, skin, liver, spleen, and lymph nodes may also be affected.

The clinical presentation of LCH is highly variable, including systemic symptoms such as fever and weight loss, as well as organ-specific findings like bone pain, mucocutaneous lesions, lymphadenopathy, or cytopenias. In this report, we present a patient admitted to the emergency department with weakness in the left extremities.

Case Presentation

An 84-year-old male patient was referred to our emergency department from an external center with a sudden onset of weakness in the left arm and left leg. His medical history was significant only for hypertension. On admission, his vital signs were stable.

Neurological examination revealed 3/5 muscle strength in both the left upper and lower extremities, with no additional neurological deficits. Initial cranial computed tomography (CT) demonstrated loss of gray-white matter differentiation in the left hemisphere. Additionally, a hypodense area consistent with a chronic subdural hematoma (CSDH) was observed in the same region.

To exclude acute ischemic stroke, diffusion-weighted magnetic resonance imaging (DWI-MRI) was performed and showed no evidence of diffusion restriction. Due to the discrepancy between clinical findings and initial imaging, contrast-enhanced brain MRI was obtained for further evaluation.

Contrast-enhanced MRI revealed enhancing lesions in the right hemisphere consistent with LCH, which were considered responsible for the patient's clinical presentation.

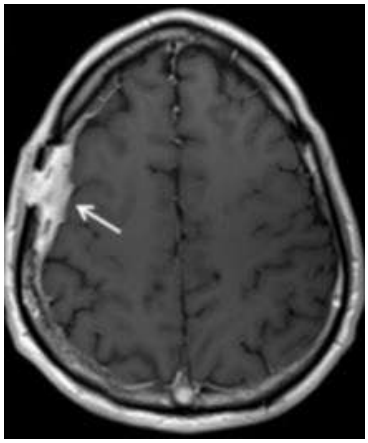

FIGURE: MRI

Discussion

LCH is a rare disease that predominantly occurs in childhood and is commonly characterized by bone involvement. Its incidence in adults is approximately 1–2 cases per million, and its occurrence in elderly patients is extremely rare. The diagnosis of LCH in an 84-year-old patient is therefore highly unusual and scarcely reported in the literature.

Central nervous system (CNS) involvement in adults is rare and generally presents in two forms:

- **Tumoral form:** characterized by focal neurological deficits due to mass effect
- **Neurodegenerative form:** presenting with ataxia, dysarthria, and progressive cognitive decline

In our case, the acute onset of motor deficit suggests the tumoral form.

Initially, the loss of gray-white matter differentiation and subdural appearance in the left hemisphere on non-contrast CT would be expected to produce right-sided neurological deficits. However, the patient presented with left hemiparesis. This discrepancy indicated that the CT findings were likely related to a chronic and asymptomatic process (possibly an old CSDH) and not responsible for the acute presentation.

The absence of diffusion restriction on DWI-MRI excluded acute ischemia. Subsequent contrast-enhanced MRI identified pathological enhancement in the right hemisphere, leading to the correct diagnosis.

Conclusion

In elderly patients presenting with acute neurological deficits, initial imaging with CT may be misleading. When diffusion-weighted MRI is normal and there is clinicoradiological mismatch, contrast-enhanced MRI is crucial for identifying uncommon diagnoses such as LCH and preventing misdiagnosis.

Keywords: Langerhans Cell Histiocytosis ,Central Nervous System ,Stroke

7765

Laboratory Evaluation of Meningiomas in the Emergency Department: A Retrospective Analysis

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Introduction

Meningiomas account for approximately 15–20% of intracranial tumors and are among the most common primary central nervous system tumors in adults, with a higher incidence observed particularly in women (1,2). This gender difference is thought to be associated, in part, with the expression of progesterone and estrogen receptors (2).

Although the etiology of meningiomas has not been fully elucidated, exposure to ionizing radiation is considered one of the strongest risk factors. In addition, genetic predisposition (particularly NF2 gene mutations), chromosomal abnormalities, hormonal influences, trauma, and certain viral agents are among the potential etiological factors (2,3).

Clinically, meningiomas are generally slow-growing and benign tumors; however, depending on their location, they can present with a wide spectrum of symptoms, including headache, seizures, focal neurological deficits, and cognitive changes (3). In patients presenting to the emergency department, these symptoms are often nonspecific, which complicates early diagnosis and prompts clinicians to rely on supportive parameters.

In recent years, the role of laboratory markers in assessing tumor biology and prognosis has been increasingly investigated. In particular, serum calcium levels have been reported to potentially influence tumor development through effects on cell proliferation and growth factors, while lactate dehydrogenase (LDH) levels may increase in relation to tumor cell metabolism and hypoxia (4). Furthermore, arterial blood gas pH values have been suggested as a potential indicator of metabolic changes in the tumor microenvironment and may be associated with tumor progression (5).

In this context, elucidating the relationship between demographic characteristics, presenting symptoms, and laboratory parameters in patients with meningiomas presenting to the

emergency department may contribute both to the diagnostic process and to understanding the biological behavior of the disease. This study aimed to evaluate demographic data, presenting symptoms, and serum calcium (Ca), lactate dehydrogenase (LDH), and arterial blood gas pH values in patients diagnosed with meningioma.

Materials and Methods

A retrospective analysis was conducted on 49 patients who presented to the emergency department between January 2020 and January 2024 and were diagnosed with intracranial meningioma.

Patient age, sex, presenting symptoms, serum Ca and LDH levels, and arterial blood gas pH values were recorded.

Statistical analysis was performed using SPSS version 26. The normality of the data distribution was assessed with the Shapiro-Wilk test. Comparisons between groups were conducted using the Mann-Whitney U test, and a p-value of <0.05 was considered statistically significant.

Results

Of the 49 patients, 34 (67.3%) were female and 16 (32.7%) were male.

The most common presenting symptom was headache (85.7%), followed by nausea and vomiting (6.1%), seizures (4.1%), and speech disturbances (4.1%).

The mean age was 56.8 ± 4.1 years in males and 52.7 ± 5 years in females.

No significant difference was observed between genders in serum Ca levels ($p > 0.05$).

Arterial blood gas pH values were within normal limits in both groups, with no significant difference ($p > 0.05$). Serum LDH levels were significantly higher in female patients compared to males ($p < 0.05$).

Discussion

In our study, meningiomas were found to occur more frequently in women, with headache being the most common presenting symptom. These findings are consistent with the literature, which reports that meningiomas are approximately twice as common in women and that

headache is the most prevalent symptom (1,2). This gender difference has been suggested to be related, in particular, to hormonal factors, especially progesterone receptor expression (2).

In our study, the mean ages of the patients were predominantly concentrated in the fifth and sixth decades of life. This finding aligns with epidemiological studies reporting that meningiomas are most frequently observed between 40 and 60 years of age (1,3). The similarity in age distribution supports the notion that the patient population in our study is representative of those reported in the literature.

No statistically significant difference was observed between female and male patients in terms of serum calcium levels. Although the literature reports that calcium ions may play a role in tumor development through effects on cell proliferation, signaling pathways, and growth factors (particularly EGF and IGF-1), it is noted that systemic calcium levels may not always reflect these effects (6). This can be explained by the local effects of calcium within the tumor microenvironment and the tight regulation of systemic levels through homeostatic mechanisms. In addition, treatments received by patients before or after diagnosis may also influence serum calcium levels.

In our study, serum LDH levels were found to be significantly higher in female patients. LDH is a key enzyme in anaerobic glycolysis and is particularly associated with increased metabolic activity and hypoxia in tumor cells (1,4,6). It is well established that LDH levels rise as a result of metabolic reprogramming in cancer cells, a phenomenon known as the Warburg effect (5). Although meningiomas are generally benign, elevated LDH levels may provide information regarding tumor cell metabolic activity and potential proliferative capacity. The literature also reports increased LDH levels in meningiomas and other brain tumors, with a decrease observed following treatment (7). In this context, the elevated LDH values observed in our study may be related to tumor metabolism and hypoxic processes.

The finding that arterial blood gas pH values were within normal limits in both sexes indicates that systemic acid-base balance is maintained. However, the literature suggests that a localized acidic environment within the tumor microenvironment can promote cell proliferation, angiogenesis, and tumor invasion (6,8). Therefore, normal systemic pH values

do not preclude the presence of local metabolic alterations within the tumor microenvironment.

In conclusion, our study indicates that the demographic characteristics and clinical findings of patients with meningioma are consistent with those reported in the literature. The observed elevation in serum LDH levels suggests that it may serve as a significant marker of tumor metabolism. In contrast, serum calcium and pH values appear to have limited discriminatory value. Larger, prospective studies are needed to more clearly define the role of these biomarkers in the diagnosis and prognosis of meningioma.

Conclusion

Meningiomas most commonly present to the emergency department with nonspecific symptoms, with headache being the predominant complaint. While serum LDH levels may provide additional information regarding tumor biology, the discriminatory value of calcium and pH measurements appears limited. The assessment of laboratory parameters alongside clinical findings in cases of suspected meningioma in the emergency setting may contribute to diagnosis and management. Larger studies with expanded sample sizes are needed to further clarify these findings.

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Colchicine-Induced Massive Rhabdomyolysis and Acute Kidney Injury Complicated by Aspiration Pneumonia: A Case Report

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Introduction

Colchicine is an agent that suppresses the inflammatory response by inhibiting microtubule polymerization; however, it carries a risk of serious systemic toxicity due to its narrow therapeutic index (1). Especially in elderly patients, impaired drug clearance can trigger massive destruction (rhabdomyolysis) in skeletal muscle and cells with rapid mitosis (2). Comorbidities such as Diabetes Mellitus (DM), Hypertension (HT), and Chronic Kidney Disease (CKD) multiply the toxic effect by making it difficult for the drug to be excreted from the body (3). Even in cases that appear hemodynamically stable, initial laboratory values can be misleading, and life-threatening multiple organ failure patterns can emerge in a short time (1).

Case

A 72-year-old female patient with known diagnoses of gout, DM, HT, and CKD presented to an external internal medicine outpatient clinic with pain in the first distal metatarsal joint of the left foot. Colchicine (2x0.5 mg) treatment was initiated for suspected gout attack; her baseline creatinine at the time of admission was measured at 1.2 mg/dL.

On the 3rd day of treatment, the patient presented to the clinic with severe nausea and vomiting, where creatinine was found to be 2.2 mg/dL and CK was 25,000 U/L. Although the patient was admitted to the internal medicine ward, her creatinine and CK values progressed despite hydration, and her general condition deteriorated. The patient developed total anuria and entered respiratory failure, requiring intubation after aspiring gastric contents during severe vomiting episodes in the early stage of colchicine toxicity.

At this stage, the patient was referred to our hospital. Upon arrival, the following values were observed: Creatinine: 4.8 mg/dL CK: 37,000 U/L LDH: 2234 U/L Phosphorus: 7.1 mg/dL pH: 7.2 Bicarbonate: 18 mmol/L Lactate: 4 mmol/L

Norepinephrine infusion was started for the hypotensive patient. A dialysis catheter was inserted for urgent hemodialysis needs, and she was admitted to the toxicology intensive care unit. Although a significant regression in creatinine, LDH, and CK values was achieved after dialysis, thorax computed tomography (CT) revealed widespread consolidation areas in the bilateral lower lobe bases and infiltrations consistent with aspiration pneumonia (Figure 1).

The patient, whose acute phase reactants showed a progressive increase, was followed up with preliminary diagnoses of septic shock and ARDS.

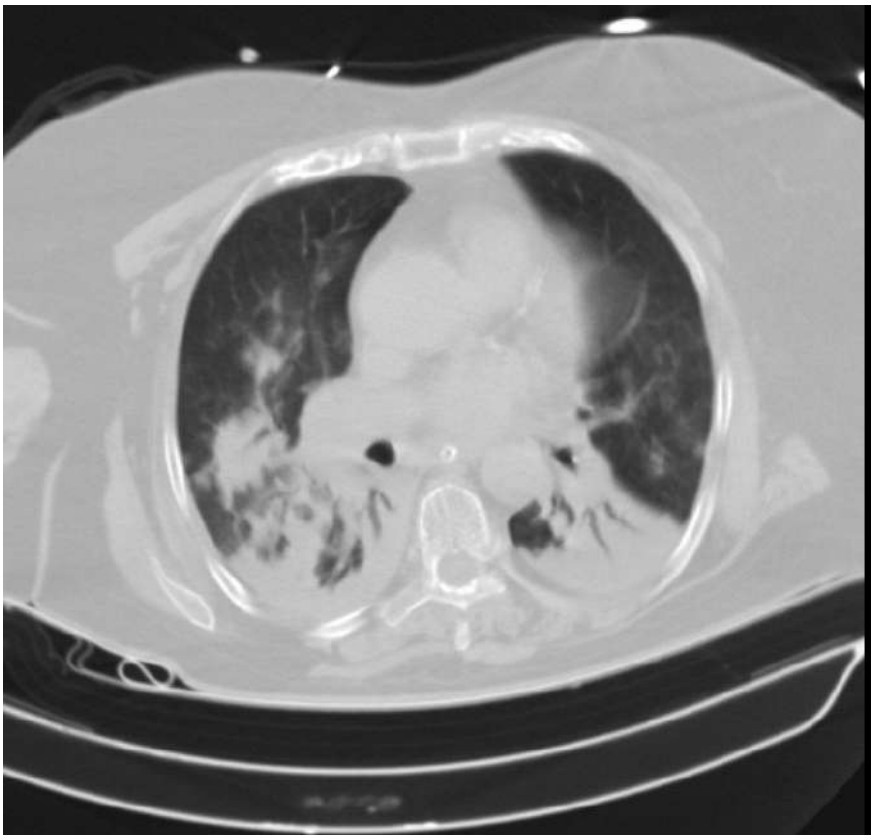

Figure 1: Patient Chest CT İmaging

Discussion

Colchicine toxicity leads to systemic collapse by disrupting microtubule flow at the cellular level. By binding to microtubule proteins (tubulin), colchicine disrupts intracellular transport and cytoskeletal structure, initiating direct lysis in tissues with rapid protein turnover (1,2). In this case, the patient's known CKD and DM diagnoses dramatically altered the pharmacokinetic process of the drug. While colchicine is largely excreted in urine; however, the drug itself creates a vicious cycle that completely zeros out clearance by clogging renal tubules via rhabdomyolysis (myoglobinuria) (3).

An initial creatinine of 1.2 mg/dL was actually an indicator of decreased functional renal reserve, considering the patient's age and CKD history. The surge of creatinine to 4.8 mg/dL,

CK to 37,000 U/L, and LDH to 2234 U/L at the time of referral proves how rapidly colchicine can cause massive tissue damage in a patient with limited renal clearance (3,4).

The severe gastrointestinal distress seen in the first stage of colchicine toxicity triggers unstoppable vomiting episodes, dramatically increasing the risk of aspiration (2). Aspiration pneumonia was the main factor worsening the prognosis in this case. Furthermore, clouding of consciousness or neuromuscular involvement developing in later stages of toxicity weakens the patient's airway protection reflexes, facilitating this process (5). In our case, although acute kidney injury and rhabdomyolysis were treated and fluid-electrolyte balance was achieved via dialysis, the widespread lung infiltrations and ARDS following aspiration necessitated infectious/inflammatory management beyond the toxic phase. The suppressed immune status against the backdrop of DM and CKD explains the progression to sepsis after aspiration and the uncontrolled increase in acute phase reactants (3,6).

Conclusion

Colchicine use in elderly patients and those with limited renal reserve carries a risk of massive rhabdomyolysis and anuric kidney injury, even if vital signs are initially stable. Severe vomiting should be evaluated not just as a drug side effect, but as a serious risk factor for aspiration pneumonia, septic shock, and multiple organ failure. In such complex cases, toxin clearance via hemodialysis alone is insufficient; proactive management of secondary pulmonary complications and multidisciplinary intensive care support are the most critical factors for survival.

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Keywords: Colchicine, Massive rhabdomyolysis, Acute kidney injury, Aspiration pneumonia

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Bee Sting-Induced Acute Coronary Syndrome: A Case of Type I Kounis Syndrome in a Young Patient

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Introduction: Kounis syndrome is an acute coronary syndrome caused by an allergic reaction. Type I Kounis syndrome occurs in patients with completely normal coronary arteries due to coronary vasospasm. It is an important and frequently underdiagnosed cause of MINOCA (Myocardial Infarction with Non-Obstructive Coronary Arteries). This report presents a young male patient who developed a STEMI (ST-Elevation Myocardial Infarction) like clinical picture following a bee sting.

Case Report: A 19-year-old male with no known chronic illness, drug use, or allergy history presented to the emergency department with sudden pressure-like chest pain and shortness of breath. These symptoms began approximately 45 minutes after he was stung by a bee. Physical examination revealed widespread urticaria on his trunk and extremities; however, he was normotensive and had no signs of angioedema. An electrocardiogram (EKG) demonstrated ST-segment elevation in the inferior leads (II, III, aVF) and reciprocal ST depression in leads I, aVL, and V1-V2. The patient was treated with H1 and H2 blockers along with intravenous corticosteroids (methylprednisolone). Urgent coronary angiography (CAG) revealed completely normal coronary arteries with no obstructive lesions, confirming the diagnosis of Type I Kounis Syndrome. Following the regression of his allergic and cardiac symptoms, the patient was discharged with full recovery approximately 36 hours later.

Conclusion: Severe allergic reactions can trigger STEMI-like conditions even in young patients with zero cardiovascular risk factors.Kounis syndrome must be considered in the differential diagnosis for patients presenting with acute chest pain and concurrent allergic symptoms, such as widespread urticaria, even in the absence of angioedema.Early recognition of this syndrome is vital for identifying the underlying etiology of MINOCA cases and ensuring the rapid administration of targeted allergy-specific therapies alongside standard cardiac care.

Keywords: Kounis Syndrome, Acute Coronary Syndrome, Bee Sting

INTRODUCTION

Kounis syndrome was first described as allergic angina by Dr. Nicholas G. Kounis and Dr. George M. Zavras in 1991, and it was explained as histamine-induced coronary artery spasm(1). It is a condition in which mast cell mediators, released during the body's severe allergic response to any agent, cause spasms in the coronary arteries or a loss of stability in existing plaques, thereby disrupting cardiac blood supply and leading to acute coronary

syndrome. Kounis syndrome is an anaphylactoid reaction that specifically and dangerously affects the heart and coronary blood vessels. At this point, it can be stated that the affinity of the mediators and cytokines released during an allergic reaction to the heart and coronary arteries is higher compared to other tissues. Kounis syndrome is generally analyzed in 3 types(2). Firstly, Type I Kounis syndrome is when coronary vasospasm restricts blood flow in patients with normal coronary arteries, causing acute coronary spasm; Type II Kounis syndrome refers to acute coronary syndrome caused by plaque erosion or plaque rupture as a result of vasospasm occurring in already atherosclerotic coronary arteries; in Type III Kounis syndrome, there is an occlusion of a drug-eluting stent. Especially Type I Kounis syndrome is one of the important, often incorrectly diagnosed, causes of MINOCA (Myocardial Infarction with Non-Obstructive Coronary Arteries)(3).

In this article, a 19-year-old male patient is presented who showed an inferior ST elevation on ECG despite remaining normotensive following an anaphylactic reaction that developed after a bee sting. Upon detecting no stenosis in the patient's coronary angiography, he was diagnosed with Kounis Type I Syndrome ; through this case, the fact that allergic reactions can trigger STEMI (ST-Elevation Myocardial Infarction)-like clinical pictures even in young and risk-free patients, as well as the difficulties in the diagnostic process, are discussed.

CASE REPORT

A 19-year-old male patient presented to our emergency department with complaints of sudden-onset, pressure-like chest pain and shortness of breath. The patient had no known history of chronic illness, medication use, or allergies in his medical history. In his anamnesis, he described being stung by a bee approximately 45 minutes before the onset of his symptoms.

In his physical examination; the patient was conscious, oriented, and cooperative. Breath sounds were natural, and there were no findings of uvular edema or angioedema. Skin examination revealed widespread urticaria on the trunk and extremities. His vital signs were stable: Blood pressure 126/82 mm/Hg (normotensive), heart rate 91/min, respiratory rate 18/min, SpO2 98%, and temperature 36.6 °C.

Figure 1: The patient's electrocardiography in the emergency department:

In the 12-lead electrocardiography (ECG) taken upon the patient's arrival;

ST segment depression was observed in leads I, aVL, and V1-V2.

ST segment elevation is present in leads II, III, and aVF (inferior).

The patient, whose ECG findings and clinical picture were compatible with an inferior STEMI, was started on H1 and H2 blockers and intravenous corticosteroids (methylprednisolone) as antihistamine treatment. The patient underwent emergent coronary angiography (CAG). The CAG showed that the coronary arteries were normal and no

occlusive stenosis was detected. With these findings, the patient was diagnosed with Kounis Syndrome Type I, which is associated with coronary vasospasm triggered by a bee sting on a background of normal coronary arteries. During the patient's follow-up, his cardiac and ECG findings regressed, and the allergic reaction subsided. The patient was discharged with complete recovery after approximately 36 hours.

DISCUSSION AND CONCLUSION

Kounis syndrome was first described as "allergic angina" by Dr. Nicholas G. Kounis and Dr. George M. Zavras in 1991, and it entered the literature as histamine-induced coronary artery spasm. The affinity of mast cell mediators and cytokines, which are released during the allergic reaction process, to the heart and coronary arteries is higher compared to other tissues. These mediators can disrupt cardiac blood supply, leading to acute coronary syndrome.

In this case we presented, acute coronary syndrome findings developed within 45 minutes following a bee sting in a young, 19-year-old male patient who had no history of chronic disease, medication use, or allergies in his medical history. In the patient's electrocardiography (ECG), ST segment elevation in the inferior leads (II, III, and aVF) and reciprocal depression (I, aVL, V1-V2) were detected, displaying a clinical picture compatible with an inferior STEMI. However, the emergent coronary angiography (CAG) of the patient, who remained normotensive, demonstrated that the coronary arteries were completely normal and there was no occlusive stenosis. This clinical picture, characterized by vasospasm restricting blood flow in patients with normal coronary arteries, is in complete accordance with Kounis Syndrome Type I(4).

At this point, it is necessary to particularly emphasize the importance of diagnosing Kounis syndrome correctly and rapidly. Type I Kounis syndrome, in particular, is one of the very important and frequently misdiagnosed causes of myocardial infarction with non-obstructive coronary arteries (MINOCA) cases. Considering the fact that allergic reactions can trigger STEMI-like clinical pictures even in young and cardiovascularly completely risk-free patients, overcoming the difficulties in the diagnostic process and recognizing the underlying allergic mechanism is of critical importance in terms of patient management.

Keeping Kounis syndrome in mind and diagnosing it correctly has vital importance in elucidating the cause of MINOCA (myocardial infarction with non-obstructive coronary arteries) cases. Timely diagnosis ensures the prompt initiation of specific treatments (H1 and H2 blockers and intravenous corticosteroids) aimed at the main triggering mechanism, the allergy, in addition to the classic coronary syndrome approach(5). This case demonstrates that allergic reactions can create severe STEMI (ST-Elevation Myocardial Infarction)-like clinical pictures even in young and risk-free patients. The patient demonstrating ischemic findings on ECG despite being normotensive once again proves the diagnostic difficulties in such cases and the importance of suspecting Kounis syndrome. In patients presenting with acute chest pain and having concurrent allergic findings such as widespread urticaria , Kounis syndrome must absolutely be evaluated in the differential diagnosis, even if angioedema is absent(6). As

a result of rapid angiographic evaluation and correct medical treatment planning directed at the underlying allergic cause, our patient was discharged with a complete recovery within approximately 36 hours.

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The Return of the "Forgotten Disease": From Simple Pharyngitis to Septic Shock

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Introduction

Lemierre syndrome (LS) is a rare but life-threatening complication of bacterial pharyngitis, first characterized by André Lemierre in 1936 after reporting 20 cases of anaerobic septicemia originating from throat infections. The condition involves the extension of an oropharyngeal infection into the lateral pharyngeal spaces, resulting in septic thrombophlebitis of the internal jugular vein (IJV). Due to the high prevalence of benign sore throats in the young, healthy population it typically affects, the diagnosis of LS is often elusive on initial presentation, frequently leading to critical treatment delays (1).

The syndrome primarily originates from bacterial throat infections, with Fusobacterium necrophorum—an obligate anaerobic, gram-negative bacillus—and Fusobacterium nucleatum being

the most common pathogens. These bacteria utilize various virulence factors, including endotoxins and exotoxins, to cause invasive disease. Other organisms, such as Streptococcus species, Bacteroides species, Staphylococcus aureus, and Klebsiella pneumoniae, have also been implicated (2-4).

While *F. necrophorum* is a component of the normal flora in the pharynx and gastrointestinal tract, certain conditions—such as viral co-infections (e.g., acute Epstein-Barr) or bacterial infections (e.g., streptococcal)—can create an environment conducive to anaerobic growth. Peritonsillar abscesses, in particular, allow the bacteria to penetrate surrounding tissues and seed the IJV. This leads to septicemia and the subsequent dissemination of septic emboli throughout the venous system (5, 6).

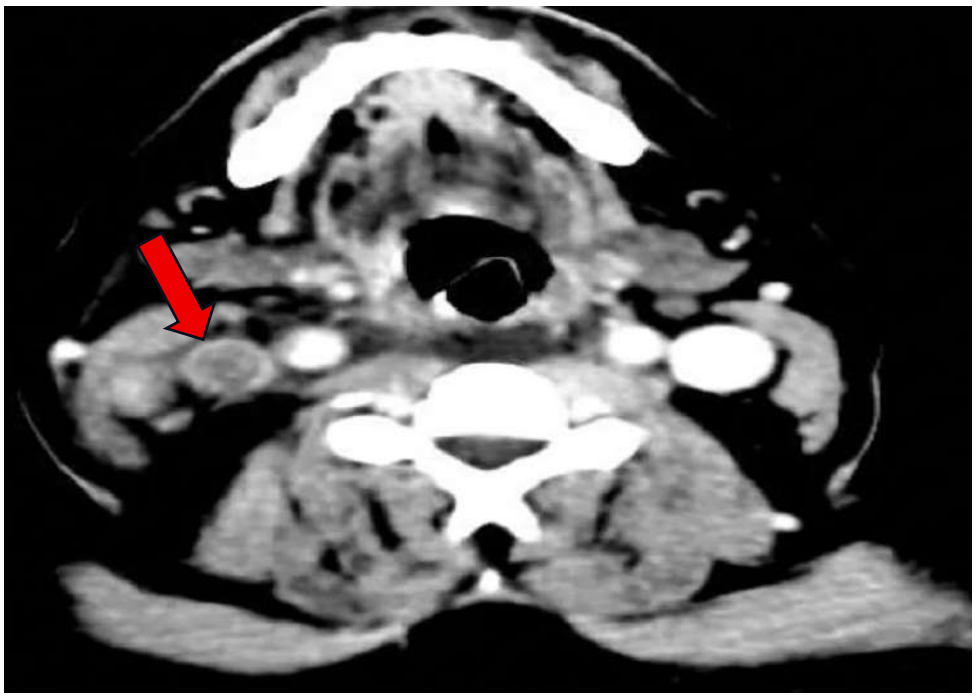
Historically, the incidence of LS declined sharply in the 1960s and 1970s with the widespread use of penicillin. however, since the late 1970s, there has been a steady rise in reported cases, potentially due to more restrictive antibiotic prescribing patterns for routine oropharyngeal infections. Despite this trend, LS remains rare, with an estimated worldwide incidence of 1 in 1,000,000. It predominantly affects adolescents and young adults, with a median age between 19 and 22 years and a notable 2:1 male-to-female ratio. Approximately 90% of patients are between the ages of 10 and 35 (1).

Case Presentation

A 23-year-old male presented to the emergency department with a primary complaint of fever, neck pain, and shortness of breath. His medical history revealed an untreated sore throat seven days prior , followed by the onset of localized neck pain three days before admission. Within the final 24 hours, the patient's condition deteriorated, manifesting as high fever, respiratory distress , generalized fatigue , and dysphagia. Upon physical examination, the patient was febrile at 39.2°C , tachycardic at 120 beats per minute , and hypotensive with a blood pressure of 100/60 mmHg. Notable clinical findings included tenderness and swelling along the right sternocleidomastoid muscle and the presence of basal rales during pulmonary auscultation.

Laboratory investigations showed a white blood cell count of 18,000/µL along with significantly elevated C-reactive protein and procalcitonin levels. Contrast-enhanced computed tomography (CT) of the neck identified a filling defect in the right internal jugular vein (Figure-1), accompanied by vessel wall thickening and surrounding soft tissue inflammation. A concurrent chest CT revealed bilateral peripheral nodular lesions, some of which exhibited cavitation consistent with septic pulmonary emboli. Blood cultures confirmed the presence of Fusobacterium necrophorum , establishing a diagnosis of Lemierre syndrome. The patient was transferred to the intensive care unit for monitoring and managed with intravenous piperacillin-tazobactam and metronidazole , supplemented by anticoagulation therapy.

Figure 1: Internal jugular vein thrombosis (red arrow)



Discussion

Lemierre's syndrome (LS), often termed the "forgotten disease," remains a rare but high-morbidity clinical entity primarily affecting healthy young adults with a mean age of 20 years (7). Once *F. necrophorum* invades the cervical vasculature, septic emboli frequently disseminate, most commonly involving the lungs (85%), followed by the joints, liver, kidneys, brain, and heart (1, 8).

Systemic bacteremia typically manifests as fever, lethargy, or shock, with septic shock occurring in approximately 7% of cases. Furthermore, up to 10% of patients develop acute respiratory distress syndrome (ARDS) requiring mechanical ventilation. Clinicians should maintain a high index of suspicion for "post-anginal sepsis" when fever and rigors suddenly emerge 4–7 days after pharyngeal symptoms have stabilized (1, 7).

Physical examination is critical; tenderness along the sternocleidomastoid (SCM) muscle serves as the earliest clinical indicator of internal jugular vein (IJV) thrombosis. In cases of clinical suspicion, any delay in obtaining a contrast-enhanced CT scan—the gold standard for diagnosis—directly correlates with increased mortality (1, 8).

Conclusıon

Lemierre syndrome is a rare but life-threatening "forgotten disease" that primarily affects healthy young adults. As seen in this case, it typically starts with a simple sore throat but progresses to internal jugular vein thrombosis and septic emboli, most commonly affecting the lungs.

Early diagnosis is critical to reducing mortality. Clinicians should maintain a high index of suspicion if a patient develops high fever and neck tenderness—specifically along the sternocleidomastoid muscle—shortly after a pharyngeal infection. Prompt imaging with contrast-enhanced CT and the

initiation of aggressive antibiotic therapy against *Fusobacterium necrophorum* are essential for successful treatment.

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Severe Burns, Respiratory Deterioration, And Cardiopulmonary Resuscitation: A Case Report Discussing Extracorporeal Membrane Oxygenation

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INTRODUCTION:

Burns are complex traumatic injuries that constitute major causes of mortality and morbidity worldwide. They have been reported to result in the death of more than 300,000 people each year (1). Severe burn is defined as a second-degree or higher-degree burn involving more than 20% of the total body surface area which is not limited solely to cutaneous tissue injury. It

may lead to a systemic clinical condition that can progress to multiple organ failure through a hypermetabolic stress response, marked inflammatory activation, increased stress hormones, and impaired microvascular permeability (1-3). Lung is one of the most frequently and earliest affected organ after severe burn. Pulmonary involvement may substantially worsen the clinical course of the burn (3). In the lungs, disruption of the integrity of the alveolo-capillary membrane due to both increased hydrostatic load caused by aggressive fluid therapy administered during resuscitation and permeability alterations triggered by various inflammatory mediators may lead to severe and progressive impairment of ventilation within a broad pathophysiological spectrum ranging from pulmonary edema to acute respiratory distress syndrome (ARDS) (4,6). Massive fluid resuscitation, an unavoidable component of acute burn management, may further aggravate this respiratory deterioration by causing prominent interstitial and alveolar edema in lung parenchyma (1,2). When all these pathophysiological processes are considered together, an important hypothesis emerges that, theoretically, ventilation may not always be provided at an adequate and effective level by endotracheal intubation (ETI) in patients with severe burns. This case report aims to discuss the possible role and potential rationale of extracorporeal membrane oxygenation (ECMO) use in patients with severe burns within the framework of the relevant pathophysiological mechanisms.

CASE PRESENTATION

A 52-year-old male textile factory worker presented to the emergency department (ED) with extensive burns that developed after a tank containing water and acetic acid at 70–80°C spilled on him. Clinical evaluation revealed extensive second-degree burn areas involving almost the entire body, with relative sparing of the feet and ankles. Although the initial pulmonary auscultation at presentation was considered normal, laryngoscopic examination of the airway revealed marked vocal cord edema, which was considered notable in terms of progressive airway obstruction. The patient's first documented vital signs at presentation are presented in detail in Table 1. Owing to developing airway failure and the risk of possible rapid clinical deterioration, ETI was performed approximately 20 minutes after presentation. Simultaneously, intravenous Ringer's lactate infusion, broad-spectrum antibiotic therapy, analgesic treatment, and tetanus prophylaxis were initiated. Approximately 10 minutes after ETI, the patient developed cardiac arrest. Cardiopulmonary resuscitation (CPR) was performed in accordance with advanced life support principles, and repeated resuscitation cycles were carried out. Despite the resuscitation attempts, return of spontaneous circulation could not be achieved, and the patient was pronounced dead. As a clinically important finding, approximately 600 mL of hemorrhagic fluid was aspirated from the endotracheal tube during CPR (Figure 1).

DISCUSSION AND CONCLUSION

Review of the current literature on ECMO use in burn patients indicates that there is no definitive and widely accepted consensus regarding in which patient group, at which timing, and within which indication framework it should be used (1,2,6-9). Nevertheless, it has been reported that mortality rates remain high in patients with severe burns who undergo ECMO for respiratory failure, and outcomes in most series have not reached the desired level. However, mortality is also extremely high among patients requiring CPR after severe burn, and the prognosis is generally very poor (10,11). Therefore, whether ECMO can be considered as a rescue treatment option in appropriately selected cases among patients requiring CPR after severe burn represents a fundamental area of discussion that is important for clinical decision-making processes and requires clarification through further research.

Table 1. The patient's documented vital signs on arrival

Blood Pressure	Heart Rate	Body Temperature	Fingertip Oxygen Saturation	Respiratory Rate
124/73 mmHg	87 beats/min	36.1 °C	93 %	26 breaths/min

Figure 1. Approximately 600 mL of hemorrhagic fluid aspirated from the endotracheal tube during cardiopulmonary resuscitation.

KEYWORDS: Acute respiratory distress syndrome, cardiopulmonary resuscitation, extracorporeal membrane oxygenation, in-hospital cardiac arrest, severe burn

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7856

Takotsubo Cardiomyopathy Following Acute Emotional Stress: A Case Mimicking Acute Coronary Syndrome

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Abstract

Takotsubo cardiomyopathy is a transient cardiomyopathy characterized by reversible left ventricular systolic dysfunction that may occur following intense emotional or physical stress and can clinically mimic acute coronary syndrome. The clinical presentation commonly includes chest pain, dyspnea, electrocardiographic changes, and elevated troponin levels, making differentiation from acute myocardial infarction challenging. Definitive diagnosis is established by excluding obstructive coronary artery disease through coronary angiography and demonstrating characteristic left ventricular wall motion abnormalities.

In this case report, a 54-year-old female patient presenting to the emergency department with dyspnea and bilateral arm numbness following intense emotional stress is described. Electrocardiography revealed QT interval prolongation and nonspecific repolarization abnormalities. High-sensitivity troponin I levels were elevated, with mild dynamic changes observed in serial measurements (124 ng/L and 126 ng/L; reference range 0–46.47 ng/L). CK-MB levels were within normal limits (2.56 ng/mL). Transthoracic echocardiography demonstrated a left ventricular ejection fraction of 45–50%, with hypokinesia involving the apical and mid-anterior segments and relatively preserved contractility of the basal segments. Emergency coronary angiography revealed no significant stenosis in the epicardial coronary arteries.

Considering the clinical findings, echocardiographic wall motion abnormalities, and the exclusion of obstructive coronary artery disease on coronary angiography, the patient was diagnosed with Takotsubo cardiomyopathy in accordance with the InterTAK diagnostic criteria. This case emphasizes that Takotsubo syndrome should always be considered in the differential diagnosis in patients presenting with elevated troponin levels and electrocardiographic changes following emotional stress.

Keywords

Takotsubo cardiomyopathy, Acute coronary syndrome, Emotional stress, Coronary angiography, Apical ballooning, Troponin elevation

Introduction

Takotsubo syndrome (TTS) is a type of cardiomyopathy that may develop following intense emotional or physical stress and can mimic acute coronary syndrome in terms of clinical, electrocardiographic, and biochemical findings (1,3). Although transient left ventricular systolic dysfunction and regional wall motion abnormalities are observed, obstructive coronary artery disease is not detected on coronary angiography (1,7).

The syndrome was first described in Japan and was named “takotsubo” due to the characteristic appearance of apical ballooning of the left ventricle during systole, resembling a traditional octopus trap (6). Epidemiologically, the majority of patients are postmenopausal women, and an emotional or physical trigger is identified in approximately 80–90% of cases (5,6).

The most widely accepted pathophysiological mechanism involves catecholamine surge–induced myocardial stunning, microvascular dysfunction, and direct myocyte toxicity (4,14). This process particularly affects the apical segments of the left ventricle, leading to pronounced wall motion abnormalities (6).

In this case report, the clinical and imaging findings of Takotsubo cardiomyopathy are presented in a patient initially evaluated with a preliminary diagnosis of acute coronary syndrome and in whom obstructive coronary artery disease was excluded by coronary angiography.

Case Presentation

A 54-year-old female patient presented to the emergency department with sudden-onset dyspnea and bilateral arm numbness following attendance at a funeral. The patient did not report chest pain. Her medical history included type 2 diabetes mellitus and hypothyroidism. She was on regular medications including metformin/sitagliptin (Janumet® 50/1000 mg), gliclazide (Diamicron® 30 mg), and levothyroxine (L-thyroxine 150 µg). There was a family history of coronary artery disease.

On physical examination, the patient was in good general condition, conscious, oriented, and cooperative. Cardiac auscultation revealed normal S1 and S2 sounds without additional sounds or murmurs. Pulmonary auscultation was normal, and no peripheral edema was observed. Vital signs were stable.

Electrocardiography showed sinus rhythm (67 bpm). No ST-segment elevation was observed; however, nonspecific repolarization changes in the anterior leads and QT interval prolongation were noted.

Laboratory investigations revealed elevated high-sensitivity troponin I levels with mild dynamic changes in serial measurements (124 ng/L and 126 ng/L; reference range 0–46.47 ng/L). CK-MB levels were within normal limits (2.56 ng/mL). Renal function tests and electrolyte levels were normal.

Transthoracic echocardiography demonstrated a left ventricular ejection fraction of 45–50%. Hypokinesia involving the apical and mid-anterior segments of the left ventricle was observed, with relatively preserved contractility in the basal segments. Pulmonary artery pressure was estimated at approximately 30 mmHg.

Emergency coronary angiography was performed with a preliminary diagnosis of acute coronary syndrome. The angiographic evaluation revealed no hemodynamically significant stenosis in the left main coronary artery, left anterior descending artery (LAD), circumflex artery (LCx), or right coronary artery (RCA).

Considering the history of intense emotional stress, elevated troponin levels, regional wall motion abnormalities on echocardiography, and the exclusion of obstructive coronary artery disease on coronary angiography, the patient was diagnosed with Takotsubo cardiomyopathy according to the InterTAK diagnostic criteria.

Natriuretic peptide levels were not measured in this patient. Cardiac magnetic resonance imaging was not performed, and the diagnosis was supported by clinical findings, echocardiographic wall motion abnormalities, and coronary angiography results. Long-term echocardiographic follow-up data were not available.

Discussion

Takotsubo syndrome is a transient cardiomyopathy that develops following intense emotional or physical stress and can mimic acute coronary syndrome (1,3). The majority of patients are postmenopausal women, and a triggering emotional or physical factor is identified in most cases (5,6).

Large patient series in the literature have demonstrated the significant role of emotional stress as a trigger (5,8). In the present case, intense emotional stress following a funeral was considered the precipitating factor.

QT prolongation and repolarization abnormalities are common electrocardiographic findings in Takotsubo syndrome and may increase the risk of ventricular arrhythmias in some patients (9,15).

Troponin elevation in Takotsubo syndrome is generally more limited compared to myocardial infarction and may be disproportionate to the degree of left ventricular dysfunction (5,11). In this case, the modest increase in troponin levels was consistent with the biochemical pattern reported in the literature.

The differential diagnosis includes acute myocardial infarction, myocarditis, and myocardial infarction with non-obstructive coronary arteries (MINOCA) (12,13). Therefore, coronary angiography is critical for accurate diagnosis in patients presenting with suspected acute coronary syndrome (1,13).

One of the notable features of this case is the absence of typical chest pain despite elevated myocardial injury biomarkers. This may pose diagnostic challenges in emergency settings and highlights the importance of considering Takotsubo syndrome in patients with a history of emotional stress (7).

This case report has several limitations. Natriuretic peptide levels were not measured, cardiac magnetic resonance imaging was not performed, and long-term echocardiographic follow-up data were unavailable. However, the diagnosis is strongly supported by the combination of clinical presentation, echocardiographic findings, and coronary angiography results (10).

Conclusion

Takotsubo syndrome is an important clinical entity that can mimic acute coronary syndrome (1). It should be considered in the differential diagnosis, particularly in patients with a history of emotional stress, elevated troponin levels, and regional wall motion abnormalities after exclusion of coronary artery disease. Early recognition in emergency departments may contribute to appropriate clinical management and prevent unnecessary invasive interventions (1,3).

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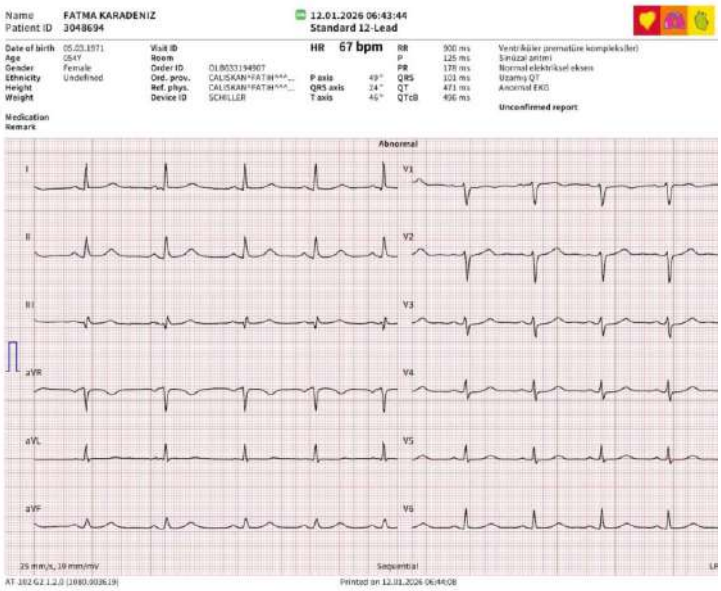

Figure 1

A 12-lead electrocardiogram obtained at presentation demonstrates sinus rhythm (67 bpm), nonspecific repolarization changes in the anterior leads, and QT interval prolongation (QTc approximately 496 ms). No acute ST-segment elevation is observed.

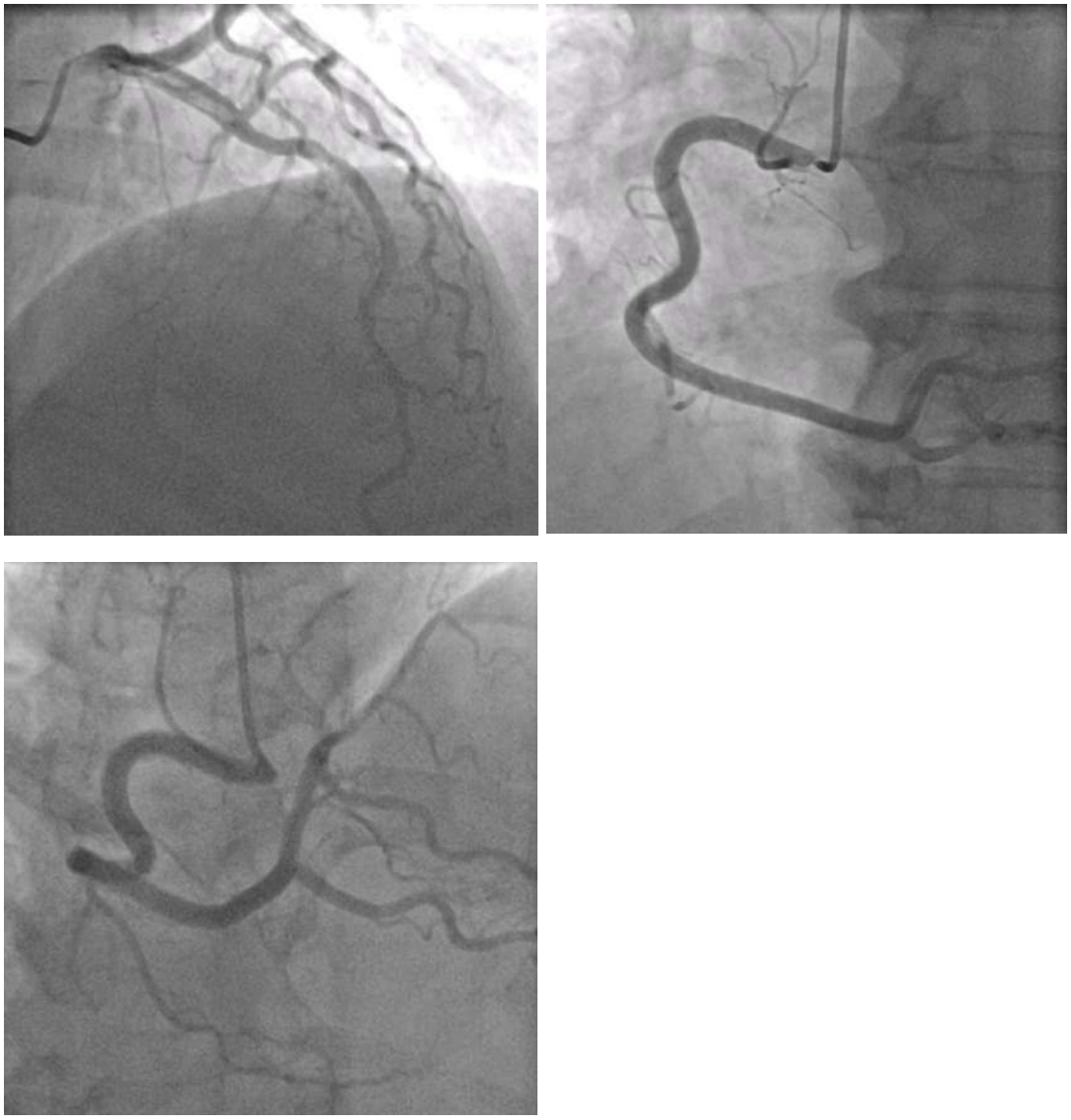

Figure 2A–C

Coronary angiography images demonstrate normal caliber of the left main coronary artery, left anterior descending artery (LAD), circumflex artery (LCx), and right coronary artery (RCA), with no hemodynamically significant stenosis or acute thrombotic lesion in the epicardial coronary arteries.

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Don't Forget the Electrolytes: Hypokalemic Periodic Paralysis as a Cause of Acute Quadriparesis

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Introduction

Hypokalemic periodic paralysis (HPP) is a hereditary muscle disorder characterized by episodic attacks resulting from genetic mutations affecting ion channels in the muscle cell membrane [1,2]. It is most commonly associated with mutations in the CACNA1S gene encoding calcium channels and the SCN4A gene encoding sodium channels [3].

Clinically, the disease presents with sudden-onset flaccid muscle weakness predominantly involving proximal muscle groups. Attacks may last from several hours to days and are typically associated with hypokalemia [1]. Common triggering factors include rest after strenuous exercise, high-carbohydrate meals, and emotional stress [2].

Although hypokalemic periodic paralysis is a rare cause of acute paralysis, early recognition is crucial because the condition is potentially reversible.

Case Presentatıon

A 27-year-old male patient presented to the emergency department with sudden-onset weakness and numbness affecting both upper and lower extremities. His medical history revealed no known systemic disease or regular medication use.

On physical examination, vital signs were stable. The patient was fully conscious with a Glasgow Coma Scale score of 15. Neurological examination demonstrated muscle strength of 2/5 in all extremities. No significant sensory deficits were identified. Bilateral pupillary light reflexes and corneal reflexes were intact. Pupils were isochoric and normoreactive, and extraocular movements were full in all directions.

Laboratory investigations revealed decreased serum potassium levels, while other biochemical and hematological parameters were within normal limits.

Based on the clinical presentation and laboratory findings, the patient was admitted to the neurology department with a preliminary diagnosis of hypokalemic periodic paralysis, and potassium replacement therapy was initiated.

Tartışma

Although rare, hypokalemic periodic paralysis represents an important cause of acute quadriparesis, particularly in young male patients [1]. The disorder may occur as a primary (familial) condition or secondary to other diseases, most notably thyrotoxicosis [4,5].

In the present case, the absence of a known systemic disease and presentation with a first attack suggested primary HPP. Nevertheless, exclusion of secondary causes remains essential for appropriate clinical management [2].

The differential diagnosis includes Guillain–Barré syndrome, myasthenia gravis, acute myopathies, and other electrolyte disturbances [1]. Guillain–Barré syndrome is typically characterized by progressive weakness and areflexia, whereas myasthenia gravis presents with fatigable muscle weakness. In contrast, HPP is distinguished by sudden-onset, usually painless muscle weakness associated with hypokalemia [2].

Conclusion

Hypokalemic periodic paralysis should be considered as a rare yet reversible cause of acute quadriparesis in young patients presenting to the emergency department. Early diagnosis and prompt initiation of appropriate electrolyte replacement therapy are essential to reduce morbidity.

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Keywords: Hypokalemic periodic paralysis, hypokalemia, muscle weakness, acute paralysis

7861

Salicylate intoxication following ingestion of topical ointments: A case report

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ABSTRACT

In this case report, we aimed to present a 28-year-old male who developed salicylate intoxication after ingesting multiple topical ointments in a suicide attempt. A 28-year-old male with a known diagnosis of schizophrenia presented to the emergency department stating that he had ingested several tubes of ointment in a suicide attempt. According to information obtained from his relatives, the patient had ingested Klo-sal lotion© (0.05% clobetasol 17-propionate), Momesalic lotion© (mometasone furoate and salicylic acid), Betasalic lotion© (betamethasone dipropionate), Hametan cream© (Hamamelis virginiana), two tubes of Advantan cream© (methylprednisolone aceponate), Üresal Forte cream© (urea [carbamide] and salicylic acid), Fucidin cream© (fusidic acid), and Duoderm solution© (salicylic acid–lactic acid). It was determined that the patient had ingested more than 6.5 g of salicylic acid–containing preparations, corresponding to a toxic dose. The measured serum salicylate level was 10.4 mg/dL. Following supportive treatment, the control salicylate level decreased to 5.6 mg/dL. After completion of intoxication monitoring and clinical improvement, a psychiatric consultation was requested. On the fourth day of intensive care unit follow-up, the patient was transferred to a hospital with a closed psychiatric ward. In patients who ingest large amounts of topical ointments with suicidal intent, the possibility of life-threatening salicylate intoxication should be considered.

Keywords: Emergency, Salicylate intoxication, Topical agents.

INTRODUCTION

Salicylate intoxication is a medical emergency that can lead to serious metabolic disturbances. Salicylates are widely available medications that are frequently used without prescription (1,2). They are commonly utilized due to their analgesic, antipyretic, and antithrombotic properties (3). In mild poisoning cases, symptoms such as tinnitus, nausea, vomiting, and abdominal pain may occur, whereas severe intoxication may lead to seizures, rhabdomyolysis, pulmonary edema, cerebral edema, and renal failure. In addition to oral drug ingestion, salicylate poisoning may also occur through other sources, including excessive application of topical agents or ingestion of salicylate-containing ointments. Because many of these preparations are liquid, highly concentrated, and lipid-soluble, they may result in rapid and severe salicylate intoxication (4). In this case report, we aimed to present a patient who developed salicylate intoxication after ingesting topical ointments in a suicide attempt.

CASE REPORT

A 28-year-old male with a known diagnosis of schizophrenia presented to the emergency department stating that he had ingested several tubes of ointment in a suicide attempt. According to information obtained from his relatives, the patient had ingested Klo-sal lotion© (0.05% clobetasol 17-propionate), Momesalic lotion© (mometasone furoate and salicylic acid), Betasalic lotion© (betamethasone dipropionate), Hametan cream© (Hamamelis virginiana), two tubes of Advantan cream© (methylprednisolone aceponate), Üresal Forte cream© (urea [carbamide] and salicylic acid), Fucidin cream© (fusidic acid), and Duoderm solution© (salicylic acid–lactic acid).The patient had no active complaints at presentation. Vital signs on admission were as follows: body temperature 36.8°C, heart rate 80 beats/min, blood pressure 130/80 mmHg, respiratory rate 14 breaths/min, and oxygen saturation 95%. Physical examination findings were unremarkable. It was determined that the patient had ingested more than 6.5 g of salicylic acid–containing preparations, corresponding to a toxic dose. For appropriate monitoring of salicylate levels and effective management, the patient was referred to a medical center with adequate clinical facilities. At the receiving hospital, the patient was admitted to the intensive care unit. The measured serum salicylate

level was 10.4 mg/dL. No other pathological findings were detected in additional investigations. After administration of supportive treatment, the patient's salicylate level decreased to 5.6 mg/dL. After completion of monitoring for intoxication and clinical improvement, a psychiatric consultation was requested. On the fourth day of intensive care unit hospitalization, the patient was transferred to a hospital with a closed psychiatric ward.

Discussion

Salicylate intoxication remains an important cause of morbidity and mortality. It may occur following acute high-dose ingestion or after chronic exposure to repeated doses (1).

Two important issues should be considered regarding the clinical course of salicylate poisoning. First, the absorption of salicylates may be variable and delayed. Therefore, patients who are asymptomatic at presentation may deteriorate during follow-up. Second, although clinical manifestations such as tinnitus, hyperventilation (respiratory alkalosis), metabolic acidosis, altered mental status, cerebral edema, and pulmonary edema are generally correlated with the ingested dose, individual variability may occur. For this reason, repeated laboratory measurements are essential in addition to clinical monitoring. Accurate interpretation of serum salicylate levels and timely implementation of enhanced elimination techniques are critical for effective treatment. In some cases, referral to a medical center with appropriate clinical capabilities may be necessary (5).In our case, the patient had no active symptoms at presentation and did not show clinical deterioration during follow-up. However, the patient was transferred to a center with adequate clinical resources to ensure proper monitoring of salicylate levels and appropriate management.

Previous studies have demonstrated that salicylates can be significantly absorbed through hydrophilic ointment bases and that intoxication may occur even with topical administration (6). In a study comparing intoxication caused by aspirin ingestion and topical salicylate exposure, topical use was reported to produce symptoms similar to those seen after aspirin ingestion (7). Another case previously reported involved a 78-year-old patient with schizoaffective disorder who developed salicylate intoxication after applying a mixture of

salicylate-containing ointments topically for perceived therapeutic benefit rather than suicidal intent (8). Therefore, it should be kept in mind that salicylate intoxication may occur due to misuse of ointments, particularly in patients with psychiatric comorbidities, regardless of whether the exposure is intentional or accidental.

Conclusion

In patients who ingest large amounts of topical ointments with suicidal intent, the possibility of life-threatening salicylate intoxication should always be considered.

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The Relationship Between Anatomical Localisation and Demographic Characteristics in Proximal Femur Fractures

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Abstract:

Objective

This study aimed to evaluate the relationship between the anatomical location of proximal femur fractures with age, gender, height, weight, and body mass index (BMI) in patients presenting to the emergency department.

Materials and Methods

This prospective study was conducted in the emergency department of a tertiary care hospital. Patients diagnosed with proximal femur fractures between 01.01.2026 and 01.03.2026 were included in the study. Fractures were classified according to their anatomical location as femoral head, femoral neck, intertrochanteric, and subtrochanteric. Patients' age, gender, height, weight, and BMI values were recorded. Statistical analyses were performed using IBM SPSS Statistics 20.0 software. ANOVA or Kruskal-Wallis tests were used to compare continuous variables according to their distribution characteristics, and $p<0.05$ was considered statistically significant.

Results

A total of 37 patients were included in the study. Fifty-four point one percent of the patients were male, and the average age was 70.5±18.4 years. The most common fracture type was femoral neck fracture (45.9%). A statistically significant relationship was found between the anatomical location of the fracture with age and height ($p=0.013$ and $p=0.025$, respectively). Femoral neck and intertrochanteric fractures were found to be more common in the older age group. No statistically significant relationship was found between gender, weight, BMI, and fracture location.

Conclusion

The anatomical location of proximal femur fractures appears to be associated with demographic factors, particularly age and height. Femoral neck and intertrochanteric fractures are more common in older age groups. These findings suggest that demographic characteristics may play a role in the occurrence of proximal femur fractures.

Keywords

Proximal femur fracture, femoral neck fracture, intertrochanteric fracture, emergency department, demographic characteristics

The Relationship Between Anatomical Localisation and Demographic Characteristics in Proximal Femur Fractures

Introduction and Objective:

Femur fractures are common injuries and are among the most significant causes of morbidity worldwide. It is estimated that between one and three million people are affected each year (1).

Femur fractures are now most commonly seen in young patients as a result of traffic accidents and workplace accidents, alongside increased daily activity and technology (2). Furthermore, the increasing prevalence of osteoporosis, alongside the growing geriatric population, appears to be the main cause of femur fractures in elderly patients. The increase in the incidence of femur fractures with age is a result of both the age-related decrease in bone mass in the proximal femur and the age-related increase in the frequency of falls. Proximal femur fractures include femoral neck and intertrochanteric fractures (over 90%) and subtrochanteric fractures (5-10%). Proximal femur fractures account for 14% of all femur fractures (3).

Femur fractures are associated with increased mortality rates. It also causes an increased socioeconomic burden. Another negative aspect of femoral fractures is that patients are at increased risk of subsequent fractures, including in the contralateral hip. The one-year risk of re-fracture worldwide ranges from approximately 2% to 10%, and the lifetime risk is estimated to be 20%, but can be as high as 55% (4).

Considering the negative aspects of proximal femur fractures, it is important to identify patients at risk. For this reason, we evaluated patients with proximal femur fractures in this study. In our study, we compared the anatomical location of proximal femur fractures presenting to the emergency department according to age, height, weight, body mass index, and gender.

Material and methods:

Our study was conducted prospectively in the emergency department of a tertiary hospital. Patients who presented to the emergency department with hip pain between 01.01.2026 and 01.03.2026 and were diagnosed with proximal femur fractures based on radiological imaging

were included in the study. Based on the results of the radiological imaging performed on the patients, the fractures were categorised into femoral head, femoral neck, intertrochanteric, and subtrochanteric fractures. The age, gender, height, and weight of all patients were recorded. Their height, weight, and body mass index (BMI) were calculated.

All data were transferred to an electronic format. Data will be analysed using IBM SPSS Statistics, Version 20.0 (IBM Corporation, Armonk New York, USA). The normality analysis of continuous data will be performed using the Shapiro-Wilk test. Descriptive statistics have been performed and, depending on the suitability of the data, will be presented as mean ± standard deviation (Mean ± SD), median, minimum (min), maximum (max) values, n or percentage (%) values. For the comparison of continuous variables, the ANOVA variance analysis test was used for four independent groups with a normal distribution, and the Kruskal-Wallis variance analysis test was used for those without a normal distribution. For statistical significance, a p-value of $p<0.05$ was considered significant.

Results:

Thirty-seven patients who visited the emergency department on the specified dates were included in the study. Of the patients included in the study, 54.1% (n=20) were male. The average age of the patients was determined to be 70.5. The average BMI of the patients was calculated as 26.1. The age, gender, height, weight, and BMI values of the patients are summarised in Table 1.

The most common type of proximal femur fracture observed in the patients included in the study was femoral neck fracture (45.9%). When the relationship between the anatomical location of the fracture and height, weight, and BMI was evaluated, the statistical relationship between age and height with fractures in all anatomical locations was significant (P values were P=0.013 and P=0.025, respectively). The relationship between the patients' age, gender, height, weight, and BMI and the anatomical location of the fracture is summarised in Table 2.

Variables				
Gender Female (n) Male (n)	**(%45.9) 17** **(%54.1) 20**			
	Mean	**Std. Deviation**	**Minimum**	**Maximum**
Age (years)	70.5	18.4	19	94
Height (cm)	166.5	7.6	150.0	180.0
Weight(kg)	72.1	10.6	55.0	93.0

BMI (kg/m^2)	26.1	3.7	20.8	36.3

Table 1: Patients' age, gender, height, weight and BMI values

Variables	**Femoral head** (n=4, %10.8)	**Femoral neck** (n=17, %45.9)	**Intertrochanteric** (n=11, %29.7)	**Subtrochanteric** (n=5,%13.5)	**P Value**
Age (Years)	52.024.7 (30-81)	79.3±9.3 (55-94)	74.3±11.3 (57-93)	47.2±23.2 (19-81)	**0.013**
Gender, Female	4 (100%)	6 (35.3%)	7 (63.6%)	3 (60.0%)	0.106
Height (cm)	161.8±3.9 (157.0-165.0)	170.4±7.1 (160.0-180.0)	161.7±6.8 (150.0-170.0)	167.6±6.2 (160.0-175.0)	**0.025**
Weight (kg)	82.0±11.2 (70.0-93.0)	73.7±8.3 (60.0-92.0)	67.2±10.9 (55.0-85.0)	69.4±10.7 (60.0-85.0)	0.118
BMI (kg/m^2)	31.3±4.1 (27.5-36.3)	25.4±2.6 (20.8-31.2)	25.7±3.7(21.5-31.2)	24.7±3.8 (21.9-31.2)	0.066

Table 2: Relationship between patients' age, gender, height, weight, and BMI with the anatomical location of the fracture

Discussion:

This study compared the demographic and clinical characteristics of patients according to the location of their femur fractures. Our findings show a statistically significant relationship between fracture type with age and height; however, no significant differences were found in terms of gender, weight, and body mass index (BMI). Furthermore, it was observed that the most common fracture type was femoral neck fracture.

Proximal femur fractures are generally more common in male patients than in female patients (5). This situation was also reflected in our study, which showed a predominance of male patients. However, no correlation was observed between fracture location and gender. This suggests that fracture type is related not only to bone mineral density but also to trauma mechanism and biomechanical factors. The circumstances leading to the fracture were not evaluated in our study.

Femoral neck fractures constitute the majority of proximal femur fractures (6). In our study, femoral neck fractures were also the most common anatomical localisation. We believe this is

related to the average age of our patients. With increasing age, the trabecular structure deteriorates, and the angle between the neck and shaft increases. This situation appears to contribute to the increase in femoral neck fractures with age (7).

Patients' low body weight reduces their bone density. Consequently, having a low BMI leads to more falls, creating a risk for femoral fractures (8). However, in our study, we observed that patients' BMI and weight were not significant in terms of the anatomical location of the fracture. The patients' height was related to the anatomical location of the fracture. It is noteworthy that patients with femoral neck fractures had a longer average height. Increased momentum during falls in taller individuals may increase the force exerted on the hip, thereby affecting the risk of fracture. However, larger studies are needed to clarify the biomechanical basis of this relationship.

Limitations:

Our study was conducted with a very small group of patients. Studies involving larger patient groups could provide more information regarding the demographic characteristics of patients and the anatomical location of fractures. Another limitation of our study is that the mechanism of trauma was not evaluated. We believe that the biomechanical characteristics of trauma may also influence the anatomical location of fractures.

Conclusion:

This study compared the demographic and clinical characteristics of patients according to femur fracture localisations. The findings revealed significant differences between fracture types, particularly in terms of age and height. The occurrence of femoral neck and intertrochanteric fractures in the older age group suggests that these fractures may be associated with osteoporotic processes. On the other hand, subtrochanteric and femoral head fractures were detected in a relatively younger age group. No statistically significant difference was found between fracture localisations in terms of gender, weight, and bone mineral density.

These results suggest that the location of femoral fractures may be related to patient demographic characteristics, and larger, multicentre studies are needed to better understand fracture types.

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8008 Spontaneous Isolated Celiac Artery Dissection: A Rare Case Demonstrating Regression Under Conservative Management

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INTRODUCTION

The celiac trunk, arising from the anterior abdominal aorta at T12, supplies arterial blood to the upper abdominal organs (1). While aortic dissections are relatively common, isolated visceral artery dissections are rare. Among them, superior mesenteric artery involvement is most frequent; isolated celiac artery dissection is extremely uncommon (2). We present a case

of spontaneous isolated celiac artery dissection with regression under conservative management.

CASE PRESENTATION

A 60-year-old female presented with acute, tearing epigastric pain of one-day duration. She reported no vomiting, fever, diarrhea, or melena. Her history included hyperlipidemia and prior breast cancer, treated with tamoxifen and atorvastatin. On examination, vital signs were normal and abdominal findings unremarkable. Laboratory workup revealed elevated D-dimer (~1500 ng/mL), with other results within normal limits.

Suspicion for aortic pathology prompted thoracoabdominal CT angiography, which revealed a dissection flap at the celiac trunk origin and a 2.5 cm segment with 12 mm aneurysmal dilatation. No additional vascular or organ abnormalities were noted.

Given the aneurysm size (<15 mm) and stable hemodynamics, conservative management was chosen, including blood pressure control and lifestyle modification. Endovascular stenting was discussed but declined by the patient. Follow-up CT at six months showed ~1 mm regression in aneurysm diameter, and continued medical management was recommended.

DISCUSSION

Isolated celiac artery dissection is rare, often asymptomatic, and may present with epigastric or back pain (3). Risk factors include hypertension, atherosclerosis, connective tissue disorders, and pregnancy (4). CT angiography is the diagnostic gold standard (5).

Management depends on hemodynamic stability, dissection extent, and aneurysm presence. In stable patients, conservative treatment with radiologic monitoring is usually sufficient; intervention is reserved for progression, rupture risk, or organ ischemia (6). In our case, conservative management was safe and effective, with regression at six months.

Clinicians should consider visceral artery dissection in patients presenting with acute severe postprandial epigastric pain, despite its rarity (7).

KEYWORDS

Aneurysm; Celiac Artery; Dissection; Spontaneous; Visceral Artery

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Right Lower Quadrant Pain Is Not Always Appendicitis: A Case of Suspected Appendiceal Neoplasm

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Keywords: Appendicitis, appendiceal neoplasm, acute abdomen

Introduction

Right lower quadrant pain is one of the most common causes of emergency department visits and the most common etiology is acute appendicitis (1). Acute appendicitis usually presents with symptoms including abdominal pain starting in the periumbilical region and localizing to the right lower quadrant, nausea, vomiting and loss of appetite (1). On physical examination, peritoneal irritation findings including tenderness in the right lower quadrant, defense and rebound provide important clues in the diagnosis. In addition to laboratory findings, imaging methods play an important role in the diagnostic evaluation. Especially abdominal ultrasonography and computed tomography are commonly used methods in the diagnosis of acute appendicitis (1).

However, appendiceal neoplasms are rare pathologies and are found in approximately 0.5-1% of all appendectomy materials (2). Among appendiceal tumors, neuroendocrine tumors, mucinous neoplasms and adenocarcinomas are the most common (3). Since they often present clinically similar symptoms with acute appendicitis, the diagnosis is frequently made by postoperative pathologic examination (3). Significantly increased diameter of the appendix,

luminal dilatation or atypical morphologic findings on imaging methods may raise suspicion for neoplasia (4). Therefore, it is important to evaluate clinical, laboratory and imaging findings together in patients presenting with right lower quadrant pain.

Case Report

A 26-year-old male patient with no known chronic disease presented to the emergency department with abdominal pain that started one day ago. It was learned that the pain was diffuse in the beginning and localized to the right lower quadrant over time. There was no history of nausea, vomiting, fever or anorexia. He had no history of trauma and stated that he had not experienced a similar complaint before.

Physical examination performed in the emergency department revealed stable vital signs. Abdominal examination revealed marked tenderness in the right lower quadrant. However, no signs of defense and rebound were observed. No pathologic findings were found in other abdominal quadrants. Laboratory tests revealed a high leukocyte count of $18.18\times10^3/\mu L$ and leukocytosis was dominated by neutrophils. C-reactive protein value was 1.7 mg/L. Other hemogram and biochemistry parameters were within normal limits.

Abdominal ultrasonography showed that the appendix did not respond to compression and was approximately 15 mm in diameter. Contrast-enhanced abdominal-pelvis tomography also showed a 15 mm appendix. Although these findings were compatible with acute appendicitis, appendiceal neoplasia was also considered suspicious because of the marked increase in the diameter of the appendix. Detailed anamnesis revealed that the patient had a family history of colon cancer in his father and grandfather. Therefore, the patient was consulted to the general surgery clinic for further evaluation and treatment planning. Laparoscopic appendectomy was planned by general surgery and the patient was transferred to the ward. After the operation, the diagnosis of appendiceal neoplasm was confirmed.

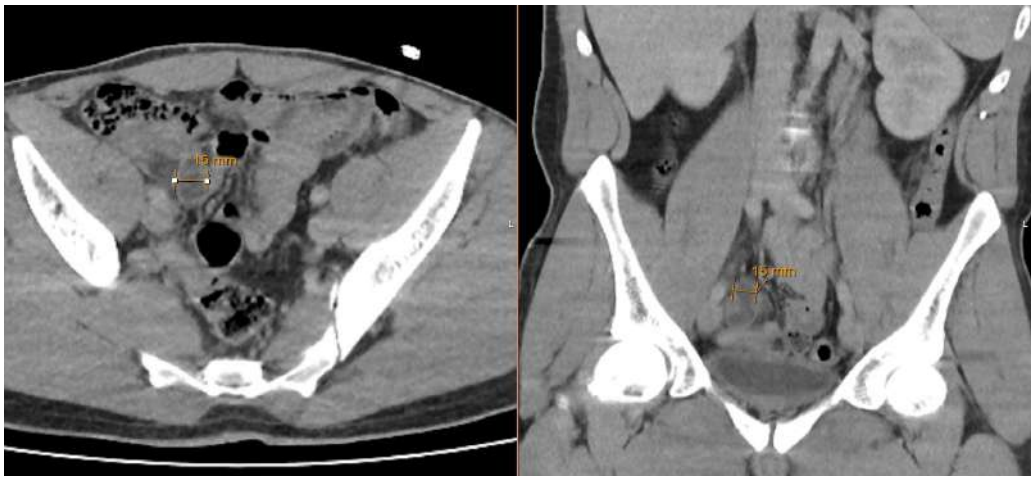

Figure 1. Dilated (≈15 mm) appendix in the right lower quadrant on abdominal CT axial and coronal section

Discussion

Preoperative diagnosis of appendiceal neoplasms is very difficult and most cases are diagnosed by pathologic examination after surgery with a prediagnosis of acute appendicitis (3). However, thanks to the widespread use of imaging modalities in recent years, suspicion of neoplasia may occur preoperatively in some cases (4). Especially ultrasonography and computed tomography examinations findings such as marked increase in appendix diameter, luminal dilatation, wall irregularity or peripheral mass appearance may be warning signs in terms of malignancy (4).

In the literature, it has been reported that an appendix diameter of 15 mm and above may raise suspicion in terms of neoplasia (5). In addition, an appendix that does not respond to compression and appears markedly dilated is among the atypical findings (4). Therefore, careful evaluation of imaging findings is important in terms of diagnostic approach.

In addition, a family history of colorectal cancer is considered a risk factor for some gastrointestinal malignancies (6). Therefore, detailed questioning of family history even in young patients is an important step that may affect the diagnostic approach.

In the present case, in a young patient who presented with right lower quadrant pain, the appendix diameter was measured as 15 mm on ultrasonography and did not respond to compression, which raised suspicion for appendiceal neoplasm. In addition, the patient's family history of colon cancer necessitated careful evaluation for malignancy. This suggests that appendiceal neoplasms, although rare, should be considered in the differential diagnosis of patients presenting with right lower quadrant pain in the emergency department.

Conclusion

Although acute appendicitis is the most common etiology in patients presenting to the emergency department with right lower quadrant pain, appendiceal neoplasms, although rare, may be included in the differential diagnosis. The possibility of malignancy should be considered especially in patients with significant appendiceal dilatation and atypical findings on imaging methods and a family history. Therefore, evaluation of clinical, laboratory and imaging findings together is important for early diagnosis and appropriate treatment planning.

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ADHD Symptoms Among Children Presenting With Trauma To The Pediatric Emergency Department

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Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood and is characterized by significant dysfunction in attention, impulse control, and motor activity levels. Consequently, children may exhibit developmentally inappropriate inattention, hyperactivity, and impulsivity (1,2). Since untreated ADHD may adversely affect a child's psychological, social, and academic

development, early diagnosis and multidisciplinary follow-up are of critical importance. Due to increased impulsivity and risk-taking behaviors, children with ADHD are reported to present more frequently with trauma and emergency department (ED) visits. This suggests that trauma may represent a clinical manifestation of ADHD symptoms (3,4).
This study aimed to evaluate the potential role of ADHD as a risk factor among children aged 5–12 years presenting to the ED with trauma and to investigate the relationship between childhood trauma and ADHD.

Methods

This prospective, cross-sectional study was conducted at a tertiary care center between January 1, 2024 and January 1, 2025. Children aged 5–12 years who presented to the ED due to trauma, whose parents provided written informed consent, and who completed the assessment scales were included. Parents completed the "Turgay DSM-IV Based Disruptive Behavior Disorders Rating Scale-1995." In addition, demographic characteristics, parental education level, trauma history within the previous year, screen exposure, and the child's social relationships were evaluated.

Results

Of the 200 children included, 63% were male, and the mean age was 9.07 years. Attention deficit symptoms were significantly more frequent in children with trauma exposure within the previous year. Hyperactivity rates were significantly higher in children who had lost their fathers and in those with parental psychiatric history. Increasing screen exposure was significantly associated with higher rates of attention deficit.

Discussion And Conclusion

This study demonstrated that ADHD symptoms are clinically relevant among children presenting to the emergency department with trauma (2). A significant relationship was identified particularly between prior trauma exposure, screen time, and ADHD symptoms (3). These findings suggest that early recognition of ADHD symptoms in pediatric trauma patients in the emergency setting may be important for appropriate referral and preventive interventions.

Keywords: Attention-Deficit/Hyperactivity Disorder, Pediatric Emergency, Screen Time, Trauma

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Superior vena cava syndrome due to mediastinal mass presenting with extensive upper extremity thrombosis: a case report

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Introduction and Objective

Superior vena cava (SVC) syndrome is a life-threatening condition caused by obstruction of venous return from the upper body, most commonly due to malignancy. It may present with subtle findings but can rapidly progress to significant morbidity. We aimed to present a case of SVC syndrome associated with a large mediastinal mass and extensive venous thrombosis.

Materials and Methods

A 40-year-old female with no known chronic disease presented to the emergency department with left arm swelling and chest pain. She had a recent history of mediastinal mass biopsy with pending pathology results. Physical examination revealed asymmetric upper extremity swelling, more prominent on the left side.

Results

Electrocardiography showed sinus tachycardia. Laboratory tests revealed elevated inflammatory markers (CRP: 207 mg/L, WBC: 14,000/mm³) with normal troponin levels. Thoracic computed tomography demonstrated a large heterogeneous mediastinal mass (approximately 112 × 59 × 140 mm) extending to the paracardiac region, surrounding the aorta, and causing intermittent compression of the superior vena cava. Bilateral pleural effusions (right dominant, up to 6 cm) and moderate pericardial effusion (up to 28 mm) were

observed. Upper extremity venous Doppler ultrasonography revealed extensive thrombosis involving the left jugular, subclavian, axillary, brachial, ulnar, and radial veins, as well as superficial veins (cephalic and basilic), consistent with deep vein thrombosis and thrombophlebitis. Echocardiography suggested possible metastatic pericardial involvement.

Conclusion

This case highlights that SVC syndrome may present with unilateral upper extremity swelling due to extensive venous thrombosis. In patients with mediastinal masses, early recognition of SVC compression and associated thrombotic complications is critical. Multidisciplinary management including oncology, radiology, and emergency medicine is essential to prevent life-threatening deterioration.

Keywords

Superior vena cava syndrome; mediastinal mass; upper extremity thrombosis; emergency medicine; pericardial effusion

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Distribution of Causative Substances and Treatment Approaches in Intentional Poisonings: A Retrospective Analysis

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ABSTRACT

Introduction: Acute intoxications are among the leading causes of emergency department visits worldwide and are associated with significant morbidity and mortality. In particular, suicidal drug and toxic substance ingestions are frequently encountered clinical conditions in emergency settings that require rapid assessment. Determining the epidemiological and clinical characteristics of these cases is important for developing early diagnosis and appropriate treatment strategies.

Material and Method: In this retrospective study, adult patients who presented to the Emergency Department of Malatya Training and Research Hospital between January 1 and December 31, 2023, due to suicidal drug or toxic substance ingestion were evaluated. Patients were selected from cases recorded under ICD-10 diagnostic code X44 in the hospital information system. Demographic characteristics, type of toxic substance ingested, treatment methods applied, and clinical outcomes were examined. Statistical analyses were performed using IBM SPSS Statistics version 27.0.

Results: Sixty patients meeting the inclusion criteria were evaluated. 86.7% of the patients were female. Admissions were most frequent in winter (38.3%) and autumn (28.3%). The most common causative agents were psychiatric drugs (45%), followed by multiple drug ingestion (15%) and analgesics (15%). Corrosive substance, agricultural pesticide, antibiotic, and alcohol ingestion were identified less frequently. Supportive treatment was applied to 48.3% of patients, while activated charcoal, gastric lavage, N-acetylcysteine, and specific antidote therapies were used in select cases. Fifty patients were followed on general wards and 10 in the intensive care unit; all patients were discharged with full recovery.

Conclusion: Suicidal toxic substance ingestions are common clinical conditions in the emergency department. Early assessment, appropriate supportive care, and careful clinical monitoring play an important role in achieving favorable clinical outcomes.

Keywords: acute intoxication, suicide attempt, emergency department, drug poisoning

Introduction

Acute intoxications are among the leading causes of emergency department visits worldwide and are associated with significant morbidity and mortality. Suicidal drug and toxic substance ingestions in particular are frequently encountered and require rapid evaluation in emergency settings. The clinical presentation in these patients can vary widely depending on the type and dose of the substance ingested and the patient's comorbidities (1,2). Recent studies report that intentional poisoning cases are more common especially in young adults and women, and are most often associated with analgesics, antidepressants, and antipsychotics (3,4). Additionally, a significant proportion of intentional poisoning cases presenting to the ED may develop intensive care requirements and serious complications (5). Therefore, evaluating the epidemiological and clinical characteristics of intoxication cases is important for establishing early diagnosis and appropriate treatment strategies.

Material and Method

Inclusion Criteria: Adult patients (≥18 years of age); patients admitted to the hospital after presenting to the ED with suicidal ingestion of drugs, corrosive substances, or similar agents; patients whose data were retrospectively accessed from the hospital information system and who presented between January 1, 2023 and December 31, 2023.

Exclusion Criteria: Patients under 18 years of age; patients whose data could not be fully accessed in the hospital data system; patients transferred to other hospitals from the ED or who left the hospital against medical advice.

Patient Selection

Data were accessed for 70 patients recorded under ICD-10 diagnostic code X44 (exposure to and accidental poisoning by other and unspecified drugs, medicaments and biological substances) in the Malatya Training and Research Hospital Akgün information system between January 1 and December 31, 2023. The discharge summaries of the patients in the Akgün information system were examined in detail. Ten patients who did not meet the inclusion criteria were excluded. The study was conducted with data from 60 patients who met the criteria.

Statistical Analysis

IBM SPSS Statistics for Windows, version 27.0 (IBM Corp.; Armonk, New York, USA) was used for statistical analyses. Descriptive statistical methods were reported as mean, median, standard deviation, percentage [25–75% (Interquartile Range – IQR)], and frequency.

Results

The study was conducted with 60 patients meeting the inclusion criteria. Fifty-two patients (86.7%) were female.

Twenty-three patients (38.3%) presented in winter, 17 (28.3%) in autumn, 16 (26.7%) in summer, and 4 (6.7%) in spring.

Nine patients (15%) presented due to multiple drug ingestion (analgesics, antibiotics, antihistamines, muscle relaxants, gastroprotective agents, and psychiatric drugs); 9 patients (15%) due to analgesics (NSAIDs and paracetamol); 27 patients (45%) due to psychiatric drugs (antidepressants, antipsychotics); 2 patients (3.3%) due to corrosive substance ingestion; 3 patients (5%) due to agricultural pesticide ingestion; 4 patients (6.7%) due to antibiotics alone; and 1 patient (1.7%) due to alcohol ingestion.

General supportive treatment was applied to 29 patients (48.3%). Activated charcoal was administered to 14 patients (23.3%), and 10 patients received activated charcoal following gastric lavage. Gastric lavage alone was performed in 3 patients, N-acetylcysteine (NAC) was given to 2 patients, and fluid infusion and specific antidote therapy were each administered to one patient.

Fifty patients were followed on internal medicine wards, 10 were admitted to the intensive care unit, and all patients were discharged with full recovery.

Discussion

This study showed that the majority of suicidal drug and toxic substance ingestions presenting to the ED were in female patients, and that psychiatric drugs were the most common causative agent group. Current studies also report that intentional overdose cases are more common especially in young adult women, with antidepressants, antipsychotics, and benzodiazepines among the most frequently implicated agents (6-8).

The study found that general supportive treatment was sufficient for a significant proportion of cases, while activated charcoal, gastric lavage, NAC, and specific antidotes were used in a more limited number of patients. This finding is consistent with current literature, which states that the cornerstone of treatment in acute poisoning consists of airway, respiratory, and circulatory support along with close clinical monitoring, with gastrointestinal decontamination and antidote use providing benefit only in selected cases (9-11). It is particularly emphasized that early administration of activated charcoal in the appropriate patient group can reduce systemic absorption (9,10).

The fact that most of our patients were monitored on general wards, fewer required intensive care, and all were discharged with full recovery suggests the importance of early evaluation and appropriate treatment approaches on prognosis. Indeed, although a proportion of acute intoxications require ICU admission, mortality can remain low with appropriate management (5,12). On the other hand, certain subgroups such as corrosive substance ingestion may follow a more severe clinical course with a notably increased risk of complications (11,12). For this reason, detailed assessment of suicidal toxic ingestions in the ED with respect to causative agent, clinical severity, and treatment requirements is important for appropriate triage and monitoring planning.

Conclusion

Among suicidal toxic substance ingestions presenting to the emergency department in 2023, female patients and psychiatric drugs were predominant. Early assessment and appropriate treatment approaches play an important role in improving patient prognosis.

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Isolated traumatic splenic laceration following blunt abdominal trauma: A case report

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Abstract

In this case report, we aimed to emphasize that conservative management may be an appropriate treatment strategy in a patient with isolated splenic injury whose trauma mechanism was consistent with the radiological findings, in light of the current literature. A patient with a history of falling while skiing was referred to our emergency department for further evaluation and management after a traumatic splenic laceration had been detected during assessment at an outside medical centre. Physical examination revealed tenderness in the lumbar region and pain with tenderness in the left upper quadrant of the abdomen. Based on the clinical findings, the patient was considered hemodynamically stable. Laboratory investigations performed in our hospital revealed no pathological abnormalities except for a decrease in haemoglobin of two units compared with the values measured at the outside centre. Abdominal ultrasonography revealed disruption of the splenic contour adjacent to the left kidney and approximately 3–4 mm of free fluid within the abdominal cavity. Thoracic computed tomography (CT) demonstrated displaced fractures of the left posterior 10th, 11th, and 12th ribs and non-displaced fractures of the left anterior 6th and 7th ribs. Based on the clinical and radiological findings, the patient was diagnosed with an isolated splenic injury. Since the patient was hemodynamically stable, conservative management was planned. After consultation with the departments of general surgery and thoracic surgery, the patient was admitted to emergency medicine clinic's intensive care unit. The patient was managed conservatively and discharged without complications. Hemodynamic stability is the key factor determining the treatment approach in isolated splenic injuries following blunt trauma. In stable patients, nonoperative management can be successfully applied with careful clinical monitoring and serial evaluations.

Keywords: Blunt trauma, Conservative management, Splenic injury

Introduction

Blunt abdominal trauma is an important cause of trauma-related morbidity and mortality, and solid organ injuries are frequently observed, particularly following high-energy mechanisms (1). The spleen is one of the organs most susceptible to blunt abdominal trauma due to its anatomical location and parenchymal structure (2). Current trauma guidelines

recommend conservative management as the standard treatment for splenic injuries in hemodynamically stable patients (3). Additionally, the presence of associated thoracic injuries may provide valuable diagnostic clues for clinicians during the evaluation and management process (4).

In this case report, we aimed to emphasize that conservative management may be an appropriate treatment strategy in a patient with isolated splenic injury whose trauma mechanism was consistent with the radiological findings, in light of the current literature.

Case Report

A patient with a history of falling while skiing was referred to our emergency department for further evaluation and management after a traumatic splenic laceration had been detected during assessment at an outside medical center. At the time of admission to our emergency department, the patient's oxygen saturation was 97%, heart rate was 84 beats/min, and blood pressure was 110/70 mmHg. On physical examination, the Glasgow Coma Scale (GCS) score was 15. The patient was conscious, cooperative, and oriented. Physical examination revealed tenderness in the lumbar region and pain with tenderness in the left upper quadrant of the abdomen. The remaining system examinations were unremarkable. Based on the clinical findings, the patient was considered hemodynamically stable. Laboratory investigations performed in our hospital revealed no pathological abnormalities except for a decrease in hemoglobin of two units compared with the values measured at the outside center. Abdominal ultrasonography revealed disruption of the splenic contour adjacent to the left kidney and approximately 3–4 mm of free fluid within the abdominal cavity. Thoracic computed tomography (CT) demonstrated displaced fractures of the left posterior 10th, 11th, and 12th ribs and non-displaced fractures of the left anterior 6th and 7th ribs. Based on the clinical and radiological findings, the patient was diagnosed with an isolated splenic injury. Since the patient was hemodynamically stable, conservative management was planned. After consultation with the departments of general surgery and thoracic surgery, the patient was admitted to emergency medicine clinic's intensive care unit. The patient was managed conservatively and discharged without complications.

Discussion

Due to its anatomical location and vascular structure, the spleen is the most commonly injured organ in blunt abdominal trauma (1,2). The first step in post-trauma management algorithms is to evaluate hemodynamic stability (5). Although contrast-enhanced computed tomography (CT) is considered the gold standard imaging modality in stable patients, FAST and ultrasonography are important tools for rapid initial evaluation (5,6). The most widely used grading system for splenic injuries is the American Association for the Surgery of Trauma (AAST) organ injury scale (7). In low-grade splenic injuries, the success rate of conservative management exceeds 90% (3,8). The presence of rib fractures, particularly lower

rib fractures, is an important clinical clue suggesting splenic injury (4). In the present case, the presence of posterior lower rib fractures supported the trauma mechanism associated with splenic injury.

Surgical intervention should be considered in cases of hemodynamic instability, persistent decline in hemoglobin, signs of peritonitis, or active contrast extravasation (3,5). In patients without these criteria, conservative management with close monitoring is considered safe (3,8). Although our patient's hemoglobin initially decreased by 2 units, no further decline was observed during follow-up, and surgical intervention was not required.

Conclusion

Hemodynamic stability is the key factor determining the treatment approach in isolated splenic injuries following blunt trauma. In stable patients, nonoperative management can be successfully applied with careful clinical monitoring and serial evaluations.

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Beyond Acute Appendicitis: Crohn's Disease as a Mimicker of Right Lower Quadrant Pain

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Introduction

Acute abdominal pain is one of the most common reasons for emergency department visits. Although acute appendicitis is primarily considered in right lower quadrant pain, inflammatory bowel diseases should also be included in the differential diagnosis. Especially in the early stages, Crohn's disease may present with non-specific clinical and radiological findings (1,2). Normal findings on imaging methods do not rule out the diagnosis, and this situation can create diagnostic difficulties in the emergency department (3). In this case report, we present a patient who was evaluated with an initial diagnosis of acute abdomen but developed findings compatible with inflammatory bowel disease during follow-up.

Case Report

A 35-year-old male patient with a known history of hypertension presented with complaints of abdominal pain and fever. The pain was localized to the right lower quadrant. On physical examination, guarding and rebound tenderness were positive in the right lower quadrant, and his temperature was 38°C. Laboratory evaluation revealed a white blood cell count of 15,000/mm³ and a CRP level of 110 mg/L.

Abdominal USG and CT performed at an external center were evaluated as normal. In the imaging repeated at our clinic, the appendix appeared normal, and no pathological findings were detected except for a few lymph nodes at the level of the cecum (Figure 1). Since acute surgical pathology was not considered by general surgery, the patient was discharged.

Figure 1 Patient's CT Images

The patient re-presented three days later with hematochezia. Following admission, a colonoscopy was performed, revealing widespread aphthous ulcers in the colon (Figure 2). The findings were evaluated as compatible with Crohn's disease or infectious colitis. The patient was discharged with a plan for further investigation and treatment.

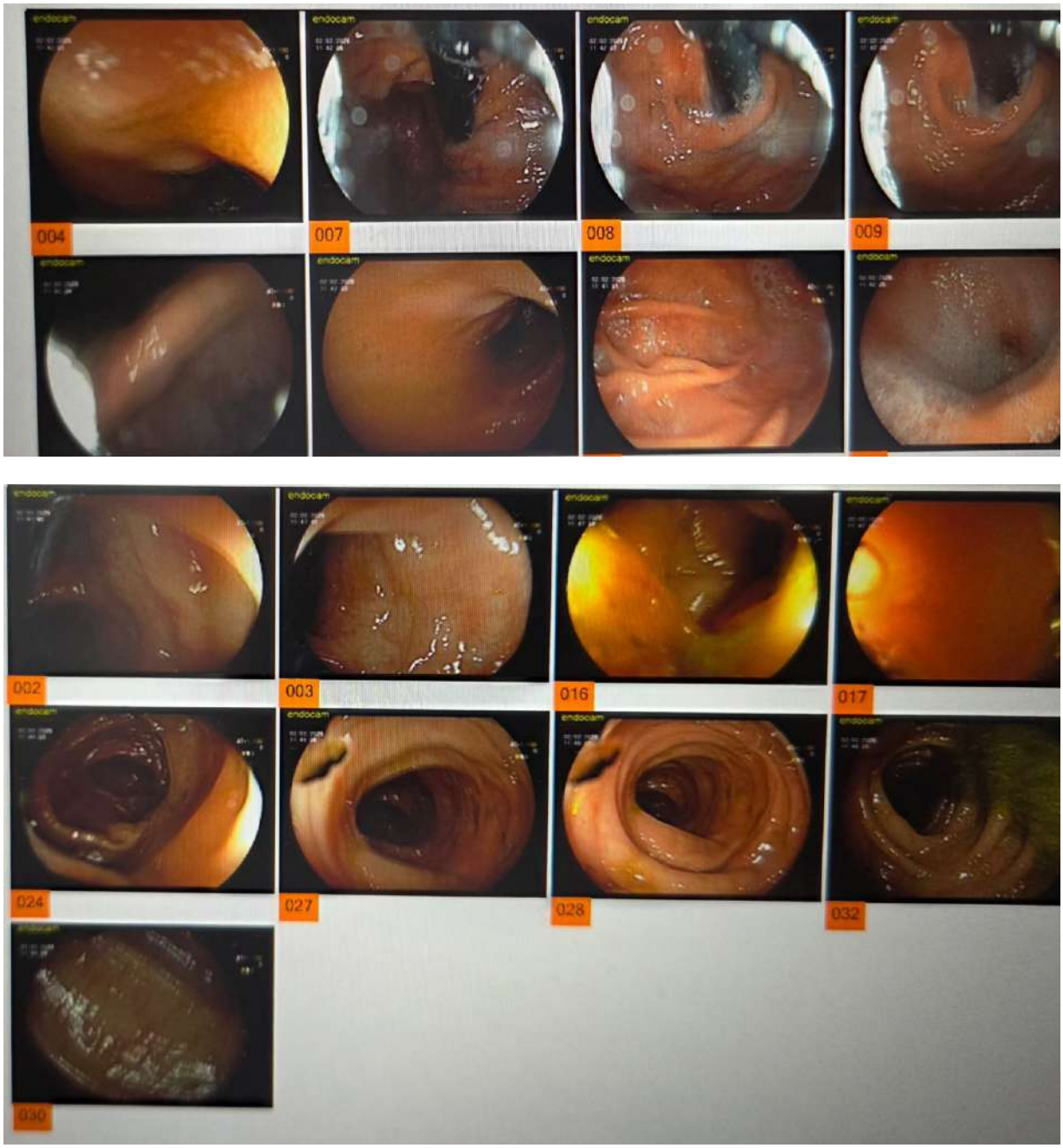

Figure 2 Patient's Colonoscopy Images

Discussion

Crohn's disease is a chronic disease characterized by segmental and transmural inflammation (1). It most commonly involves the terminal ileum and cecum; therefore, it may show clinical similarity to acute appendicitis (2,4). The presence of fever, leukocytosis, and right lower quadrant tenderness can strengthen the initial surgical diagnosis.

In the early stages, CT findings can be minimal; bowel wall thickening or typical mesenteric inflammation findings may not always be observed (3,5). Mesenteric lymphadenopathy may be the sole radiological finding. Elevated CRP is associated with disease activity but is not specific (6).

Aphthous ulcers on endoscopy are among the typical early findings (7). Nevertheless, infectious colitis should be considered in the differential diagnosis and supported by histopathological examination (2).

This case demonstrates that inflammatory bowel diseases should be kept in mind in acute abdomen patients who present with radiologically normal findings but high inflammatory markers.

Conclusion

In patients presenting to the emergency department with right lower quadrant pain and high inflammatory markers, inflammatory bowel diseases should be considered in the differential diagnosis even if imaging methods are normal. Early-stage Crohn's disease may not be radiologically detectable and requires close clinical follow-up.

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Keywords: Crohn's disease, acute abdomen, inflammatory bowel disease

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Concomitant diabetic ketoacidosis and cerebrovascular event: A case report

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ABSTRACT

This case report aims to emphasize that a concomitant cerebrovascular event should be considered in patients with diabetic ketoacidosis (DKA) presenting with altered consciousness. A 69-year-old patient with known diabetes mellitus and hypertension was brought to the emergency department by relatives with complaints of confusion that had started six hours earlier, inability to recognize family members, and markedly elevated capillary blood glucose levels. Laboratory analysis revealed a blood glucose level of 592 mg/dL, potassium level of 5.2 mmol/L, and urine ketone positivity (2+). Arterial blood gas analysis showed a pH of 7.00 and a bicarbonate level of 6.2 mmol/L. Diffusion-weighted magnetic resonance imaging, obtained due to altered consciousness, demonstrated diffusion restriction in the left occipital region consistent with an infarction. Despite appropriate medical management, the patient developed severe metabolic acidosis and respiratory failure due to the coexistence of DKA and acute stroke and died on the seventh day of intensive care unit follow-up. The coexistence of acute ischemic stroke and DKA may have a fatal course, particularly in elderly diabetic patients. In patients diagnosed with DKA, the possibility of a concomitant cerebrovascular event should always be considered when altered consciousness is present.

Keywords: Cerebrovascular event, Diabetic ketoacidosis, Emergency.

INTRODUCTION

In diabetic patients presenting with acute alterations in consciousness, not only metabolic causes but also concomitant cerebrovascular events should be considered. Although the coexistence of diabetic ketoacidosis (DKA) and acute ischemic stroke is rare, it may be associated with a poor clinical outcome. Acute ischemic stroke is frequently observed in elderly diabetic patients, particularly in those with additional vascular risk factors.DKA is a life-threatening acute metabolic complication characterized by hyperglycemia, ketonemia, and metabolic acidosis. According to the American Diabetes Association criteria, the diagnosis is established when blood glucose exceeds 250 mg/dL, arterial pH is below 7.30, and bicarbonate levels are below 18 mEq/L (1). Diabetes mellitus is an independent risk factor for stroke because it accelerates macrovascular atherosclerosis and increases

endothelial dysfunction and inflammatory responses (2). Previous studies have reported that acute stroke may trigger DKA or worsen existing metabolic imbalance (3). Hyperglycemia in patients with acute stroke is associated with poor prognosis. Elevated glucose levels may exacerbate ischemic injury through increased lactic acidosis, enhanced free radical production, and disruption of the blood–brain barrier (4).This case report aims to highlight that a concomitant cerebrovascular event should be considered in patients with DKA who present with altered consciousness.

CASE REPORT

A 69-year-old patient with known diabetes mellitus and hypertension was brought to the emergency department by relatives due to confusion that had begun six hours earlier, inability to recognize family members, and a markedly elevated capillary blood glucose measurement. On admission, vital signs were as follows: body temperature 36.8 °C, heart rate 85 beats/min, blood pressure 150/90 mmHg, respiratory rate 20 breaths/min, and oxygen saturation 92%.Capillary blood glucose at presentation was above the measurable range. The patient's general condition was moderate, and both orientation and cooperation were impaired. Physical examination revealed no muscle strength deficit and no lateralizing neurological signs. Other system examinations were unremarkable. Laboratory investigations showed a blood glucose level of 592 mg/dL and a potassium level of 5.2 mmol/L. Urinalysis revealed ketone positivity (2+). Arterial blood gas analysis demonstrated a pH of 7.00 and a bicarbonate level of 6.2 mmol/L. Due to altered consciousness, diffusion-weighted magnetic resonance imaging (MRI) was performed and demonstrated diffusion restriction in the left occipital region consistent with cerebral infarction. Treatment was initiated promptly, and consultations were obtained from the neurology and endocrinology departments. The patient was admitted to the intensive care unit (ICU).On the second day of ICU follow-up, the acidosis improved. Carotid Doppler ultrasonography and echocardiography revealed no pathological findings.On the sixth day of ICU follow-up, elevated CO_2 levels were detected in arterial blood gas analysis. The patient was initially placed on non-invasive mechanical ventilation; however, due to intolerance, endotracheal intubation was required. Despite intensive treatment, the patient died on the seventh day of ICU follow-up.

DISCUSSION

DKA is a life-threatening acute metabolic complication characterized by hyperglycemia, ketonemia, and metabolic acidosis. According to the American Diabetes Association criteria, a blood glucose level >250 mg/dL, arterial pH <7.30, and bicarbonate level <18 mEq/L are diagnostic (1). In our case, the patient presented with a blood glucose level of 592 mg/dL, pH of 7.00, and bicarbonate level of 6.2 mEq/L, indicating severe DKA.

Diabetes mellitus is an independent risk factor for acute ischemic stroke, and hyperglycemia during stroke is associated with poor prognosis (2,4). Previous studies have

reported that acute stress conditions may trigger DKA, while DKA itself may aggravate thrombotic processes through dehydration and hypercoagulability (3).During DKA, increased levels of pro-inflammatory cytokines and activation of the coagulation cascade, together with hemoconcentration, may increase thrombotic risk and contribute to the development of cerebrovascular events (6). In our case, diffusion-weighted MRI demonstrated infarction in the left occipital region, indicating that the altered consciousness was not solely due to metabolic causes but also associated with a cerebrovascular event.

The mainstay of DKA treatment includes fluid replacement, insulin infusion, and correction of electrolyte imbalance (1). The role of bicarbonate therapy in severe acidosis is limited, and routine use has not been shown to reduce mortality (5). In our patient, despite appropriate treatment in accordance with the literature, the coexistence of severe metabolic acidosis and respiratory failure due to DKA and acute stroke resulted in death on the seventh day of ICU follow-up.

CONCLUSION

The coexistence of acute ischemic stroke and DKA may have a fatal course, particularly in elderly diabetic patients. In patients diagnosed with DKA, the possibility of a concomitant cerebrovascular event should be considered when altered consciousness is present.

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Sick Sinus Syndrome Presenting With Recurrent Symptomatic Bradycardia: A Case Report

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Abstract

In this report, we aimed to describe the diagnostic and management processes of sick sinus syndrome in a patient presenting with recurrent bradycardia. A 41-year-old male patient presented to the emergency department complaining of sweating and the sensation of a slowed pulse. The patient had no known comorbidities. His medical history revealed that he had presented to another medical centre with similar complaints approximately two months earlier and had undergone coronary angiography, which demonstrated no significant coronary artery stenosis. The patient reported using acetylsalicylic acid. At presentation to the emergency department, his vital signs were as follows: oxygen saturation 97%, heart rate 61 beats/min, temperature 36.3°C, and blood glucose level 107 mg/dL. Electrocardiography demonstrated sinus rhythm. A detailed medical history revealed that the patient had presented to different emergency departments several times with similar complaints and had been discharged after evaluation revealed no acute pathology. The patient was placed under monitoring and laboratory tests were ordered. During monitoring, a sudden decrease in heart rate to approximately 20 beats/min was observed. During the bradycardia episodes, the patient exhibited marked somnolence and confusion. Based on the clinical findings and monitoring results, the preliminary diagnoses of sinus bradycardia and sick sinus syndrome were considered. The patient was referred to an external centre for further evaluation and assessment of the need for permanent pacemaker implantation. Laboratory test results that became available after the patient's referral showed that electrolyte levels and cardiac biomarkers, including troponin, were within normal limits. Sick sinus syndrome should be considered in the differential diagnosis of patients presenting with recurrent symptomatic bradycardia. Permanent pacemaker therapy significantly improves the prognosis in appropriately selected patients.

Keywords: Bradycardia, Pacemaker, Sick sinus syndrome.

Introduction

Sick sinus syndrome (SSS) is a clinical condition characterised by impairment of the heart's physiological pacemaker activity due to dysfunction of the sinoatrial node (1). Although it most commonly develops secondary to idiopathic degenerative fibrosis, it may also occur as a consequence of ischaemic heart disease, infiltrative diseases, or medication use (1).

Clinical manifestations encompass a broad spectrum and may include syncope, dizziness, fatigue, exercise intolerance, and altered consciousness (2). Intermittent rhythm disturbances may complicate the diagnostic process and often necessitate prolonged rhythm monitoring (3).

In this report, we aimed to describe the diagnostic and management processes of sick sinus syndrome in a patient presenting with recurrent bradycardia.

Case Presentation

A 41-year-old male patient presented to the emergency department complaining of sweating and the sensation of a slowed pulse. The patient had no known comorbidities. His medical history revealed that he had presented to another medical centre with similar complaints approximately two months earlier and had undergone coronary angiography, which demonstrated no significant coronary artery stenosis. The patient reported using acetylsalicylic acid. At presentation to the emergency department, his vital signs were as follows: oxygen saturation 97%, heart rate 61 beats/min, temperature 36.3°C, and blood glucose level 107 mg/dL. His Glasgow Coma Scale score was 15, and he was oriented and cooperative. Electrocardiography demonstrated sinus rhythm. A detailed medical history revealed that the patient had presented to different emergency departments several times with similar complaints and had been discharged after evaluation revealed no acute pathology. The patient was placed under monitoring and laboratory tests were ordered. During monitoring, a sudden decrease in heart rate to approximately 20 beats/min was observed. An ECG recording was attempted during this episode; however, the bradycardia attacks were very brief and resolved spontaneously, preventing electrocardiographic documentation of the episode.During the bradycardia episodes, the patient exhibited marked somnolence and confusion. Based on the clinical findings and monitoring results, the preliminary diagnoses of sinus bradycardia and sick sinus syndrome were considered. The patient was referred to an external centre for further evaluation and assessment of the need for permanent pacemaker implantation.

Laboratory test results that became available after the patient's referral showed that electrolyte levels and cardiac biomarkers, including troponin, were within normal limits.

Discussion

Although sick sinus syndrome is commonly considered a disease of older adults, it may also occur in younger patients (2). Diagnosis may be challenging due to intermittent bradycardia episodes and may require advanced rhythm monitoring methods, such as Holter monitoring (3).

The presence of symptomatic bradycardia constitutes an important indication for permanent pacemaker implantation, even in the absence of syncope (4). Current guidelines recommend permanent pacing therapy in patients in whom bradycardia is documented or strongly correlated with symptoms (4).

In this case, secondary causes were excluded because electrolyte levels were within normal limits and no markers of myocardial injury were detected. Therefore, primary sinoatrial node dysfunction was considered the most likely diagnosis. The inability to document rhythm disturbance during short-term monitoring in the emergency department does not exclude the diagnosis, and further evaluation is warranted when clinical suspicion persists (3).

Conclusion

Sick sinus syndrome should be considered in the differential diagnosis of patients presenting with recurrent symptomatic bradycardia. Permanent pacemaker therapy significantly improves the prognosis in appropriately selected patients.

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Cardiac Arrest Across Three Settings: Duration of Cardiopulmonary Resuscitation and Clinical Outcomes in Home, Ambulance, and In-Hospital Cases

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Abstract

Cardiac arrest is a critical clinical condition that develops as a result of the sudden loss of the heart's effective mechanical activity and rapidly results in death if not intervened immediately. Current guidelines recommend maintaining advanced life support at the scene rather than transferring the patient. In this study, the clinical outcomes of cardiopulmonary resuscitation (CPR) and advanced life support applied in different settings (home, ambulance, hospital) and the factors affecting the return of spontaneous circulation (ROSC) were investigated.

The median age of the patients included in the multicenter prospective study was 73 (63.75–86.25), and the most common comorbidity was hypertension (52.2%). While 84.8% of the cases were witnessed arrests, the rate of bystander CPR remained at 30.4%. Of the arrests, 56.5% were due to cardiac etiology, 58.7% occurred at home, and a non-shockable rhythm (asystole/PEA) was detected in 62.2%. The median CPR duration was recorded as 48.5 minutes, and the total adrenaline dose as 10 mg. ROSC was achieved in 45.7% of the patients; 30-day mortality was determined as 63.2%, and in-hospital mortality as 70.7%.

In statistical analyses, CPR duration ($p < 0.001$), total adrenaline dose ($p = 0.003$), cardiac etiology ($p < 0.001$), history of coronary artery disease ($p = 0.004$), and systolic/diastolic

blood pressure values at admission were found to be significantly associated with ROSC success. However, no statistically significant effect of arrest location ($p = 0.196$) and the presence of ROSC on 30-day mortality was detected. High mortality rates despite successful resuscitation reflect the difficulties in managing post-resuscitation complications such as multiple organ failure.

Cardiac etiology, history of coronary artery disease, and stable hemodynamic parameters at admission stand out as the strongest predictive factors for ROSC. Despite the high rate of witnessed arrests, the low rate of bystander CPR emphasizes the need to expand basic life support training at the community level. Early intervention and targeted post-resuscitation care are critical in reducing mortality and improving neurological outcomes.

Introduction

Cardiac arrest is a clinical condition characterized by loss of consciousness, pulselessness, and apnea resulting from the sudden loss of the heart's effective mechanical activity, which rapidly leads to death if not intervened urgently. Cardiopulmonary resuscitation (CPR) is applied in its treatment, and with successful intervention, the return of spontaneous circulation (ROSC) can be achieved. Current guidelines recommend maintaining advanced life support at the scene during CPR rather than transferring the patient, as the quality of CPR may decrease and interruptions may occur during the transfer process. Therefore, studies evaluating the effect of the setting in which CPR is performed on clinical outcomes are needed. This study aimed to compare the clinical outcomes of CPR and advanced life support performed in home, ambulance, and hospital settings; additionally, subgroups were analyzed in terms of neurological recovery and prognosis.

Methods

This study was designed as a prospective, multicenter, observational cohort. All adult cases aged ≥18 years who developed in-hospital or out-of-hospital cardiac arrest and underwent CPR were included; traumatic arrests, pregnant women, and patients with missing data were excluded. The diagnosis of arrest was made based on loss of consciousness, apnea/agonal breathing, and pulselessness. CPR duration was defined as the time from the first chest compression to sustainable ROSC. Demographic, clinical, and resuscitation data (initial rhythm, defibrillation, intubation, adrenaline, witnessed arrest, and bystander CPR) were prospectively recorded using a standard form. The primary outcome was determined as 30-day survival, and the secondary outcome as neurological status at discharge (CPC). Data were compared according to the type of setting (home, ambulance, hospital). In statistical analyses, appropriate parametric/non-parametric tests, ROC analysis, Kaplan–Meier, and multivariate logistic regression were used.

Results

The median age of the patients included in the study was 73 (63.75–86.25), and the most common comorbidity was hypertension with 52.2% (n=24). While 84.8% (n=39) of the cases were witnessed arrests, bystander CPR was performed on 30.4% (n=14) of them. Of the arrests, 56.5% were based on cardiac etiology and 58.7% (n=27) occurred in the home setting. In the initial rhythm analysis, a non-shockable rhythm (asystole/PEA) was detected at a rate of 62.2%.

During the intervention process, the median total CPR duration was recorded as 48.5 minutes, and the total administered adrenaline dose was 10 mg. Return of spontaneous circulation (ROSC) was achieved in 45.7% (n=21) of the patients, and ROSC was sustained for more than 20 minutes in 40% (n=16) of the patients. The 30-day mortality rate was determined as 63.2%, and in-hospital mortality as 70.7%.

In statistical analyses; total CPR duration ($p < 0.001$), total adrenaline dose ($p = 0.003$), cardiac etiology ($p < 0.001$), and history of coronary artery disease ($p = 0.004$) were found to be significantly associated with ROSC success. Furthermore, systolic ($p = 0.009$) and diastolic ($p = 0.008$) blood pressures at admission showed a significant difference in the group that achieved ROSC. Conversely, no statistically significant effect of arrest location ($p = 0.196$) and the presence of ROSC on 30-day mortality ($p = 0.943$) was detected.

Discussion

This study examined the factors affecting the return of spontaneous circulation (ROSC) and survival in cardiac arrest cases through a multicenter and prospective approach. The median age of the patients in our study was found to be 73, which is consistent with the 64-79 age range reported in the ILCOR global report. Although the vast majority of our cases (84.8%) were witnessed arrests, offering an advantage in terms of intervention speed, it is noteworthy that the rate of basic life support provided by bystanders (bystander CPR) remained at the level of 30.4%. It is known in the literature that bystander CPR rates vary between 19.1% and 79.0% depending on the region and that this intervention is of critical importance for survival.

When arrest etiology was examined, it was observed that 56.5% of the cases were due to cardiac causes and that cardiac etiology was statistically significantly associated with ROSC success ($p < 0.001$). The ERC 2025 guidelines emphasize that coronary artery disease is responsible for 80% of sudden cardiac deaths. In our study, the presence of a history of coronary artery disease also emerged as a factor significantly affecting ROSC success ($p = 0.004$). In the initial rhythm analysis, a non-shockable rhythm (asystole/PEA) was detected in 62.2% of the patients. While the ERC 2025 guidelines emphasize the importance of early adrenaline administration to increase survival in non-shockable rhythms, the significantly lower total adrenaline dose administered in the group achieving ROSC ($p = 0.003$) in our study supports the fact that circulation returns in earlier stages and prolonged CPR durations negatively affect prognosis.

In the group achieving ROSC, systolic (p = 0.009) and diastolic (p = 0.008) blood pressures and heart rate (p = 0.009) at admission were found to be significantly higher. These findings demonstrate the determining role of physiological reserve and perfusion pressure at the time of arrest in the return of circulation. According to ILCOR data, 30-day survival rates in out-of-hospital cardiac arrests vary between 3.1% and 20.4%. Despite the initial ROSC rate of 45.7% achieved in our study, the fact that 30-day mortality occurred at 63.2% demonstrates the difficulties of the post-resuscitation care process and the management of complications such as multiple organ failure (33.3%) on survival.

Conclusion

The results of our study revealed that cardiac etiology, history of coronary artery disease, and stable hemodynamic parameters at admission are the strongest predictive factors for ROSC success in cardiac arrest cases. The shortness of CPR duration and the low total adrenaline dose required are the primary indicators of successful resuscitation. Despite the high rate of witnessed arrests, the fact that bystander CPR rates are not at the desired level indicates the need to disseminate basic life support training throughout the general public. In conclusion, early intervention and targeted post-resuscitation care are critical for reducing high mortality rates and improving neurological outcomes.

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Mesalazine-Associated Recurrent Hemorrhagic Pericarditis and Cardiac Tamponade: An Immune-Mediated Drug Adverse Effect

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Abstract

Introduction

Mesalazine (5-aminosalicylate) is a commonly used agent in the treatment of inflammatory bowel diseases and is generally considered safe (1,6). However, in rare cases, immune-mediated cardiac adverse effects may develop (2–5). These complications may present as pericarditis, pericardial effusion, and rarely cardiac tamponade (3–5).

Case

A 24-year-old male patient with a diagnosis of ulcerative colitis presented to the emergency department with increasing dyspnea and chest pain. His history revealed a previous episode of

pericardial effusion during mesalazine therapy two years prior. Bedside transthoracic echocardiography performed in the emergency department demonstrated massive pericardial effusion, and emergency pericardiocentesis was performed due to suspected cardiac tamponade. A total of 570 mL of hemorrhagic fluid was drained. Pericardial fluid analysis revealed an exudative nature according to Light's criteria. Microbiological investigations showed no growth. Clinical improvement following discontinuation of mesalazine, along with the development of peripheral eosinophilia, supported the diagnosis of mesalazine-associated immune-mediated recurrent pericarditis.

Conclusion

Although rare, mesalazine may lead to serious cardiac complications. In patients with ulcerative colitis presenting with pericardial effusion and tamponade, drug-related cardiotoxicity should always be considered in the differential diagnosis.

Keywords

Mesalazine, Pericarditis, Pericardial effusion, Cardiac tamponade, Ulcerative colitis

Introduction

5-aminosalicylate (5-ASA) derivatives are anti-inflammatory agents widely used in the treatment of ulcerative colitis (6). Mesalazine is one of the most commonly used drugs in this group and is generally well tolerated (6). However, in rare cases, serious systemic adverse effects may occur (2–5). These adverse effects include pulmonary, renal, pancreatic involvement, as well as cardiac complications (2–5).

Among mesalazine-associated cardiac complications, pericarditis, myocarditis, and myopericarditis have been reported (2–5). Most of these conditions are thought to result from immune-mediated hypersensitivity mechanisms (3,13).

Although the clinical presentation usually occurs within the first weeks after initiation of the drug, in some cases it may develop months or even years later (5,7).

The development of massive pericardial effusion leading to cardiac tamponade is extremely rare (3–5). Additionally, hemorrhagic pericardial effusion has been reported in only a limited number of cases in the literature (11).

In this report, a case of recurrent hemorrhagic pericarditis and cardiac tamponade associated with mesalazine use is presented.

Case Presentation

A 24-year-old male patient presented to the emergency department with progressively worsening dyspnea and chest pain over the past week. His medical history revealed a

diagnosis of ulcerative colitis and a prior episode of pericardial effusion during mesalazine therapy two years earlier. That episode had resolved with medical treatment. At the time of the current presentation, his ulcerative colitis was in clinical remission, and he had no active gastrointestinal symptoms.

On physical examination, heart rate was 123 beats/min, blood pressure 99/64 mmHg, temperature 36°C, and oxygen saturation 100%. Cardiac auscultation revealed muffled heart sounds. Jugular venous distension and pulsus paradoxus were not observed. Respiratory examination showed decreased breath sounds at the lung bases.

Laboratory investigations revealed hemoglobin 9.5 g/dL, platelet count 257,000/mm³, C-reactive protein 173 mg/L, and erythrocyte sedimentation rate 84 mm/h. High-sensitivity troponin I level was <2.5 ng/L. Coagulation parameters were INR 1.28, PT 14.8 seconds, and aPTT 36.9 seconds.

Bedside transthoracic echocardiography in the emergency department showed a left ventricular ejection fraction of 55% and a massive circumferential pericardial effusion. Findings of right atrial collapse were present, consistent with cardiac tamponade.

Emergency pericardiocentesis was performed, and a total of 570 mL of hemorrhagic pericardial fluid was drained. The patient was subsequently admitted to the coronary intensive care unit for close hemodynamic monitoring.

Pericardial fluid analysis showed a leukocyte count of 2945 cells/µL with polymorphonuclear predominance (67.8%) and an erythrocyte count exceeding 40,000/mm³. Biochemical analysis revealed protein 56 g/L, albumin 31.1 g/L, and LDH 991 U/L. Serum total protein was 64.8 g/L and serum LDH was 205 U/L. According to Light's criteria, the pericardial fluid was exudative (protein ratio 0.86, LDH ratio 4.83).

Microbiological studies, including bacterial and fungal cultures, showed no growth. Direct microscopic examination revealed no microorganisms. Cytological evaluation was negative for malignancy.

Post-procedural hemoglobin level was 8.8 g/dL.

Mesalazine therapy was discontinued, and anti-inflammatory treatment with colchicine was initiated. During follow-up, significant improvement was observed in chest pain and dyspnea, along with a decrease in inflammatory markers. After achieving clinical stabilization, the patient was discharged with outpatient follow-up.

Discussion

Mesalazine-associated cardiac toxicity is rare but clinically significant (2–5). Cases of pericarditis and myocarditis associated with mesalazine use have been reported in the

literature (2–5). Most of these complications are thought to be related to immune-mediated hypersensitivity reactions (3,13).

In the present case, the development of massive hemorrhagic pericardial effusion and cardiac tamponade is noteworthy. The high protein and LDH content of the pericardial fluid supports an inflammatory process. The exudative nature according to Light's criteria further supports this finding (9,12).

The hemorrhagic nature of the pericardial fluid necessitates consideration of differential diagnoses such as malignancy, trauma, tuberculosis, and coagulopathy (11,12).

The presence of a prior episode of pericardial effusion during mesalazine therapy and the recurrence of a more severe clinical presentation following re-exposure suggest the "rechallenge phenomenon." This is considered strong evidence for causality in drug-related adverse reactions (14).

Although the exact pathophysiology of mesalazine-associated pericardial involvement is not fully understood, type I and type IV hypersensitivity reactions, eosinophilic inflammation, and immune complex-mediated inflammation have been proposed (3,13).

The cornerstone of management is discontinuation of the offending drug (2,3). In most cases, rapid clinical improvement is observed following drug withdrawal and anti-inflammatory therapy (8,9). In patients with massive pericardial effusion and cardiac tamponade, pericardiocentesis is life-saving (8,10).

Conclusion

Although rare, mesalazine may cause serious cardiac complications such as recurrent hemorrhagic pericarditis and cardiac tamponade (2–5). Drug-related cardiotoxicity should always be considered in cases of unexplained pericardial effusion in patients with ulcerative colitis. Early diagnosis, discontinuation of the offending agent, and appropriate treatment can prevent severe complications (8).

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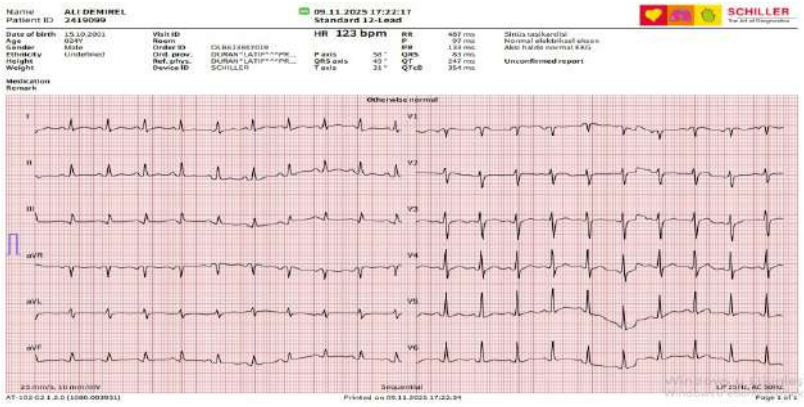

Figure 1 Electrocardiogram (ECG)

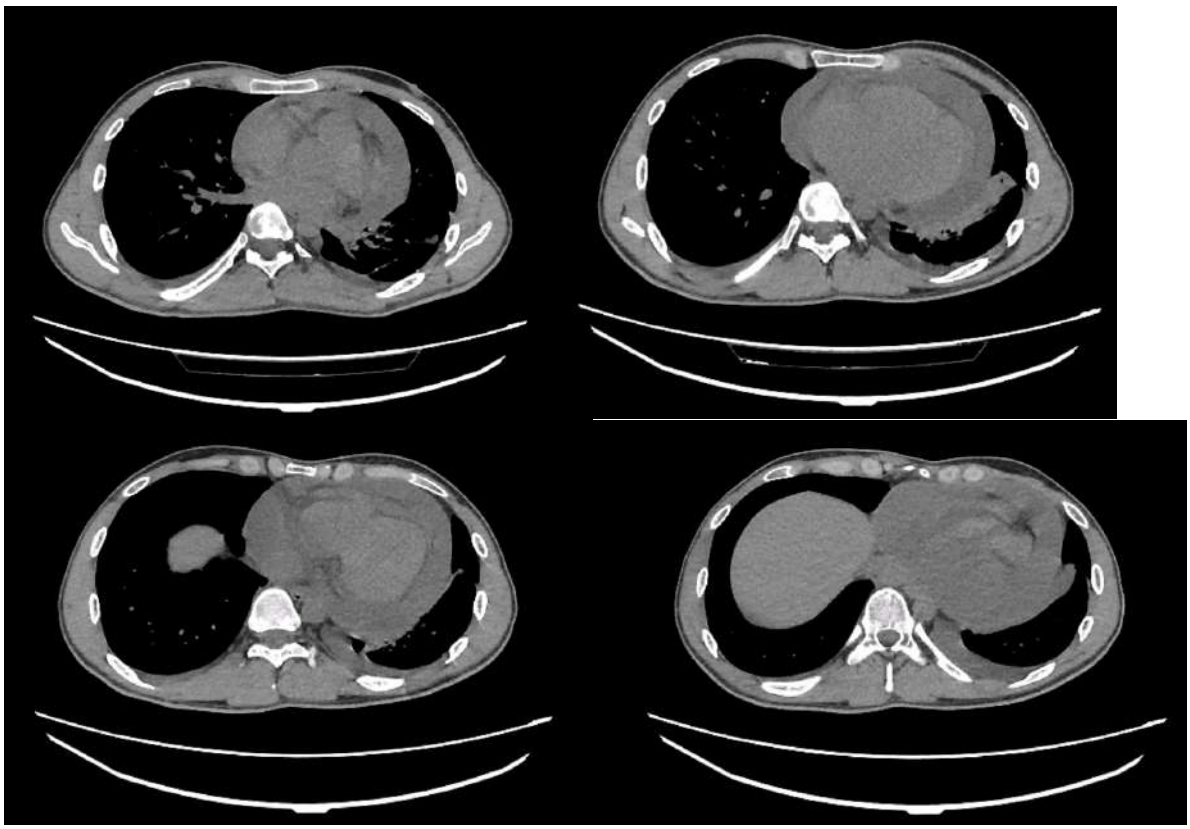

Figure 2A–D

8376

Cyanide intoxication: Three case reports

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Abstract

In this case series, we aim to present three patients who were exposed to cyanide gas following a fire at a recycling facility. Three men, aged 18, 19, and 21, presented to our emergency department after being exposed to cyanide gas and carbon monoxide following a fire at a recycling facility. All three reported noticing a strange odour. Decontamination was performed in the Chemical, Biological, Radiological, and Nuclear (CBRN) unit. All patients were admitted to the toxicology intensive care unit. They received supportive treatment and hydroxocobalamin as an antidote. After 48 hours of observation, all patients, whose symptoms had subsided, were discharged in good health. It should be remembered that in patients presenting with hemodynamic instability and arrhythmias after fire/smoke inhalation, early decontamination, intensive supportive care, and early administration of hydroxocobalamin in appropriately selected patients, along with a high clinical suspicion, are crucial in the treatment of cyanide poisoning.

Keywords: Cyanide; Hydroxocobalamin; Intoxication.

Introduction

Cyanide is a chemical with high toxicity, naturally present in many plants and widely used in industrial processes (1). It is among the fastest-acting lethal toxins known and exerts its effects primarily through mitochondrial injury (2). Cyanide poisoning may occur via oral ingestion, inhalation, or dermal contamination (3). Clinical manifestations of acute cyanide toxicity vary according to the route and dose of exposure; however, patients commonly present with gastrointestinal symptoms such as nausea and vomiting, as well as non-specific neurologic symptoms including headache, dizziness, and weakness (4). Following inhalational exposure, patients may perceive an odor similar to bitter almonds (5). This is typically associated with inhalation of cyanide gas generated by combustion of certain materials during fires (6).

In this case series, we aim to present three patients who were exposed to cyanide gas following a fire at a recycling facility.

Case Reports

Case 1

An 18-year-old man presented to our emergency department following exposure to cyanide gas and carbon monoxide (CO) after a fire at a recycling facility. He reported that he initially did not notice any unusual odor; however, after re-entering the facility to extinguish the fire, he perceived an unfamiliar smell that he could not describe and subsequently developed nausea and vomiting. On arrival, vital signs were: blood pressure 70/40 mmHg, heart rate 63 beats/min, respiratory rate 22/min, temperature 36.6°C, and oxygen saturation 96% on room air. He was in good general condition, alert, oriented, and cooperative. Electrocardiography (ECG) showed sinus arrhythmia (Figure 1). Physical examination was otherwise unremarkable. Decontamination was performed in the CBRN (chemical, biological, radiological, and nuclear) unit, and symptomatic treatment was initiated for nausea/vomiting and hypotension. After consultation with the national poison center (114), hydroxocobalamin 5 mg was requested as an antidote due to hypotension. The patient was admitted to the toxicology intensive care unit (ICU).

During ICU follow-up, the patient developed lactic acidosis and sinus bradycardia/arrhythmia. After administration of hydroxocobalamin 5 mg, arterial blood gas parameters improved and lactate levels returned to the normal range. Hypotension, arrhythmia, and bradycardia resolved, and the patient was discharged in good condition after 48 hours of observation (Figure 2).

Case 2

A 19-year-old man presented after exposure to cyanide gas and CO due to the same recycling facility fire. He stated that he noticed an odor resembling bitter almonds at the scene, followed by dizziness, nausea, and headache. On admission, vital signs were: blood pressure 90/60 mmHg, heart rate 52 beats/min, blood glucose 85 mg/dL, oxygen saturation 96% on room air, respiratory rate 16/min, and temperature 36.6°C. The patient had a Glasgow Coma Scale (GCS) score of 15, and was alert, oriented, and cooperative. ECG showed normal sinus rhythm (Figure 3). Physical examination was unremarkable. Decontamination was performed in the CBRN unit, and symptomatic treatment was initiated for nausea/vomiting and hypotension.

During ICU monitoring, lactic acidosis and sinus bradycardia/arrhythmia developed (Table 1). Blood gas parameters improved and lactate levels normalized following hydroxocobalamin 5 mgtherapy. Due to refractory hypotension and persistent nausea/vomiting, repeat hydroxocobalamin 5 mg was administered in addition to supportive

care. After infusion, hypotension, arrhythmia, and bradycardia resolved, and the patient was discharged after 48 hours of follow-up (Figure 4).

Case 3

A 21-year-old man presented following exposure to cyanide gas and CO after the same fire. He reported perceiving a bitter almond–like odor at the scene and subsequently developed dizziness and cold sweating. On admission, vital signs were: blood pressure 80/40 mmHg, heart rate 53 beats/min, blood glucose 65 mg/dL, oxygen saturation 92% on room air, respiratory rate 16/min, and temperature 36.6°C. The patient had a GCS score of 14 and was somnolent but alert, oriented, and cooperative on examination. ECG showed sinus arrhythmia. The patient had a strong, acrid soot odor and visible contamination; therefore, decontamination was performed in the CBRN unit. Physical examination revealed no abnormalities. Oxygen supplementation was provided, and 5% dextrose intravenous infusion was initiated for hypoglycemia. After consultation with the national poison center (114), hydroxocobalamin 5 mg was requested as an antidote due to hypotension and arrhythmia. The patient was admitted to the toxicology ICU.

During ICU follow-up, the patient developed lactic acidosis and sinus bradycardia/arrhythmia (Table 1). Arterial blood gas parameters improved after hydroxocobalamin 5 mg antidotal therapy, and lactate levels normalized (Figure 5). Given refractory hypotension and hypoglycemia, repeat hydroxocobalamin 5 mg was administered alongside supportive care. Following infusion, hypoglycemia and arrhythmia resolved, and the patient was discharged after 48 hours of observation (Figure 6).

Discussion

Cyanide is a rapidly acting toxin that may be encountered together with carbon monoxide (CO) in cases of fire and smoke inhalation, especially in environments where synthetic materials burn. Interpretation of blood cyanide concentrations can be challenging due to issues related to tissue/blood distribution, endogenous production and elimination, and analytical limitations. Therefore, in practice, diagnosis is often based on exposure history and clinical findings, and treatment is frequently initiated without waiting for confirmatory tests (1).

Similarly, in our cases, the diagnosis was established based on exposure history and the clinical picture, and treatment was started before blood cyanide levels were available

Pathophysiologically, cyanide toxicity primarily targets the mitochondria by inhibiting cytochrome c oxidase, thereby impairing cellular oxygen utilization and causing histotoxic hypoxia, rapid energy failure, and prominent cardiovascular and central nervous system involvement. Although clinical manifestations vary by exposure route and dose, presentations may range widely and include nausea and vomiting, altered mental status, seizures, lactic

acidosis, and abnormal vital signs. Supportive care remains the cornerstone of management, with antidotal therapy added in appropriate cases (2).

In our series, sinus bradycardia, refractory hypotension, and lactic acidosis were observed, and complete clinical recovery occurred following hydroxocobalamin antidotal treatment.

Among available antidotes, hydroxocobalamin has become a prominent option, as its cobalt-containing structure binds cyanide to form cyanocobalamin, facilitating detoxification. Its pharmacokinetic and pharmacodynamic properties support its clinical utility in cyanide poisoning. Nonetheless, supportive care remains fundamental, and antidote availability may vary by region and clinical context (3).

In our cases, direct communication with the national poison center (114) enabled procurement of the antidote from regional centers, and clinical improvement was observed following administration.

In the literature, survival in acute cyanide intoxication is often associated with early recognition and rapid treatment, whereas delays may lead to poor outcomes. Case reports have also highlighted the importance of aggressive supportive care, even during the process of obtaining antidotal therapy, in patients presenting with severe shock and metabolic acidosis (e.g., ingestion-related cases) (4,5).

In our series, the achievement of hemodynamic and rhythm stabilization after hydroxocobalamin and supportive therapy, together with early discharge and absence of mortality.

The cyanide toxicity might be addressed not only through direct binding/chelating strategies but also through broader metabolic approaches. These perspectives are based on the metabolic shift induced by cyanide from oxidative phosphorylation toward glycolysis and propose interventions at the level of intermediary metabolism. Although such strategies are not yet part of routine clinical practice, they may, in the future, provide adjunctive options alongside standard antidotes (6,7).

Conclusion

It should be remembered that in patients presenting with hemodynamic instability and arrhythmias after fire/smoke inhalation, early decontamination, intensive supportive care, and early administration of hydroxocobalamin in appropriately selected patients, along with a high clinical suspicion, are crucial in the treatment of cyanide poisoning.

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Figure 1. Admission electrocardiogram (ECG) of Case 1.

Figure 2. Discharge electrocardiogram (ECG) of Case 1.

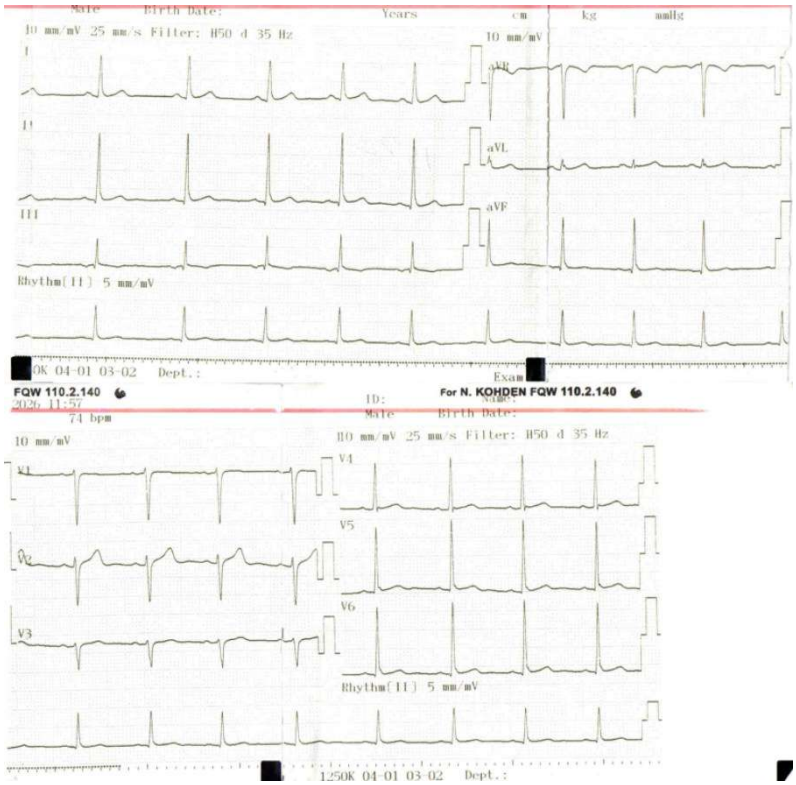


Figure 3. Admission electrocardiogram (ECG) of Case 2.

Figure 4. Discharge electrocardiogram (ECG) of Case 2.

Figure 5. Admission electrocardiogram (ECG) of Case 3.

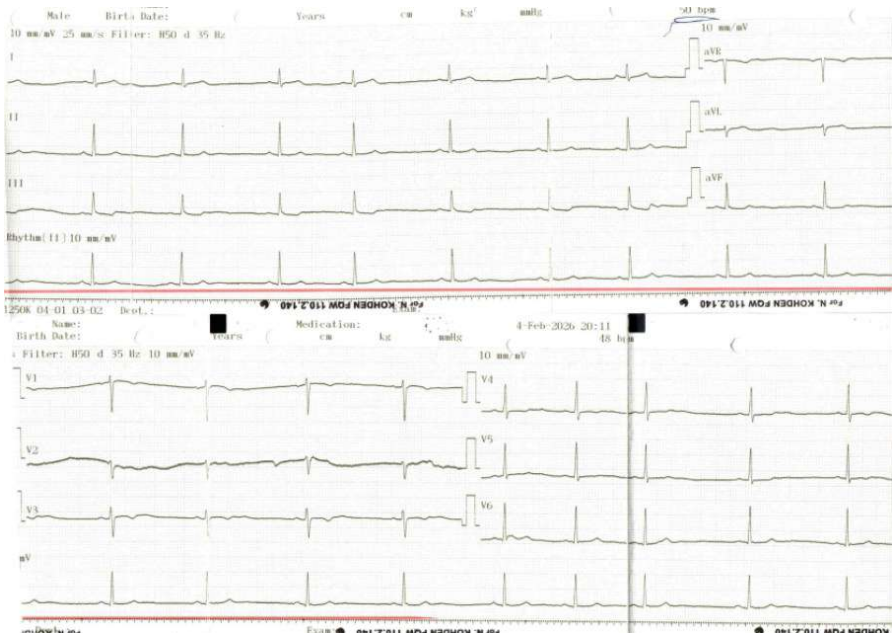
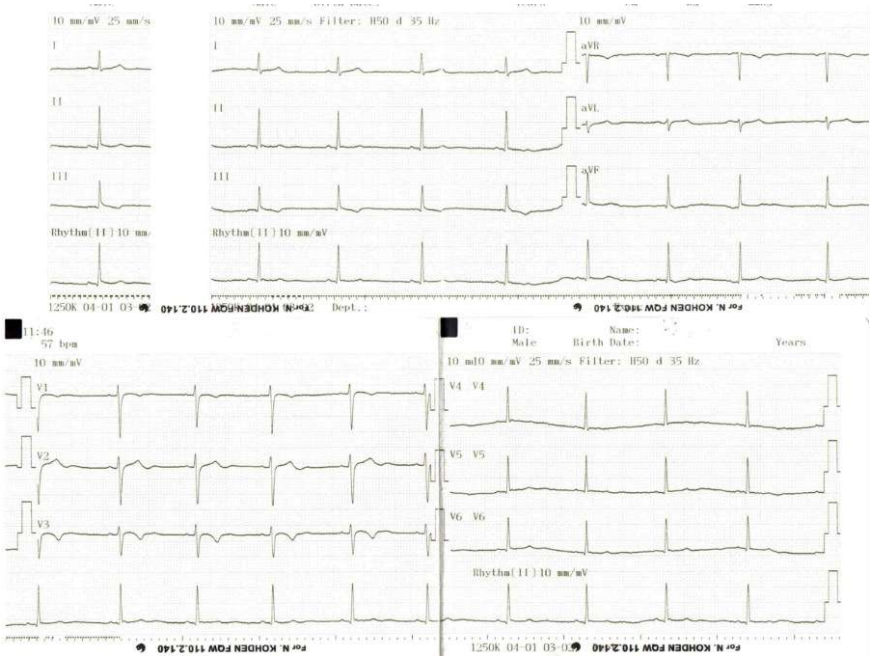


Figure 6. Discharge electrocardiogram (ECG) of Case 3.



8438

Systemic Botulinum Toxin Toxicity After Endoscopic Intragastric Injection for Weight Loss

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Introduction

Botulinum toxin, the most potent biological toxin known, has become an important therapeutic agent in a wide range of clinical applications (1). It is increasingly used as a minimally invasive alternative for weight loss in patients with obesity. Although most adverse effects are mild and localized, systemic effects such as generalized weakness distant from the injection site have been reported (2). While botulinum toxin type A (BTX-A) therapy is generally considered safe and well tolerated, its short-term efficacy in obesity remains variable (3). This case aims to highlight a rare but clinically significant complication following endoscopic intragastric administration.

Case Report

A 60-year-old female was admitted with dyspnea, diplopia, dysphagia, and generalized weakness after undergoing endoscopic intragastric botulinum toxin injection for weight loss. The procedure had been performed 10 days earlier, and her symptoms progressively worsened thereafter.

Neurological examination revealed muscle strength of 4/5 proximally and 5/5 distally in the upper extremities. In the lower extremities, strength was 2/5 proximally and 4/5 distally. Laboratory tests and central nervous system imaging showed no significant abnormalities. Based on the clinical findings and recent history of botulinum toxin administration, systemic botulinum toxin effect was suspected.

The patient was admitted to the intensive care unit for close monitoring and received botulinum antitoxin therapy.

Discussion

Systemic toxicity following endoscopic intragastric botulinum toxin injection is extremely rare. However, high doses, deep-layer injections, or inadvertent injection near vascular structures may increase the risk of systemic dissemination. In this case, as the procedure was performed at an external center, the precise mechanism of systemic involvement could not be determined.

This case highlights that endoscopic intragastric botulinum toxin injection, although considered minimally invasive, may result in clinically significant systemic toxicity. Careful patient selection, appropriate dosing, and close post-procedural monitoring are essential to prevent delayed recognition of potentially life-threatening complications.

Key words: Adverse effects, Botulinum toxin, Intragastric injection, Obesity, Systemic toxicity

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8449

A Bayesian Diagnostic Perspective in Aortic Dissection: A 12-Year Retrospective Analysis of Low-Risk Phenotypes Using ADD-RS

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1. Introduction

Acute aortic dissection (AAD) is a catastrophic vascular emergency associated with substantial morbidity and mortality, particularly when diagnosis is delayed. Mortality in untreated proximal dissections increases rapidly during the first hours following symptom onset, making early recognition critically important. Despite advances in imaging modalities and emergency care systems, timely diagnosis remains challenging because the clinical presentation is often heterogeneous and may mimic more common conditions such as acute coronary syndrome, pulmonary embolism, musculoskeletal pain, or gastrointestinal disease.

Although sudden severe chest or back pain is traditionally considered the hallmark symptom of aortic dissection, a considerable subset of patients presents with atypical or incomplete clinical findings. Neurological deficits, syncope, isolated abdominal pain, unexplained hypotension, or even relatively nonspecific symptoms may dominate the presentation. Consequently, some patients initially appear to belong to low-risk clinical categories, potentially contributing to delayed imaging and missed diagnoses.

To improve diagnostic standardization, the Aortic Dissection Detection Risk Score (ADD-RS) was developed as a structured clinical tool integrating predisposing conditions, pain characteristics, and examination findings. Current diagnostic algorithms frequently combine ADD-RS with D-dimer testing to guide imaging decisions. Several studies have demonstrated high sensitivity for low ADD-RS combined with negative D-dimer results; however, uncertainty remains regarding the true distribution of low-risk phenotypes among confirmed dissection cases encountered in routine emergency practice.

In addition, most previous studies have focused primarily on sensitivity and negative predictive value rather than on Bayesian diagnostic reasoning. In real-world emergency medicine, clinicians continuously update diagnostic probability based on pretest likelihood, biomarkers, imaging findings, and evolving clinical information. Bayesian approaches may therefore provide a more clinically meaningful framework for interpreting diagnostic strategies in suspected aortic dissection.

The present study aimed to retrospectively evaluate the distribution of ADD-RS categories among confirmed aortic dissection patients over a 12-year period and to interpret the diagnostic performance of ADD-RS and D-dimer within a Bayesian framework. Particular

emphasis was placed on identifying the proportion of patients who initially appeared to have low or intermediate clinical risk despite confirmed disease.

2. Materials and Methods

2.1 Study Design and Setting

This retrospective observational cohort study was conducted at Bandırma Onyedi Eylül University Faculty of Medicine and its affiliated emergency department network. The institution serves as a regional tertiary referral center with approximately 300,000 annual emergency department visits.

Electronic medical records between October 1, 2014, and October 1, 2026, were retrospectively reviewed.

2.2 Patient Selection

Patients aged ≥18 years with radiologically confirmed acute aortic dissection diagnosed by thoracoabdominal computed tomography angiography (CTA) were eligible for inclusion.

Patients with chronic aortic dissection, traumatic aortic injury, isolated intramural hematoma without definite dissection, or incomplete clinical documentation were excluded.

During the study period, 178 patients met inclusion criteria.

2.3 Control Population

During the same period, 12,036 patients underwent thoracoabdominal CTA because of suspected acute aortic pathology or severe thoracoabdominal symptoms. Patients without aortic dissection constituted the potential control pool.

From this population, 356 patients were randomly selected using an approximate 1:2 case-control ratio to create the comparison cohort.

2.4 Data Collection

Demographic characteristics, presenting symptoms, hemodynamic findings, neurological deficits, laboratory values, D-dimer levels, imaging findings, Stanford classification, treatment strategy, and in-hospital mortality were recorded.

ADD-RS scores were retrospectively calculated based on documented clinical findings using the three standard domains:

1. High-risk conditions
2. High-risk pain features

3. High-risk examination features

Scores were categorized as:

- ADD-RS 0
- ADD-RS 1
- ADD-RS >1

2.5 Bayesian Diagnostic Analysis

Diagnostic performance measures including sensitivity, specificity, positive likelihood ratio (LR+), and negative likelihood ratio (LR−) were calculated for D-dimer and ADD-RS categories.

Bayesian post-test probability estimates were derived using likelihood ratios and estimated pretest probabilities based on the study population. Diagnostic reasoning was interpreted conceptually using Bayesian principles rather than advanced probabilistic modeling.

2.6 Statistical Analysis

Continuous variables were summarized as mean ± standard deviation or median (interquartile range), depending on distribution. Categorical variables were presented as counts and percentages.

Group comparisons were performed using chi-square or Fisher's exact tests for categorical variables and Student's t-test or Mann–Whitney U test for continuous variables.

A two-sided p value <0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics version 26.0.

3. Results

3.1 Baseline Characteristics

A total of 178 patients with confirmed acute aortic dissection were included. Mean age was 61.2 years, and 71% were male. Sudden chest and/or back pain was present in 74.3% of patients. Hypotension was observed in 1.2%, and neurological deficits were identified in 3.5%. D-dimer levels were elevated in 161 patients (90.4%).

According to Stanford classification:

- 104 patients (58.4%) had type A dissection
- 61 patients (34.3%) had type B dissection

The remaining patients had incomplete classification data. Mean symptom onset-to-emergency department arrival time was approximately 67 minutes, while mean diagnosis time was 79 minutes. Overall in-hospital mortality was 27%. Baseline demographic and clinical characteristics are summarized in **Table 1**.

Table 1. Baseline demographic and clinical characteristics of confirmed aortic dissection patients.

Variable	Value
Mean age	61.2 years
Male sex	71%
Sudden chest/back pain	74.3%
Elevated D-dimer	90.4%
Stanford type A	58.4%
Stanford type B	34.3%
In-hospital mortality	27%

3.2 Distribution of ADD-RS Categories

Retrospective ADD-RS analysis demonstrated that:

- 6.7% of patients had ADD-RS 0
- 37.6% had ADD-RS 1
- 55.6% had ADD-RS >1

Overall, 44.4% of confirmed dissections fell into low-to-intermediate clinical risk categories (ADD-RS ≤1). The distribution of ADD-RS categories is presented in **Table 2 and Figure 2.**

Table 2. Distribution of ADD-RS categories among confirmed aortic dissection patients.

ADD-RS Category	n	%
0	12	6.7
1	67	37.6
>1	99	55.6

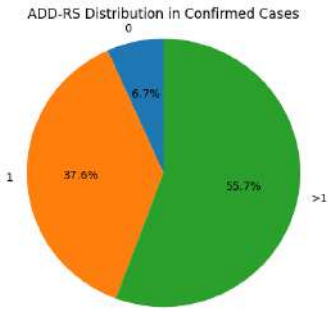

Figure 2. Distribution of ADD-RS Categories Among Patients With Confirmed Acute Aortic Dissection.

3.3 Diagnostic Performance of D-Dimer

D-dimer demonstrated:

- Sensitivity: 90.4%
- Specificity: 64.9%
- Positive likelihood ratio: 2.58
- Negative likelihood ratio: 0.15

The combination of ADD-RS and D-dimer improved diagnostic discrimination compared with isolated clinical assessment alone. Diagnostic performance metrics are summarized in **Table 3**.

Table 3. Diagnostic performance of D-dimer in suspected acute aortic dissection.

Diagnostic Parameter	Value
Sensitivity	90.4%
Specificity	64.9%
Positive likelihood ratio (LR+)	2.58
Negative likelihood ratio (LR−)	0.15

3.4 Bayesian Diagnostic Interpretation

Within a Bayesian framework, negative D-dimer results substantially reduced post-test probability among low-risk patients but did not completely eliminate diagnostic possibility.

Similarly, low ADD-RS categories were associated with lower estimated pretest probability; however, a clinically meaningful proportion of confirmed dissections remained within these categories.

These findings suggest that Bayesian integration of clinical assessment and biomarkers may provide a more realistic diagnostic approach than rigid dichotomous classification alone.

4. Discussion

This retrospective study demonstrated that nearly half of confirmed aortic dissection patients retrospectively fell into low-to-intermediate clinical risk categories according to ADD-RS. Although high-risk presentations predominated overall, a substantial minority of patients lacked classical high-risk phenotypes.

These findings are clinically important because diagnostic delay remains one of the major contributors to mortality in acute aortic dissection. Emergency physicians frequently rely on pattern recognition and initial clinical probability assessment when deciding whether advanced imaging is warranted. However, the present findings suggest that reliance on classical high-risk presentation alone may be insufficient.

The observed sensitivity of D-dimer was consistent with previous literature demonstrating high but imperfect diagnostic performance. Importantly, the Bayesian perspective highlights that diagnostic tests do not function in isolation; rather, their clinical value depends on pretest probability.

In practical terms, a negative D-dimer in a very low-risk patient may reduce post-test probability sufficiently to defer imaging, whereas the same result in a patient with concerning clinical features may remain insufficiently reassuring.

The concept of “low-risk phenotype” deserves particular attention. In emergency medicine, clinicians may unconsciously equate low-risk appearance with low disease probability. However, our findings suggest that acute aortic dissection may occasionally masquerade as relatively benign presentations, emphasizing the need for continued diagnostic vigilance.

This study should be interpreted in light of several limitations. The retrospective single-center design limits causal interpretation and generalizability. ADD-RS was calculated retrospectively and therefore depended on documentation quality. Selection bias may also exist because only patients undergoing CTA were included. Furthermore, Bayesian interpretation was conceptual and clinically oriented rather than based on advanced probabilistic simulation models.

Nevertheless, the study reflects real-world emergency department practice over a prolonged study period and provides clinically relevant insight into the distribution of low-risk phenotypes among confirmed dissection patients.

5. Conclusions

A considerable proportion of patients with confirmed acute aortic dissection retrospectively demonstrated low-to-intermediate clinical risk profiles according to ADD-RS. Although D-dimer showed high sensitivity, no single parameter was sufficient to fully exclude disease.

A Bayesian diagnostic framework integrating clinical probability assessment and biomarker interpretation may improve diagnostic reasoning and reduce missed diagnoses in suspected acute aortic dissection.

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Case Report: Acute Ischemic Stroke with The Hyperdense MCA Sign.

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Introduction:

The hyperdense middle cerebral artery sign (HMCAS) on a CT scan is a crucial radiological finding in the early detection of acute arterial thrombotic occlusion (1). It is typically observed within the first 90 minutes of the ischemic event and can be one of the earliest indicators of MCA occlusion (2). While magnetic resonance imaging (MRI) is considered standard for stroke diagnosis, it may not be feasible in certain patients, particularly those with implanted cardiac devices such as permanent pacemakers. In such situations, rapid diagnosis must rely on CT-based imaging.

We present a case of acute ischemic stroke in a patient with a permanent pacemaker, where MRI could not be performed, and the diagnosis of MCA occlusion was established based on the hyperdense MCA sign and CT angiography findings. This case highlights the critical role of CT imaging in the early identification and management of large vessel occlusion when MRI is contraindicated.

Case:

A 65-year-old male patient presented to our emergency department with acute-onset vomiting while eating, followed by decreased level of consciousness, inability to hold his head up, and collapse to the left side. The patient underwent mechanical aortic valve replacement 17 years ago. A permanent pacemaker was implanted 15 years ago, with generator replacement performed every five years.

The patient has been receiving quetiapine 150 mg, metoprolol tartrate, and warfarin sodium. Warfarin had been administered as one tablet daily for six days per week and half a tablet on

one day per week. Two weeks ago, the patient's INR was measured at 3.38; therefore, the warfarin regimen was adjusted to half a tablet on Monday, Wednesday, and Friday. This week, the INR was found to be 2.7, and the patient took only half a tablet on Thursday.

Initial vitals of the patient's revealed as follows: temperature: 36,1°C, pulse: 77 bpm, oxygen saturation 99% on room air, blood pressure:123/61mmHg, and blood glucose level:93 mg/dl. Neurological examination revealed a Glasgow Coma Scale (GCS) score of 9. The patient did not open eyes to painful stimuli but responded verbally to auditory stimuli. Pupils were isochoric and reactive to light bilaterally. The oculocephalic reflex was present. There was no forced gaze deviation. A flattening of the left nasolabial fold was noted. The right extremities demonstrated spontaneous movements, whereas the left extremities showed early drift on motor examination. The National Institutes of Health Stroke Scale (NIHSS) score was 17. Abdomen and lung examination revealed no abnormalities. ECG showed sinus rhythm with a wide QRS complex. The patient's inital laboratory results were as follows: INR:1.73, eGFR: 72, AST:20 U/L, ALT:12 U/L, GGT: 25 U/L, LDH:231 U/L, total bilirubin 0.66mg/dl, direct bilirubin: 0.21 mg/dl, amylase: 31U/L, Lipase: 30 U/L, CRP 12.10 mg/L, hemoglobin (HGB): 12.2 g/dl, red blood cells (RBC):4.41 million/ul, white blood cells (WBC):10,86 10*3/Ul, platelets (PLT): 319 10*3/Ul.

Brain CT showed no evidence of intracranial hemorrhage; however, a hyperdense artery sign was observed in the right MCA M1 segment. On CT angiography, occlusion of the right MCA M1 segment was confirmed. MRI could not be performed due to the patient's permanent pacemaker. As the timing of the patient's last warfarin dose was unknown, intravenous thrombolysis with tPA was not administered. After consultation with interventional radiology, a decision was made to proceed with thrombectomy. Follow-up brain CT demonstrated loss of gray–white matter differentiation and hypodensity in the right MCA territory, consistent with ischemic edema. The patient was extubated one day after thrombectomy, with a GCS score of 14. During intensive care unit follow-up, the patient's body temperature was 36.7°C. Lung sounds were decreased bilaterally on auscultation. Thoracic CT revealed ground-glass opacities secondary to pulmonary edema, interlobular septal thickening, peribronchial thickening, bilateral pleural effusions in the basal segments of the lower lobes, and associated passive atelectasis. In consultation with the infectious diseases and pulmonology departments, appropriate antibiotic therapy was initiated. After 16 days in the intensive care unit, the patient was transferred to the neurology ward for further management.

Discussion:

This case highlights the critical role of non-contrast CT in the early diagnosis of acute ischemic stroke, especially in patients with a permanent pacemaker in whom MRI cannot be performed. In acute stroke, rapid imaging is essential for timely treatment decisions. In this patient, the hyperdense MCA sign on the initial CT scan was an early and important finding that suggested large vessel occlusion. CT angiography confirmed right MCA M1 segment occlusion, and the patient was quickly referred for mechanical thrombectomy. This case demonstrates that CT and CT angiography can be sufficient for diagnosis and treatment

planning when MRI is contraindicated. Careful evaluation of early CT findings is essential, as recognition of the hyperdense MCA sign can directly affect patient outcomes.

Figure 1: A hyperdense MCA sign is observed in the right MCA M1.

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Sepsis in the emergency department, procalcitonin, rapid diagnosis paradigm: Report on the literature

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ABSTRACT

Introduction and Aim: Sepsis remains a very serious and life-threatening condition if not detected early and treated promptly. In severe sepsis cases, a clinical picture resulting in death can be seen, accompanied by septic shock along with at least one organ dysfunction and hyperfusion. In sepsis, microorganisms spreading throughout the body via the bloodstream create many virulence factors in circulation. Thus, various defense mechanisms are activated in the bloodstream, and an inflammatory response occurs. Immune cells secrete inflammatory proteins, and thus the clinical picture progresses to tissue damage and organ failure. The best clinical approach in sepsis is early diagnosis and broad-spectrum antibiotic treatment. In rapid diagnosis, procalcitonin (PCT) is an important biomarker for blood infections and inflammatory response. In this context, the aim of this study is to evaluate the role and

accuracy of PCT as a biomarker in the diagnosis of septic patients in emergency departments (EDs).
Materials and Methods: A retrospective worldwide literature review revealed that many biomarkers are used in the rapid diagnosis of sepsis. An electronic database search showed that PCT ranked first in rapid diagnosis.
Results and Conclusion: In the diagnosis of sepsis, pathogenic antigens to be serologically tested in blood are important biomarkers. Among the biomarkers of host response are CRP, lactate, and PCT. However, since CRP and lactate can show significant increases in different complications, it has been emphasized that PCT is relatively more useful. Worldwide studies have reported high PCT levels as a significant indicator of bacterial infections. The rate and degree of PCT increase provide important clues to the emergency room physician about which patient will die and which patient will be admitted to intensive care. This situation provides the opportunity to create a sepsis management algorithm in emergency rooms guided by PCT. While bacterial and host response biomarkers alone do not provide a definitive diagnosis for sepsis, they play a guiding role in the rapid diagnosis and treatment of the disease. A definitive diagnosis of sepsis should be made by evaluating the patient's history, physical examination, and multiple diagnostic tests. However, a rapid, albeit not definitive, diagnosis will significantly reduce the need for hospitalization and the prescription of antimicrobial drugs. Therefore, blood cultures should always be taken for sepsis diagnosis, and two or more biomarkers should be used in conjunction with them. Among biomarkers, PCT is a good option for aiding in rapid diagnosis. It is thought that the use of PCT, especially in critical care settings such as emergency departments where assessments are repeated, would be beneficial as a stepping stone to rapid diagnosis.

Keywords: Procalcitonin, sepsis, rapid diagnosis

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More Than Just a Name: Implementing "John Doe" Protocols in a Critically Injured Patient

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Introduction

As the busiest and most chaotic part of modern medicine, emergency rooms are often the first and most important place for "unknown patients" to go. These people are often unconscious, seriously cognitively impaired, or don't have identification papers because of political or social reasons. Concerning these patients, whose names can't be revealed, the "John/Jane Doe" procedure is used, which is similar to another protocol used in medical literature [1]. Taking care of these cases takes both medical knowledge and a strong sense of right and wrong so that bureaucratic problems don't get in the way of the patients' health [2]. An emergency medicine expert doesn't see anonymity as a state of uncertainty that stops the medical process. Instead, they see it as a "red alert" situation that makes the patient more vulnerable and raises the bar for ethical care on the "right to life" [3].

Case

The male patient, who was possibly thirty-five years old, was taken to our emergency room by emergency medical services after he hurt himself in a fall. The medical process for the patient began right away under the "unknown patient" procedures, even though the patient did not have any identification papers and could not give a medical history because they did not speak English or did not want to give information. The first medical exam showed that the person was in a coma (GCS = 15) and that their blood pressure and heart rate were stable. During the thorough physical exam, however, a 4 cm cut that went all the way through to the bone in the right frontal area, widespread abrasions in the right zygomatic area, and a major deformity with local tenderness that compromised the anatomical integrity in the right wrist were all seen.

The multitrauma procedure said that all laboratory and x-ray tests should be ordered at the same time through a temporary barcode, without waiting for the patient to be identified. To lower the chance of infection, broad-spectrum intravenous antibiotic therapy and tetanus prevention were given right away. Advanced imaging methods showed a broken inner table of the right frontal bone, minor pneumocephalus (indicating air buildup inside the skull), a T3-T4 spinous process fracture, and a displaced fracture in the distal right radius.

An emergency reduction and stabilizing with a short arm splint was done by the orthopedics department on the broken radius. The Thoracic Surgery department did conservative monitoring for the bilateral venous congestion seen on the thoracic tomography, which was consistent with a lung contusion. The Neurosurgery department operated on the patient

because of life-threatening complications like a compression fracture and pneumocephalus, and the surgery was an emergency. After the surgery, the patient was stable for 8 days of clinical follow-up, and their basic needs were met with the help of the Psycho-Social Support unit. This made it safe for them to go home.

Discussion

This case shows that taking care of unidentified patients in emergency medicine is not a "passive waiting process," but a "active protection and intervention process" that protects the patient's rights and makes sure they stay healthy [4,5]. Every second spent trying to figure out who the patient is before medical help arrives could mean death, especially in cases of worsening illnesses like pneumocephalus and intracranial fractures.

All over the world, the "John/Jane Doe Protocol" is what keeps the medical system running. This procedure gives each patient a unique temporary identity until their real identity is found [6]. This way, operational steps can be carried out with no room for error. This process is supported by the concept of "Implied Consent," which makes the doctor the patient's legal and moral guardian at this delicate point where the patient can't speak up for their own rights [7]. Also, it's important to remember that every unidentified trauma case is a "forensic case" by definition, and that all efforts must be coordinated with the police in steps like fingerprint recognition [8,9].

Conclusion

For this reason, not being able to identify someone does not mean that medical care should be put off. In the medical world, "the gap in the file" just means that there is no name, and this should never get in the way of the patient's holy "right to life". This case's successful coordination is a moral example of how anyone, even someone who can't be named, can fully benefit from all that modern medicine has to offer.

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Keywords: Emergency Department, Unknown Patient, John Doe Protocol, Multitrauma, Ethics.

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Open-Book Pelvic Fracture in a Severe Polytrauma Patient Following a Fall from Height: A Case Report

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Introduction

Open-book pelvic fractures represent a form of pelvic ring disruption associated with high-energy trauma, such as falls from height or motor vehicle accidents (1). They are caused by anteroposterior compression forces and are characterized by disruption of the pubic symphysis with widening of the anterior pelvic ring (2). These injuries are clinically significant due to associated pelvic instability and a high risk of massive hemorrhage from the richly vascular pelvic cavity (1).

We present a case of a young female patient with severe multiple traumas following a fall from height, highlighting the presence of an open-book pelvic fracture with profound hemodynamic instability and a complex ICU course with delayed in-hospital mortality.

Case

A 32-year-old female was admitted to our emergency department with multiple trauma following a fall from a fourth-floor window. On arrival, the patient was in critical condition with unstable vital signs and impaired consciousness. Neurological examination revealed a

Glasgow Coma Scale (GCS) score of 3. Pupils were anisocoric with reduced light reflex (IR −2/−3). Motor strength could not be assessed. Physical examination showed right periorbital and maxillary ecchymosis. Due to severe neurological and respiratory compromise, the patient was orotracheally intubated upon admission.

Initial vital signs indicated severe hemodynamic instability with blood pressure (BP) ranging from 40–110/20–61 mmHg, heart rate between 120–145 beats per minute, and oxygen saturation was 98–99% under mechanical ventilation. Peripheral pulses were weak and capillary refill time was prolonged (>3 sc). Cardiac rhythm was regular on electrocardiography.

On initial examination bilateral breath sounds were decreased, and thoracic CT confirmed bilateral pneumothorax and left sided rib fractures (4th-6th). Right apical and left lateral thoracostomies were performed.

Cranial CT revealed pneumocephalus, subarachnoid hemorrhage and multiple craniofacial fractures. Radiological findings were also consistent with diffuse cerebral edema, including sulcal effacement, ventricular compression, and approximately 7 mm midline shift. Abdominopelvic CT revealed an approximately 2 cm mesenteric hematoma and minimal intra-abdominal free fluid within the pelvis and the Retzius space.

Pelvis CT findings were consistent with an Open Book Pelvic Fracture, including symphysis pubis diastasis, widening of the right sacroiliac joint, and displaced fractures of the right superior and inferior pubic rami. Associated soft tissue hemorrhage and suprapubic subcutaneous hematoma were also observed.

Additional injuries included multiple long bone fractures, most notably a comminuted fracture of the left femur.

Initial laboratory findings were as follows: INR: 1.02, eGFR: 72, Creatinine: 1.03 mg/dL, AST: 179 U/L, ALT: 170 U/L, GGT: 20 U/L, LDH: 543 U/L, total bilirubin 0.36 mg/dL, direct bilirubin: 0.10 mg/dL, amylase: 113 U/L, Lipase: 198 U/L, CRP 1.40 mg/L, hemoglobin (HGB): 12.5 g/dL, red blood cells (RBC): 4.16 million/ul, white blood cells (WBC): 14.44 10*3/Ul, platelets (PLT): 287 10*3/Ul.

Arterial blood gas analysis showed pH 7.112, pCO2 43.5 mmHG, pO2 61.8 mmHG, HCO3 12.5 mmol/L, base excess -14.4.

During follow-up, a progressive rise in inflammatory markers, including C-reactive protein (CRP): 296, was observed in the context of severe polytrauma.

The patient was managed in the intensive care unit. Due to severe traumatic brain injury and multisystem trauma, surgical intervention was not performed by neurosurgery and orthopedics teams. Conservative management included sedation, mechanical ventilation, and antibiotic

therapy. Importantly, a hammock sling traction was applied as a non-invasive method for stabilization of the open-book pelvic fracture and reduction of pelvic volume.

Despite intensive care management, on hospital day 12, the patient developed cardiac arrest with absence of pulse and blood pressure. Cardiopulmonary resuscitation was initiated with chest compressions, however the patient could ot be resuscitated and was pronounced dead.

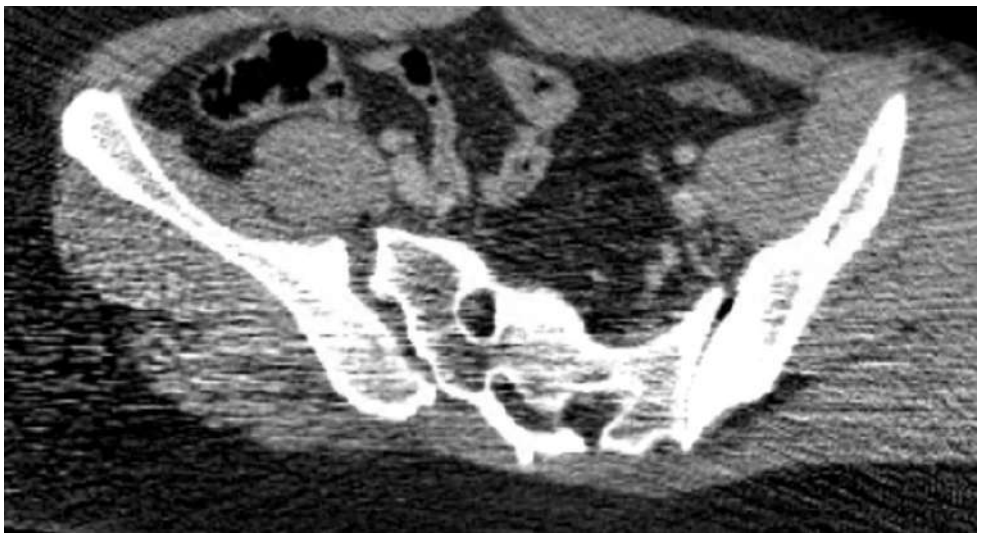

Figure 1: Pelvis CT findings were consistent with an Open Book Pelvic Fracture, including symphysis pubis diastasis, widening of the right sacroiliac joint, and displaced fractures of the right superior and inferior pubic rami.

Discussion

Open-book pelvic fractures predispose to significant hemorrhage due to disruption of pelvic ring integrity, which increases pelvic volume and reduces the tamponade effect of the retroperitoneum (3). While early management focuses on hemodynamic stabilization and mechanical reduction of pelvic volume (1), definitive treatment of open-book fractures is highly individualized, depends on the patient's overall clinical status and remain secondary to hemorrhage control (4). Non-invasive stabilization techniques, such as pelvic binders or external compression methods, can be used to restore pelvic tamponade and control ongoing hemorrhage (3); in our present case, hammock sling traction was applied for pelvic stabilization.

In this case, the patient presented with profound hypotension and severe metabolic acidosis, consistent with shock physiology in the setting of major polytrauma. Although the pelvic fracture likely contributed to the initial instability, severe traumatic brain injury, bilateral pneumothorax, and multiple fractures indicate a multifactorial clinical course.

This case highlights the challenges of managing open-book pelvic fractures in the setting of severe multisystem trauma and emphasizes the importance of individualized, multidisciplinary decision-making, particularly when definitive surgical management is not possible.

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Chest Pain and Elevated Troponin in Pregnancy: Diagnostic Challenges of Mild Pericarditis

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Introduction

Pericarditis is a rare cardiac condition during pregnancy but carries the risk of maternal and fetal complications [1]. Viral etiology is among the most common causes [2]. In patients presenting with chest pain during the second trimester of pregnancy, pericarditis should be considered in the differential diagnosis [1,3]. This case demonstrates that mild cases of probable viral pericarditis during pregnancy can be successfully managed with conservative treatment and close follow-up.

Case

A 26-year-old woman at 16 weeks of gestation presented to the emergency department with a complaint of chest pain that had persisted for 5 days. The pain was particularly aggravated in the supine position. There was no dyspnea, cough, or fever. She had a history of upper respiratory tract infection during the previous week. The patient's vital signs were as follows: blood pressure 107/73 mmHg, heart rate 85 beats/min, SpO_2 99%, and body temperature

36.7°C. Physical Examination: System examinations were unremarkable. Laboratory Findings: Troponin levels were 66 ng/L, then 64 ng/L, and subsequently 38 ng/L (upper limit: 14 ng/L). CRP was 7 mg/L (upper limit: 5 mg/L), WBC was 15,000/µL, and hemoglobin was 9.2 g/dL.ECG: Evaluated as normal sinus rhythm. Imaging / Consultations: Cardiology: Echocardiography showed an ejection fraction of 65%; right heart chambers and cardiac valves were evaluated as normal. A preliminary diagnosis of pericarditis was considered. Obstetrics and Gynecology: No obstetric pathology was detected; daily fetal heart rate monitoring was recommended. Management and Follow-up: No medical treatment was administered because of teratogenic risk. During follow-up, the patient's symptoms decreased, and she was discharged with recommendations.

Discussion

Pericarditis during pregnancy is rare, and establishing the diagnosis can be particularly challenging during the second trimester. Viral pericarditis generally follows a mild course and may respond to medical treatment. However, nonsteroidal anti-inflammatory drugs and other therapeutic agents may carry teratogenic risks during pregnancy. Therefore, in patients with mild symptoms, a conservative approach and close follow-up may be a safe option. Elevated troponin levels indicate an inflammatory response, and the patient's hemodynamic stability influenced the treatment decision [1,2,3].

Conclusion

This case demonstrates that probable mild viral pericarditis occurring during the second trimester of pregnancy can be managed with close follow-up without the need for medical treatment [1,2]. A multidisciplinary approach is important in pregnant patients presenting with cardiac symptoms [3].

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Keywords: Pregnancy, Pericarditis, Case Report, Troponin, Conservative Management

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Wellens Syndrome Identified Through Atypical Presenting Complaints: A Case Report

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INTRODUCTION:

Wellens syndrome has a characteristic electrocardiographic pattern defined by biphasic or deep, symmetric T-wave inversions, particularly in the precordial leads and most commonly in leads V2 and V3, and is often associated with a history of recently resolved or regressed chest pain [1,2]. These findings carry highly important clinical and prognostic value in suggesting the presence of critical stenosis in the left anterior descending artery (LAD) [1,2]. Wellens syndrome must be recognized early because it indicates a substantial risk of severe ischemia involving the vessel supplying the anterior wall [1,2]. In the European Society of Cardiology (ESC) Acute Coronary Syndromes guideline, Wellens syndrome is evaluated within the spectrum of acute coronary syndromes without persistent ST-segment elevation or other signs of acute vessel occlusion and, in this respect, is regarded as a high-risk clinical condition that differs from classical ST-elevation patterns but requires a similar degree of attention, caution, and rapid clinical decision-making [1]. Although the underlying mechanism is thought to be spontaneous reperfusion following transient occlusion of the LAD, this reperfusion is known not to be reassuring because the restored coronary perfusion is not sustained and has an unstable natüre [1-4]. Therefore, patients are at high risk for progressive myocardial ischemia and extensive anterior myocardial infarction, and they must be recognized in a timely manner, appropriately risk-stratified, and evaluated and managed without delay using an invasive approach [1-4].

CASE PRESENTATION:

A 67-year-old male patient presented to the emergency department at approximately 05:30 with a complaint of sore throat and described marked fatigue and a sensation of weakness in both upper extremities accompanying this complaint. His medical history revealed that he had presented to his family physician two days earlier because of an upper respiratory tract infection, but stated that there had been no marked improvement in his symptoms despite the current treatment. The anatomical region that the patient described as sore throat and indicated with his hand was consistent with the infraglottic area, which was noteworthy in

suggesting that the presenting complaint might differ from the simple upper airway pathology initially implied. The vital signs at presentation are presented in detail in Table 1.

An ECG was obtained from the patient, who was found to be hypertensive on initial evaluation and whose general condition appeared distressed. The initial ECG findings are presented in Figure 1. Because these findings were considered potentially compatible with Wellens syndrome, a cardiology consultation was requested for further evaluation in terms of possible critical coronary ischemia. The follow-up ECG obtained during observation is presented in Figure 2. Serial ECG changes were interpreted by the cardiology team as consistent with ST-segment elevation myocardial infarction (STEMI), and urgent coronary angiography was recommended for the patient. Owing to bed occupancy in the coronary intensive care unit of our hospital, the patient was transferred to another center where coronary angiography could be performed. This process is also important in demonstrating that presenting complaints that initially appeared relatively nonspecific and misleading were understood, through careful clinical evaluation and serial ECG monitoring, to have progressed within a short period to a high-risk acute coronary syndrome.

DISCUSSION AND CONCLUSION:

In elderly patients, obtaining a detailed medical history and performing a careful physical examination during the diagnostic evaluation process are of fundamental importance in correctly shaping the pathway to diagnosis [2]. This case clearly demonstrates that, particularly in patients with atypical presenting complaints, evaluating the depth of the history and physical examination findings not as independent elements but together within an integrated clinical framework may have life-saving consequences in reaching the correct diagnosis in a timely manner, rapidly directing the patient to the appropriate specialty, and planning effective treatment without delay [2]. In addition, this case is noteworthy in showing that classical chest pain symptomatology may not always predominate in the elderly population, and that misleading descriptions of localization and accompanying nonspecific symptoms may mask serious underlying cardiac pathologies[2].

Table 1. The patient's vital signs on arrival

Blood Pressure	Heart Rate	Body Temperature	Fingertip Oxygen Saturation
219/141 mmHg	84 beats/min	36.1 °C	97 %

Figure 1. Initial electrocardiogram of the patient

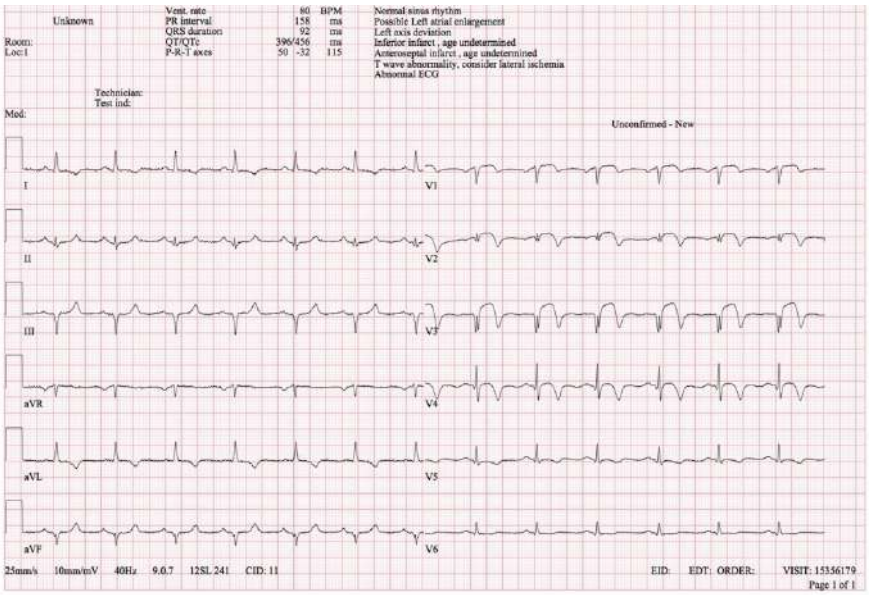
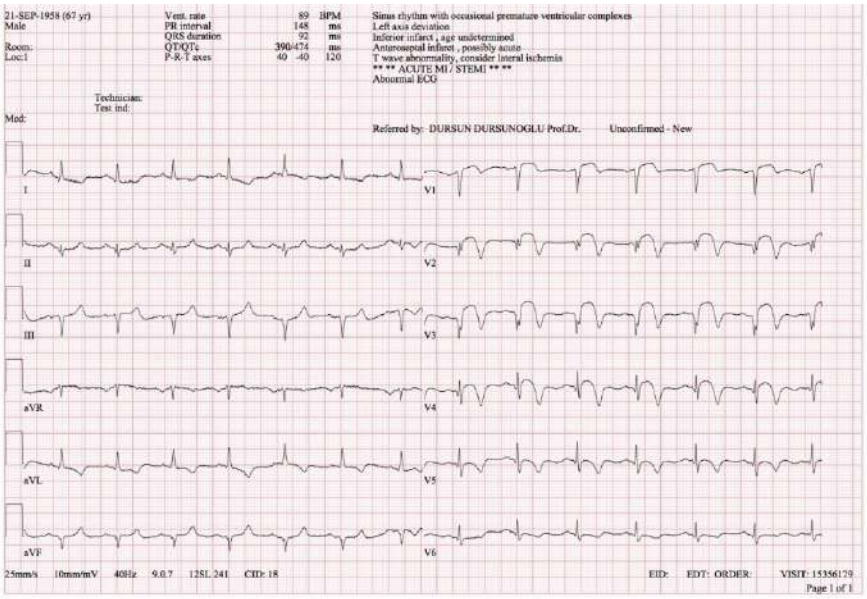


Figure 2. Follow-up electrocardiogram



KEYWORDS: Acute coronary syndrome, atypical symptom, electrocardiography, sore throat, Wellens syndrome

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Evaluation of Clinical and Laboratory Findings in Pediatric Electrical Injuries: An Emergency Department-Based Retrospective Study

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Abstract

Objective: Although electrical injuries are rarely seen in the pediatric age group, they are emergencies that can lead to severe clinical outcomes. This study aimed to evaluate the demographic, clinical, and laboratory characteristics of pediatric electrical injury cases presenting to the emergency department (ED) and to investigate the factors associated with hospitalization.

Materials and Methods: This retrospective observational study included patients under the age of 18 who presented to the ED and were diagnosed with electrical injury between January 1, 2025, and January 1, 2026. Demographic data, clinical outcomes, and laboratory parameters were obtained from the hospital information system. Statistical analyses were performed using the SPSS 27 software. Appropriate parametric and non-parametric tests were utilized following normal distribution conformity tests.

Findings: A total of 37 patients were included in the study. Of the patients, 75.7% were male. Hospitalization was required for 59.5% of the cases. The mean age of hospitalized patients was found to be significantly lower (p=0.044). In terms of laboratory parameters and

inflammatory indices, no statistically significant difference was found between hospitalized patients and those followed up as outpatients ($p>0.05$). The hemoglobin value tended to be lower in the hospitalization group, but this difference was not statistically significant ($p=0.065$).

Conclusion: A significant proportion of patients with pediatric electrical injuries are hospitalized. While age stands out as a factor associated with hospitalization, the prognostic value of laboratory parameters appears to be limited. Clinical evaluation and close monitoring should remain the primary determinants in patient management.

Keywords: Pediatric electrical injury, emergency department, child, inflammatory markers, hospitalization, prognosis

Introduction

Although electrical injuries are rare in childhood, they represent one of the traumatic emergencies that can cause significant morbidity and mortality (1). In particular, low-voltage domestic accidents constitute the most common cause of electrical injury in the pediatric age group, whereas high-voltage exposures are associated with more severe clinical presentations (2,3). The vast majority of electrical injuries in children occur accidentally, with curious behavior and environmental risk factors playing an important role (4).

The effects of electrical current on the body vary depending on the voltage, duration of the current, the entry-exit pathway, and tissue resistance (5). Cardiac arrhythmias, burns, neurological damage, and musculoskeletal injuries are among the primary complications of electrical injuries (6). Especially in pediatric patients, the systemic effects of electrical current can be more pronounced due to the smaller body surface area and the specific conductivity characteristics of tissues (7).

Clinical evaluation of pediatric electrical injuries presenting to the emergency department is often challenging. A significant portion of patients may initially be asymptomatic, but cardiac or neurological complications may develop during the subsequent course (8). Therefore, early risk assessment and the determination of appropriate follow-up strategies are of great importance.

Studies regarding pediatric electrical injuries in the literature are limited, and a lack of up-to-date data focusing specifically on emergency department presentations is noteworthy (3,6). In this study, we analyzed the demographic, clinical, and laboratory characteristics of pediatric electrical injury cases presenting to the ED over the past year and compared laboratory parameters with hospitalization status.

Materials and Methods

This study was planned as a retrospective observational study evaluating pediatric electrical injury cases who presented to the Emergency Department of Malatya Training and Research Hospital between January 1, 2025, and January 1, 2026. Patients were screened through the hospital information management system using ICD codes. Patients under 18 years of age and diagnosed with electrical injury were included in the study. Cases with missing data or unverified diagnoses were excluded.

The patients' demographic characteristics (age, sex), clinical outcomes (hospitalization status), and laboratory parameters at the time of presentation were recorded. The evaluated laboratory parameters were determined as: albumin (g/L), amylase (U/L), C-reactive protein (CRP, mg/L), white blood cell count (WBC, $x10^9$/L), platelet count ($x10^9$/L), hemoglobin (g/dL), neutrophil count ($x10^9$/L), lymphocyte count ($x10^9$/L), and monocyte count ($x10^9$/L). Additionally, the CRP-to-albumin ratio (CAR) and lymphocyte-to-albumin ratio (LAR) were calculated as inflammatory indices.

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics. The conformity of variables to normal distribution was evaluated using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Normally distributed variables were expressed as mean ± standard deviation, while non-normally distributed variables were expressed as median and interquartile range (IQR, 25th–75th percentiles). For intergroup comparisons, the Independent Samples t-test was used for normally distributed data, and the Mann–Whitney U test was used for non-normally distributed data. Categorical variables were presented as numbers and percentages (%). In all analyses, the statistical significance level was accepted as $p < 0.05$.

Findings

A total of 37 pediatric patients were included in the study. Of the patients, 75.7% were male and 24.3% were female. While 59.5% of the cases were hospitalized, 40.5% were followed up as outpatients.

In the normal distribution analysis, it was found that age, amylase, CRP, neutrophil, monocyte, and CAR values did not show a normal distribution; whereas albumin, WBC, platelet, hemoglobin, lymphocyte, and LAR values demonstrated a normal distribution.

In comparisons made according to hospitalization status, the age variable was found to be significantly lower in the hospitalized group ($p=0.044$). No statistically significant difference was detected between the groups in terms of albumin, WBC, platelets, hemoglobin, lymphocytes, and LAR (all p values >0.05). Similarly, no significant difference was observed regarding amylase, CRP, neutrophils, monocytes, and CAR values. Although the hemoglobin

value tended to be lower in the hospitalized group, this difference did not reach statistical significance (p=0.065).

Discussion

In this study, it was shown that the rate of hospitalization is high in pediatric electrical injury cases, and a younger age group is particularly associated with hospitalization. This finding suggests that electrical injuries in pediatric patients should be evaluated with greater clinical caution.

It is well known that the effects of electrical current on the organism depend on the voltage, duration, and systemic pathway of the current within the body (1). It has been reported that electrical injuries in the pediatric age group mostly result from low-voltage domestic sources and generally exhibit a milder clinical course (2,3). Nevertheless, some studies emphasize that delayed complications can arise even in patients who are initially asymptomatic (8).

In our study, no significant relationship was found between inflammatory markers or hematological parameters and hospitalization. This situation suggests that the prognostic value of laboratory parameters may be limited during the early period of electrical injuries. In the systematic review by Dechent D et al., it was similarly stated that the clinical outcome depends mostly on exposure characteristics (7).

Current literature reports that cardiac effects are prominent in electrical injuries, and therefore clinical observation and monitoring of patients are crucial. Waldmann V et al. emphasized that the risk of arrhythmia in electrical injuries necessitates clinical follow-up (9). Similarly, pediatric studies have shown that clinical evaluation and monitoring strategies are more decisive than laboratory parameters (10).

The demonstrated relationship between age and hospitalization suggests that young children represent a particularly vulnerable group. Therefore, it is of paramount importance to consider the age factor in clinical decision-making processes for pediatric electrical injuries.

Conclusion

A significant proportion of patients with pediatric electrical injuries are hospitalized. While age was found to be associated with hospitalization, the prognostic value of laboratory parameters is limited. Clinical evaluation and close monitoring should be prioritized.

Limitations

This study is retrospective, single-centered, and limited by a small sample size. Crucial parameters such as the voltage and duration of the electrical exposure could not be evaluated.

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Massive pulmonary embolism in a patient with glioblastoma multiforme: Clinical presentation and management

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Abstract

In this case report, we present a case of massive pulmonary embolism that developed in a patient diagnosed with glioblastoma multiforme. A patient who had undergone surgery for glioblastoma multiforme and was receiving chemotherapy presented to our emergency department with complaints of right leg swelling and acute dyspnea. Physical examination revealed pretibial oedema in both legs, with the right leg more swollen than the left and a noticeable difference in circumference. Laboratory results were as follows: C-reactive protein 92.4 mg/L, procalcitonin 0.09 ng/mL, D-dimer 3.170 µg/mL, urea 35 mg/dL, creatinine 0.51 mg/dL, and glomerular filtration rate 108 mL/dak/1.73 m². Arterial blood gas analysis showed pH 7.544, pO_2 70 mmHg, pCO_2 29.9 mmHg, HCO_3 25.2 mEq/L, and lactate 2.72 mmol/L. Computed tomography pulmonary angiography was performed. It demonstrated a saddle-type massive pulmonary embolism extending into all lobar and segmental branches of the bilateral distal pulmonary arteries, partially obstructing blood flow. Based on these clinical findings and diagnostic results, the patient was consulted with the pulmonology department and admitted to the pulmonary intensive care unit. Patients with glioblastoma multiforme represent a high-risk group for venous thromboembolism. Pulmonary embolism should always be considered and ruled out in the presence of sudden dyspnea and extremity swelling.

Keywords: Dyspnea, Glioblastoma multiforme, Pulmonary embolism.

Introduction

The risk of VTE in patients with cancer is approximately 4–7 times higher than in the general population [1]. The incidence of thromboembolism in patients with high-grade gliomas has been reported to be as high as 20–30% [2]. Pulmonary embolism is one of the most important causes of sudden clinical deterioration in patients with malignancy [3]. Therefore, in this case report, we present a case of massive pulmonary embolism that developed in a patient diagnosed with glioblastoma multiforme (GBM).

Case Report

A patient who had undergone surgery for GBM and was receiving chemotherapy presented to our emergency department with complaints of right leg swelling and acute dyspnea. On admission, the patient's vital signs were as follows: oxygen saturation 83%, heart rate 123 bpm, blood pressure 100/60 mmHg, and body temperature 36.7 °C. During the medical history evaluation, the patient was found to be using Keppra© (levetiracetam) 500 mg, Dekort© (dexamethasone) 10 mg, Diaformin© (metformin HCl), and 30 units of moderate effect insulin in the evening. Physical examination revealed pretibial edema in both legs, with the right leg more swollen than the left and a noticeable difference in circumference. Lung auscultation was unremarkable, and the other system examinations were within normal limits. Laboratory results were as follows: C-reactive protein (CRP) 92.4 mg/L, procalcitonin 0.09 ng/mL, D-dimer 3.170 µg/mL, urea 35 mg/dL, creatinine 0.51 mg/dL, and glomerular filtration rate (GFR) 108 mL/dak/1.73 m². Arterial blood gas analysis showed pH 7.544, pO_2 70 mmHg, pCO_2 29.9 mmHg, HCO_3 25.2 mEq/L, and lactate 2.72 mmol/L. Since renal function parameters were within normal limits, computed tomography pulmonary angiography (CTPA) was performed. CTPA demonstrated a saddle-type massive pulmonary embolism extending into all lobar and segmental branches of the bilateral distal pulmonary arteries, partially obstructing blood flow. Based on these clinical findings and diagnostic results, the patient was consulted with the pulmonology department and admitted to the pulmonary intensive care unit.

Discussion

Malignancy is an independent risk factor for thrombosis [1]. Brain tumors are among the cancer types with the highest risk of thromboembolism [2]. The pathogenesis involves tissue factor expression, increased cytokine release, and immobilization [1]. If untreated, mortality in massive pulmonary embolism can reach up to 30% [3].

Early clinical suspicion is crucial for diagnosing pulmonary embolism. Clinical probability scoring systems can help reduce unnecessary imaging. However, rapid decision-making in cases of massive pulmonary embolism significantly reduces mortality. Accurate risk stratification prevents unnecessary thrombolytic therapy while ensuring timely treatment in high-risk patients [4].

Prolonged immobilization, surgical interventions, malignancy, pregnancy and the postpartum period, oral contraceptive use, and genetic thrombophilia are among the important predisposing factors [5]. Symptoms of pulmonary embolism are generally nonspecific. However, the most common manifestations include sudden-onset dyspnea, pleuritic chest pain, and tachycardia [6]. Wells and Geneva scores are widely used clinical probability

scoring systems in the diagnosis of pulmonary embolism. These systems categorize patients into low-, intermediate-, and high-probability groups. Using these scores may help avoid unnecessary imaging studies [7,8]. Among laboratory parameters, D-dimer has a high negative predictive value in patients with low clinical probability [9]. However, the gold standard diagnostic method for pulmonary embolism remains computed tomography pulmonary angiography (CTPA) [9]. Ventilation/perfusion scintigraphy and lower extremity Doppler ultrasonography are additional supportive diagnostic tools when clinical suspicion for pulmonary embolism is high [4]. Our patient was a brain tumor patient who presented with shortness of breath and was diagnosed with pulmonary embolism via CTPA.

Conclusion

Patients with GBM represent a high-risk group for venous thromboembolism. Pulmonary embolism should always be considered and ruled out in the presence of sudden dyspnea and extremity swelling.

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CONTRIBUTION OF BEDSIDE LUNG ULTRASONOGRAPHY TO DIAGNOSE IN CASES CONSIDERED COVID-19 IN THE EMERGENCY DEPARTMENT

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Introduction:
The use of FL-USG has become widespread during the pandemic period, especially in areas such as ED where patient diagnosis and stabilization must proceed synchronously (1). Studies have shown that FL-USG provides information in correlation with CT about lung involvement in patients with difficulty diagnosing COVID-19 (1). Pathological changes seen in COVID-19 are particularly suitable for evaluation with FL-USG due to their peripheral and peripleural location (2).

In this study, we applied 6-ZONE to our patients, in which the patient is in a sitting position and the lung parenchymal tissue is evaluated from 6 different regions, in order to reduce the risk of contamination to the practitioner and to minimize the impact on droplet spread. Our aim in our study is to determine the diagnostic value of 6-ZONE in the ED. It also aims to determine the correlation between 6-ZONE and CT results for the diagnosis of COVID-19.

Material and Methods: This prospective observational clinical study included 50 patients who presented to the ED with suspicion of COVID-19. All participants underwent thorax CT, 6-ZONE and Real-Time Polymerase Chain Reaction (RT-PCR) test, but the reference diagnostic method was determined as thorax CT.

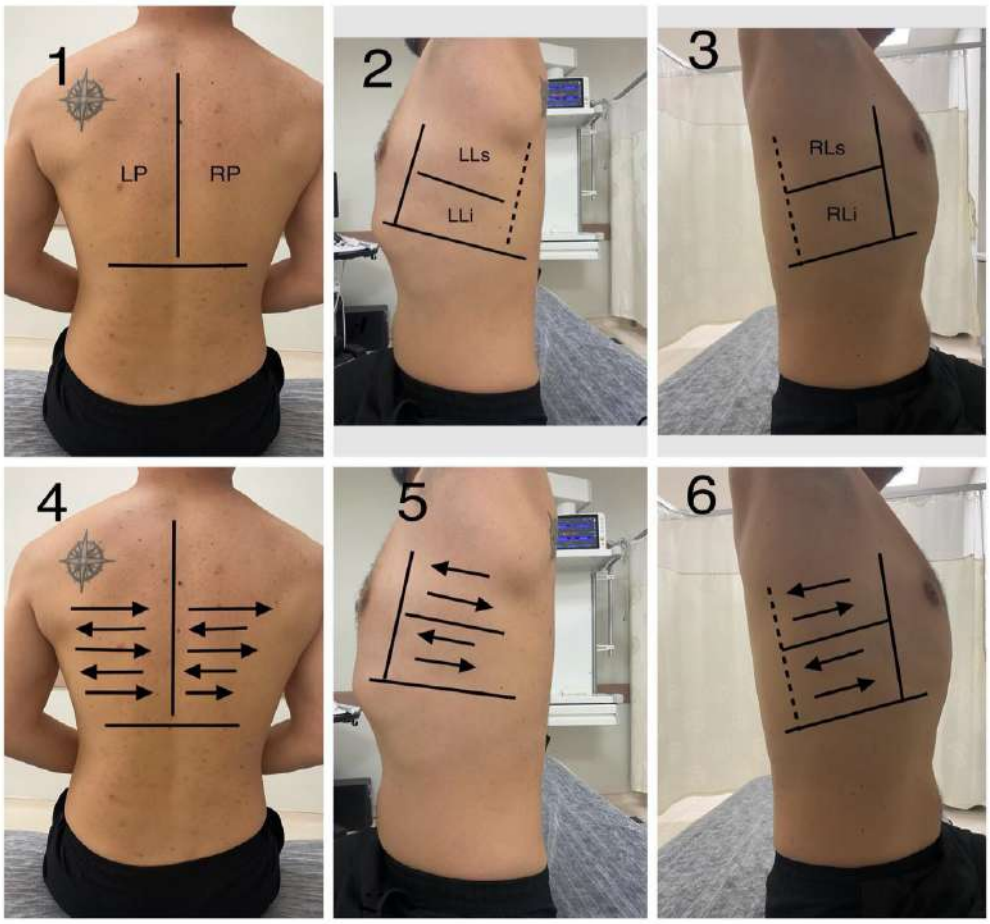
First of all, symptom evaluation and physical examination were performed by the emergency physician. Demographic data (age, gender), vital signs (pulse, fever, blood pressure, respiratory rate, oxygen saturation), symptoms (dyspnea, tachypnea, fever, cough, flu-like symptoms, extreme weakness, worsening of symptoms, if there is chronic respiratory disease, new loss of smell and taste, diarrhea, sore throat) were recorded in the standardized study form. Then, before performing diagnostic tests, FL-USG was performed according to the 6-ZONE technique (3) and the findings detected for each lung region (pleural thickening/disorder/effusion, subpleural consolidation, increase/fusion of B lines, air bronchograms, hepatization, absence of lung sliding movement) was noted on the data collection form by the same practitioner. Later, lung CT findings interpreted by physicians unaware of the study were obtained from the hospital information management system.

During the application, the patient was in a sitting position with his back turned in front of the practitioner and the USG device. This position was preferred because it would minimize the risk of contamination due to prolonged exposure. This position was preferred because it

would minimize the risk of contamination due to prolonged exposure. In addition, the sitting position is more comfortable for many patients, especially those with respiratory distress, and makes it easier to reach the posterior lung areas. There are studies reporting that the awake sitting position prevents alveolar collapse, increases oxygenation, and in some cases protects against intubation (4). While scanning the patient's lung zones, a comparative scan was performed with the other side (3).

While the probe was in the sagittal plane and the notch was pointing at the patient's head, scanning was started by sliding the probe backwards and forwards along the half of the thorax, between the posterior axillary line and the paravertebral line, up to the mid-scapular line. Afterwards, scanning was performed using the lawn mower technique, starting from the most posterior and lower point of the thorax, to evaluate the distance between the posterior and anterior axillary lines. We repeated these for the upper and lower lateral zones. Since COVID-19 disease usually shows bilateral involvement, the other half of the thorax was also scanned in the same way (Figure 1).

Figure 1: Anatomy and scanning technique for FL-USG in patient with suspected COVID-19.

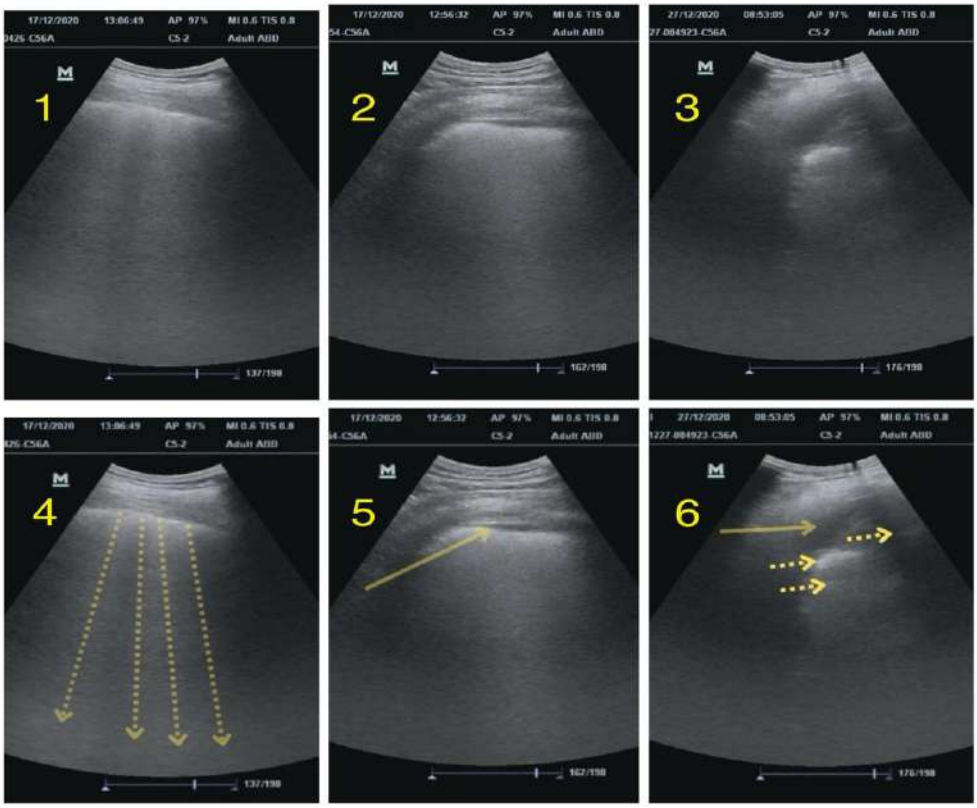


(1) Landmarks for scanning include the inferior thorax at the level of the diaphragm, the spine, the scapula, and the posterior axillary line. Lung zones to be scanned include the areas under the scapula border known as the right posterior (RP) and left posterior (LP) zones. Laterally, landmarks include the posterior axillary line to the anterior axillary line. Lateral lung zones to be scanned include the right lateral superior (RLs) and (3) right lateral inferior (RLi), and on the left include the left lateral superior (LLs), and (2) left lateral inferior (LLi). (4) To obtain scans, position a linear or curvilinear probe sagittal with probe marker cranially to the patient and slide the probe across the posterior surfaces. Repeat this technique in the (6) right and (5) left lateral zones.

Results: Lung imaging evaluations are shown in Table 2. According to thorax CT, findings in favor of pneumonia were detected in 58% of the cases (n=29), 27 of which were reported as COVID-19 pneumonia and 2 patients as bacterial pneumonia.

Based on 6-ZONE, the most common findings of COVID-19 pneumonia were pleural thickening (86% (n=43)), followed by pleural irregularity (76% (n=38), and increase/convergence of B lines 74% (n=37) (Figure 2).

Figure 2. Some imaging examples of COVID-19 patients.



(1, 4) Dashed yellow arrows indicate the increase in B lines. (2, 5) The yellow continuous arrow indicates subpleural consolidation. (3, 6) Solid yellow arrows indicate consolidation, while multiple dashed yellow arrows indicate air bronchograms.

According to FL-USG, 52% of the patients (n=26) were evaluated as COVID-19 pneumonia. However, FL-USG lung was found to be false positive in 2 patients and false negative in 3 patients. Accordingly, the sensitivity of FL-USG was calculated as 96.3%, specificity as 89.6%, positive predictive value as 94%, and negative predictive value as 86.6% (Table 1).

Table 1. Test characteristics of FL-USG in diagnosing COVID-19 pneumonia.

	Value	**95% CI**
Sensitivity	96,3	81,30-99,91%
Specificity	89,6	86,60-94,20%
Positive likelihood ratio	0,03	0,01-,028
Negative likelihood ratio	5,54	3,7-9,4
Positive predictive value	94	84,40-98,20%
Negative predictive value	86,6	79,10-90,30%

FL-USG: Focused Lung Ultrasonography, CI: Confidence Interval

Conclusions: 6-ZONE performed in the ED for the diagnosis of suspected COVID-19 has high sensitivity and specificity. Additionally, the use of the 6-ZONE should be favored as a standard training and imaging protocol for emergency physicians to use bedside FL-USG to aid in the diagnosis and exclusion of COVID-19 in the ED setting.

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Hemostatic Resuscitation in Proximal Upper Extremity Amputations: The Critical Role of REBOA and the XABCDE Approach

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Introduction: Proximal upper extremity injuries are classified as "junctional" injuries because they involve the axillary regions where the limb meets the torso. Due to their anatomical structure, these regions do not allow for standard tourniquet application and present severe challenges in controlling massive hemorrhage. In these injuries, known in the literature as "non-compressible" bleeding sites, the inadequacy of traditional resuscitation methods increases mortality rates. In this case report, we aim to present the importance of current hemostatic protocols and the X-ABCDE algorithm through a case involving an axillary amputation.

Case Report: A 15-year-old male child laborer was admitted to our emergency department with a total right shoulder amputation after his right arm was caught in a plastic crushing machine. It was learned that the patient, who went into cardiac arrest following massive hemorrhage at the scene, underwent cardiopulmonary resuscitation (CPR) for 60 minutes during the pre-hospital period. Upon admission to the hospital, the patient was intubated, showed no perfusion, and asystole was detected. The clinical presentation revealed extensive tissue loss at the shoulder level, along with bone and neurovascular exposure. The intervention team initiated the massive blood transfusion protocol and advanced cardiac life support. However, due to the inability to achieve proximal arterial control and the subsequent development of an "empty heart" presentation, the patient was pronounced dead after 45 minutes of advanced life support.

Discussion: The fundamental problem in proximal upper extremity amputations is that following the transection of the axillary artery, the elastic fibers within the vessel wall retract the vessel end behind the clavicle (deep retraction), and an effective compression surface is lacking. This situation highlights the inadequacy of the traditional ABC (Airway-Breathing-Circulation) sequence in emergency department practice. Current ATLS and Tintinalli approaches emphasize the X-ABCDE algorithm (with priority given to exsanguination control) to prevent preventable trauma deaths. Chest compressions performed without establishing vascular integrity worsen the prognosis by pumping the limited intravascular volume out of the active vascular lesion. It must be noted that in a hypovolemic system, mechanical compression cannot generate an effective stroke volume.

In cases where conventional tourniquets fail to achieve hemostasis, Foley catheter balloon tamponade placed into the wound tract or arterial lumen provides temporary control in over 90% of cases. Although REBOA (Resuscitative Endovascular Balloon Occlusion of the Aorta) is the gold standard for subdiaphragmatic hemorrhage, its use in these injuries is limited because the axillary region lies above REBOA Zone 1. Furthermore, the early application of specialized junctional tourniquets, such as the SAM Junctional Tourniquet, buys time until surgical intervention can be performed.

Conclusion: Proximal amputations and junctional injuries represent the highest-risk patient group in emergency department management. In the management of traumatic cardiac arrest, the primary goal should not be chest compression, but rather "intravascular volume replacement" and "proximal control of the active bleeding site." For a successful resuscitation, the X-ABCDE algorithm should be adopted, and specific techniques such as Foley catheter tamponade and junctional tourniquets should be deployed at the very beginning of the resuscitation process.

Keywords: Axillary amputation; junctional injury; hemostatic resuscitation; X-ABCDE; foley tamponade

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A Diagnostic Pitfall in Trauma Management: Incidental Acute Cholangitis

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INTRODUCTION: In emergency medicine, Computed Tomography (CT) is a cornerstone for identifying both traumatic injuries and incidental findings. Non-traumatic pathologies are observed in 30% to 50% of trauma-indicated CT scans, with 5% to 15% requiring urgent intervention(1,2). In this report, we present a case of acute cholangitis that was incidentally identified during the diagnostic workup of a multi-trauma patient.

CASE: A 62-year-old male patient presented to our emergency department following an intra-vehicular traffic accident. The patient's Glasgow Coma Scale (GCS) score was 15, and vital signs were within normal limits. Physical examination revealed crepitus and tenderness consistent with subcutaneous emphysema in the right thoracic region. Additionally, right upper quadrant tenderness was noted, though it was initially unclear whether this was secondary to trauma or an underlying biliary pathology. Imaging and laboratory investigations were initiated. Thoracic CT revealed fractures of the right 9th, 10th, 11th, and 12th ribs along with subcutaneous emphysema. Although no hepatic hematoma or laceration was detected on abdominal imaging, significant ectasia of the bile ducts and thickening of the ductal walls were identified (Figures 1). The patient was admitted to the Thoracic Surgery department due to multiple rib fractures and subcutaneous emphysema; a tube thoracostomy was subsequently performed during follow-up. A General Surgery consultation was requested regarding the cholangitis findings, and the patient underwent further etiological investigation and management concurrently with his thoracic stabilization. Following the initial trauma stabilization, the patient was admitted for observation. During the inpatient follow-up, MRCP imaging was performed, which confirmed the diagnosis of acute cholangitis secondary to choledocholithiasis. The biliary tree was notably affected, with marked dilatation of the intrahepatic bile ducts. The common bile duct (CBD) was significantly distended, measuring 16 mm at its maximum diameter, and contained multiple calculi (stones), providing a clear etiology for the clinical presentation. (Figure 2).

CONCLUSION: The use of Computed Tomography (CT) in emergency departments is of critical importance not only for diagnosing traumatic injuries but also for detecting incidental pathologies that may be life-threatening. Literature indicates that the prevalence of incidental findings in regional CT scans of trauma patients varies significantly, ranging from 30% to 50%(1,3,4,5). This rate is notably higher in the geriatric population, where chronic comorbidities and age-related degenerative changes increase the likelihood of discovering non-traumatic pathologies(6). While focusing on a patient's mechanical injuries in the ER, it is essential to maintain a systematic screening approach to ensure that concomitant pathologies are not overlooked. As demonstrated in this case, maintaining a high index of suspicion for incidental but urgent etiologies is vital for optimizing patient prognosis and preventing diagnostic delays in the trauma setting

Keywords: Computed Tomography, rib fracture, cholangitis

Figure 1.

Figure 2.

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8947

The Relationship Between Routine Laboratory Parameters, Clinical Outcomes, and Nursing Care in Patients with Acute Appendicitis: A Retrospective Study

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Introduction

Acute appendicitis is one of the most common causes encountered in emergency surgical practice worldwide and requires accurate diagnosis and prompt treatment [1]. Its incidence increases particularly after adolescence, along with a higher risk of complications [2]. In addition to clinical findings, laboratory parameters play an important role in the diagnosis of the disease and in the evaluation of prognosis [3].

Inflammatory markers obtained from laboratory tests may reflect the degree of inflammation in acute appendicitis [4]. Various studies have demonstrated that parameters such as the neutrophil-to-lymphocyte ratio (NLR), C-reactive protein (CRP), platelet-to-lymphocyte ratio (PLR), and albumin are associated with the severity of inflammation and the risk of complications [5].

C-reactive protein (CRP) is a highly sensitive marker in acute inflammatory processes and elevated CRP levels are generally associated with more severe inflammation and prolonged hospital stay [1]. It has also been reported that systemic inflammatory indices have not only diagnostic but also prognostic value and may influence the length of hospital stay in patients with acute appendicitis [2].

Routine laboratory parameters may assist in rapid clinical decision-making in the emergency department. However, studies systematically examining the relationship between these parameters and clinical outcomes—particularly the length of hospital stay—in patients with acute appendicitis are limited in the literature [3,4,5]. Therefore, the aim of this study is to evaluate the relationship between routine laboratory parameters and clinical outcomes in patients diagnosed with acute appendicitis.

In emergency departments, nurses play a crucial role in the initial assessment, monitoring, and preparation of patients with acute appendicitis for treatment [6]. The effective evaluation of routine laboratory data by nurses may contribute to the early recognition of changes in the patient's clinical condition and the timely implementation of appropriate nursing care interventions.

Materials and Methods

This study retrospectively analyzed patients diagnosed with appendicitis who presented to our emergency department within the past month. Patients under 18 years of age and those with incomplete data were excluded from the study.

Statistical Analysis

The statistical analysis of the data obtained in this study was performed using IBM SPSS Statistics 27 (Version 27; IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro–Wilk test. Categorical variables were presented as frequencies and percentages. For continuous variables, those with a parametric distribution were reported as mean ± standard deviation, while non-parametric variables were expressed as median and interquartile range (IQR) or minimum–maximum values. The independent samples t-test was used for parametric data, and the Mann–Whitney U test was applied for non-parametric data. Correlation analyses were conducted between continuous variables. A p-value of <0.05 was considered statistically significant.

Results

A total of 29 patients were included in the study, of whom 15 (51.7%) were male and 14 (48.3%) were female. The median age was 29 years (IQR: 22–45).

By gender:

- There were no significant differences in age ($p = 0.776$), CRP levels ($p = 0.793$), albumin levels ($p = 0.913$), or length of hospital stay ($p = 0.217$).
- Hemoglobin levels were significantly lower in female patients ($p = 0.001$).

Correlation analysis:

- A moderate positive correlation was observed between neutrophils and monocytes ($r = 0.476$, $p < 0.01$).
- A moderate negative correlation was detected between neutrophils and lymphocytes ($r = -0.455$, $p < 0.05$).
- A moderate negative correlation was observed between lymphocytes and length of hospital stay ($r = -0.476$, $p < 0.01$).
- A moderate negative correlation was found between platelets and CRP levels ($r = -0.520$, $p < 0.01$).
- A moderate negative correlation was observed between platelets and length of hospital stay ($r = -0.574$, $p < 0.01$).
- A moderate positive correlation was detected between CRP levels and length of hospital stay ($r = 0.598$, $p < 0.01$).

Discussion

Acute appendicitis is one of the most frequently encountered conditions in emergency departments worldwide [1]. In our study, the median age of the patients was 29 years (IQR: 22–45). Similarly, the literature reports that appendicitis is most common in the second and third decades of life [7]. This may be explained by the higher activity of lymphoid tissue in young adults and the more frequent occurrence of hyperplasia in the appendix lumen, which can lead to obstruction.

We found that hemoglobin levels in female patients were significantly lower than those in male patients. As demonstrated in the study examining the relationship between acute appendicitis and age, gender, and leukocyte values, hemoglobin levels can be influenced by demographic factors such as sex and may show significant differences between groups [8]. This finding can be explained by the physiologically lower hemoglobin levels typically observed in women.

We observed a moderate positive correlation between neutrophils and monocytes. Although a similar study did not directly examine the neutrophil–monocyte correlation, the concurrent increase of these cell lines during the inflammatory process is consistent with our findings and supports the notion that the systemic inflammatory response in acute appendicitis is mediated through multiple cellular components [9].

We observed a moderate negative correlation between neutrophils and lymphocytes. As emphasized in the study on the diagnostic value of the neutrophil-to-lymphocyte ratio (NLR) in acute appendicitis, NLR is an important marker for the diagnosis of the condition, with increased neutrophil counts accompanied by decreased lymphocyte levels reflecting the severity of inflammation [10]. In this context, the negative correlation observed in our study reflects the underlying mechanism of NLR elevation and is consistent with the existing literature.

A moderate negative correlation was observed between lymphocyte levels and length of hospital stay. A similar study emphasized the importance of inflammatory markers in the diagnosis of the disease and indirectly, in assessing its clinical severity [11]. Although that study did not directly examine the relationship between lymphocyte levels and hospital stay, it has been reported that lymphocyte counts may decrease with increasing inflammation, which could be associated with a more severe clinical course. This finding is consistent with our observation that lower lymphocyte levels are associated with prolonged hospital stay, suggesting that lymphocytes may serve as a prognostic marker.

We observed a moderate negative correlation between platelet counts and CRP levels. A similar study reported inverse relationships between platelet counts and inflammatory markers in patients with acute appendicitis [12]. Our findings support the notion that the combined evaluation of platelets and CRP may serve as a potential indicator for estimating the inflammatory burden and clinical severity of the disease.

A moderate negative correlation was observed between platelet counts and length of hospital stay. A similar study reported that lower platelet counts in patients with acute appendicitis may be associated with prolonged hospital stays [12]. This finding highlights the impact of the inflammatory response on hematologic parameters and suggests that platelet levels may provide prognostic information regarding the clinical course of the disease.

A moderate positive correlation was observed between CRP levels and length of hospital stay. Similarly, elevated CRP levels have been reported to be associated with more severe inflammation and prolonged hospital stays [13]. These findings support the use of CRP not only as a diagnostic marker but also as a prognostic indicator for assessing disease severity and predicting the duration of hospitalization.

Conclusion

The findings of this study indicate that routine laboratory parameters in patients with acute appendicitis are important not only for diagnostic purposes but also for providing prognostic information regarding disease severity and clinical course.

Key findings include:

Hemoglobin levels in female patients were significantly lower compared to males, consistent with sex-related physiological differences.

Correlations among neutrophils, monocytes, and lymphocytes demonstrate that the inflammatory response in acute appendicitis is mediated through multiple cellular components.

The neutrophil-to-lymphocyte ratio (NLR) and platelet counts were associated with the severity of inflammation and may serve as potential predictors of hospital stay.

Lymphocyte and platelet counts, as well as CRP levels, showed significant associations with the length of hospital stay; lower lymphocyte and platelet levels combined with higher CRP were related to prolonged hospitalization.

In conclusion, the combined evaluation of routine hematologic and inflammatory parameters may support clinical decision-making in patients with acute appendicitis, aid in predicting disease severity, and help optimize resource utilization. However, confirmation of these findings requires larger, prospective and multicenter studies. Furthermore, integrating these parameters into nursing care processes can provide early warning in patient monitoring, thereby enhancing the quality of care. Effective use of routine laboratory data by nurses may support a multidisciplinary approach to patient management and contribute to the improvement of clinical outcomes.

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9011 **A case of intramural hematoma of the ascending aorta**

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INTRODUCTION

Aortic Intramural Hematoma (IMH) is an important type of acute aortic syndrome that can be life-threatening. It presents with symptoms and a clinical course similar to aortic dissection. Unlike aortic dissection, it is characterized by the development of a hematoma within the media layer following hemorrhage, without the presence of an intimal tear or flap. It may cause a crescent-shaped or circumferential thickening appearance in the aortic wall. (1)

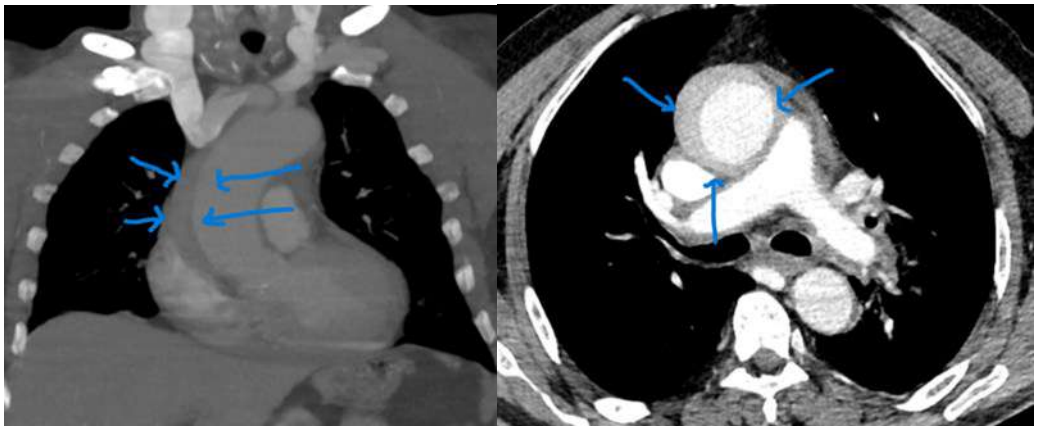
Aortic intramural hematoma can be detected more easily in recent years with the routine use of diagnostic methods such as Magnetic Resonance Imaging (MRI), Computed Tomography (CT), and Transesophageal Echocardiography (TEE). Since IMH located in the ascending aorta has very high mortality and morbidity, early surgical treatment is recommended. In contrast, in uncomplicated IMH located in the descending aorta, medical treatment with antihypertensive agents is generally recommended, and if pain and progression of the hematoma can be controlled in these typically elderly patients, mortality and morbidity are reported to be lower. (2)

In this case report, we presented a patient with ascending aortic intramural hematoma who was admitted to our emergency department.

CASE

A 76-year-old male patient was brought to the emergency department by the 112 emergency medical team after he lost consciousness while shaving at home. During history taking, it was learned that he had also been experiencing back pain recently as an additional symptom. On admission, the patient's vital signs were as follows: Glasgow Coma Scale (GCS) 15, temperature 36.7 °C, pulse 89 bpm, blood pressure 80/50 mmHg, oxygen saturation 93%, respiratory rate 16/min.

In laboratory tests, troponin was 17 and D-dimer was 853. Imaging studies revealed an ascending aortic diameter of 57 mm and an intramural hematoma in the ascending aorta. Tomography reports indicated that there was no dissection. The patient was consulted to cardiovascular surgery and was taken to emergency operation. The patient died during surgery.



CONCLUSION

In elderly patients presenting to the emergency department with syncope, the differential diagnosis should be kept broad, and aortic pathologies should also be considered.

KEY WORDS: Aorta, Syncope, Backache

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9250

From acute abdomen to digoxin intoxication: A case report

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Abstract

In this case, we aimed to present a patient who was initially transferred with a presumptive diagnosis of acute abdomen due to gastrointestinal complaints, but was ultimately diagnosed with digoxin intoxication because of severe bradycardia, hypotension, altered mental status, atrial fibrillation with a slow ventricular response on electrocardiography, and ST-segment changes. The patient presented to the emergency department of another hospital with nausea, vomiting, abdominal pain, and altered

consciousness. Based on the initial assessment, the patient was considered to have an acute abdomen and was referred to our emergency department. Past medical history revealed digoxin use for atrial fibrillation. According to information obtained from relatives, the patient had experienced influenza-like symptoms for one week and had been using antibiotics for two days. On arrival, the patient's blood pressure was 80/40 mmHg and heart rate was 33 beats/min, with diffuse abdominal tenderness on examination. The electrocardiography showed atrial fibrillation with a slow ventricular response, along with T-wave inversion and ST-segment depression. Atropine 1 mg IV bolus was administered. As bradycardia and hypotension persisted despite atropine, a transcutaneous external pacemaker was applied. The patient received 20 mg of DigiFab®, followed by a second dose due to ongoing bradycardia and hypotension. Subsequently, the patient's clinical status and electrocardiography findings improved, and the patient was discharged on the third day of intensive care unit admission. In patients presenting with an acute abdomen, careful evaluation of clinical findings and thorough review of medications are essential, as these may be critical for diagnosing potentially fatal digoxin intoxication.

Keywords: Abdominal pain; Digoxin intoxication; Transcutaneous external pacemaker.

Introduction

Digoxin is a medication derived from the leaves of the plant *Digitalis purpurea*, which has been used in medicine since the eras of Ancient Egypt and the Roman Empire. Its principal mechanism of action is inhibition of the Na^+/K^+-ATPase pump in cardiac myocytes, thereby increasing myocardial contractility; additionally, through its negative chronotropic effect, it reduces heart rate. Digoxin is a cardiac glycoside used primarily in the treatment of cardiac arrhythmias and various other cardiac conditions, and it may produce signs of toxicity when serum concentrations exceed 2 ng/mL. In toxic exposure, involvement may occur predominantly in the gastrointestinal (GI) system, as well as neurologic and cardiac systems (1).

Because digoxin has a narrow therapeutic index, careful attention to toxicity is particularly important in older adults. In addition to neurologic symptoms and cardiac conduction disturbances, non-specific GI symptoms may also occur and can mimic an acute abdomen. The most common manifestations are non-specific nausea, vomiting, and abdominal pain; therefore, it is frequently confused with acute abdominal conditions. In acute intoxications, concomitant medications—especially antibiotics—should be carefully reviewed (2).

Cardiac adverse effects include, most commonly, sinus bradycardia, as well as atrial fibrillation (AF) with a slow ventricular response, AF, ventricular ectopic beats, and atrioventricular (AV) blocks of any degree. Even at therapeutic digoxin levels, a characteristic hammock-shaped ST-segment depression beginning from the isoelectric line and causing

depression at the J point (the "Salvador Dalí moustache" appearance), T-wave flattening, and increased U-wave amplitude may be observed (3).

In this report, we aimed to present a patient who was transferred with an initial presumptive diagnosis of acute abdomen due to GI complaints, but was ultimately diagnosed with digoxin intoxication in the setting of severe bradycardia, hypotension, altered mental status, atrial fibrillation with a slow ventricular response on ECG, and ST-segment changes.

Case Report

An 82-year-old woman presented to the emergency department (ED) of another hospital with nausea, vomiting, abdominal pain, and altered mental status that began after taking her medications. She was assessed as having a suspected acute abdomen and was transferred to our ED.

Upon arrival at our ED, it was learned that she had been taking digoxin for atrial fibrillation (AF). According to relatives, she had experienced influenza-like symptoms for one week and had been using antibiotics for the past two days. Vital signs were as follows: blood pressure 80/40 mmHg, blood glucose 112 mg/dL, heart rate 33 beats/min, temperature 36.3°C, and oxygen saturation 93% on room air. Physical examination revealed diffuse abdominal tenderness; other system examinations were unremarkable. The ECG demonstrated AF with a slow ventricular response, accompanied by T-wave inversion and ST-segment depression (Figure 1). No pathological findings were identified in laboratory tests.

The patient was placed on continuous monitoring and intravenous isotonic saline was initiated. Atropine® 1 mg IV bolus was administered. Because bradycardia and hypotension did not improve despite atropine, a transcutaneous external pacemaker (TEP) was applied and set to demand mode at 60 beats/min with an output of 5 mA. Following external pacing, blood pressure was 80/50 mmHg and heart rate was 60 beats/min, and the patient's mental status improved.

Serum digoxin level was ordered; however, based on clinical and ECG findings, a diagnosis of digoxin intoxication was made without waiting for the result. After consultation with the poison center, digoxin-specific Fab fragments (DigiFab®) were requested. On hospital day 2, the serum digoxin level founded as 3.5 ng/mL. Until Fab therapy became available, the patient was monitored in the intensive care unit (ICU) with TEP support. She received 20 mg DigiFab®. As the patient remained bradycardic and hypotensive, a second dose of DigiFab® was administered. Subsequently, the patient's clinical status and ECG findings improved (Figure 2), and she was discharged on ICU day 3 with a recommendation for outpatient cardiology follow-up.

Discussion

Digoxin is a cardiac glycoside that requires careful monitoring for adverse effects and toxicity, particularly in older adults, due to its narrow therapeutic index (1). In clinical practice, digoxin toxicity may begin with non-specific GI complaints (nausea/vomiting, abdominal pain) and be mistaken for an acute abdomen; it can progress with neurologic symptoms (confusion/delirium) and cardiac conduction disturbances (2). In our case, the patient presented with nausea, vomiting, and abdominal pain.

In older patients, digoxin toxicity may not develop solely as a result of overdose. Concomitant infections, drug–drug interactions, changes in renal function, and electrolyte disturbances may increase the risk of toxicity. Notably, digoxin levels may rise to critical concentrations particularly with tetracyclines, clarithromycin, and erythromycin (2). In our patient, the recent initiation of a clarithromycin-containing antibiotic regimen and the temporal association of symptom onset after medication intake suggested a potential interaction; thus, the diagnosis was considered clinically before laboratory confirmation.

Cardiac manifestations of digoxin toxicity span a broad spectrum: sinus bradycardia, AF with a slow ventricular response, ventricular ectopy, and AV blocks of all degrees are among the potentially life-threatening arrhythmias (3). In this case, AF with a slow ventricular response on ECG, scooped ST-segment depression, severe atropine-refractory bradycardia, and concomitant resistant hypotension supported a “life-threatening” presentation of toxicity.

Initial management includes continuous monitoring, hemodynamic support, correction of electrolyte abnormalities, and arrhythmia management. Digoxin-specific Fab fragments (DigiFab®) constitute the cornerstone antidotal therapy, particularly in the presence of life-threatening arrhythmias or significant hemodynamic instability (4). In our patient, stabilization was achieved using external pacing after only transient/insufficient response to atropine, and Fab therapy was planned without waiting for serum level results due to clinical severity. The serum digoxin concentration of 3.5 ng/mL on day 2 provided biochemical support for toxicity.

However, it has been emphasized in the literature that serum digoxin level does not always correlate directly with clinical severity; therefore, decisions should be guided by an integrated assessment of clinical status, ECG findings, and hemodynamics (5). Digoxin intoxication cases presenting with abdominal pain have also been reported previously, highlighting the critical value of medication history and early ECG in acute abdomen-like presentations (6). In our case, although the patient was transferred with a presumptive diagnosis of acute abdomen, the presence of bradycardia and conduction abnormalities on ECG, together with a history of digoxin use, was decisive in establishing the correct diagnosis. As in all patients, this case again underscores that thorough history-taking and physical examination remain among the clinician’s most powerful diagnostic tools.

Conclusion

In patients presenting with an acute abdomen-like clinical picture, careful review of the patient's medication history and meticulous evaluation of clinical findings are essential, as these may be critical for diagnosing an underlying potentially fatal condition such as digoxin intoxication.

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Figure 1. Admission electrocardiogram (ECG).

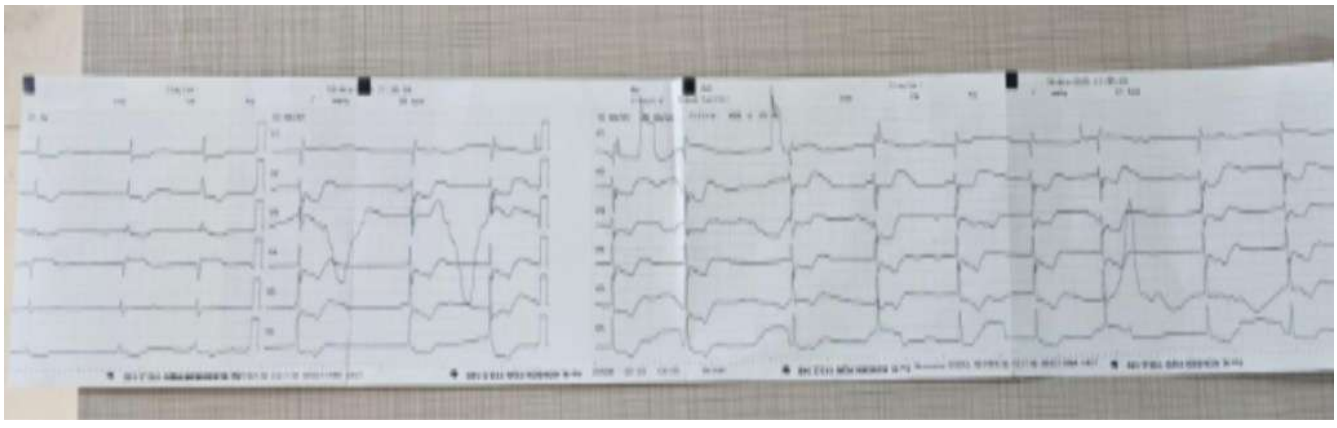
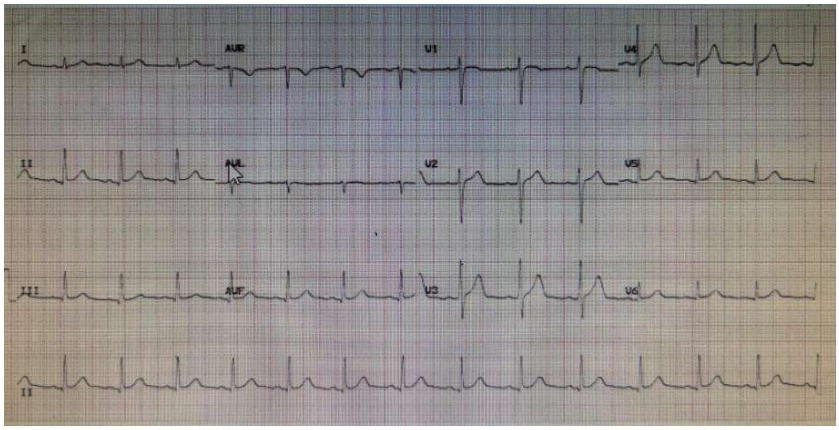


Figure 2. Discharge electrocardiogram (ECG).



9251

When Imaging Fails to Detect the Etiology of Paralysis: A Case of Adult SCIWORA in the Emergency Department

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Introduction

Spinal cord injuries are generally associated with vertebral fractures or dislocations. However, in SCIWORA, a neurological deficit is present that cannot be explained by conventional radiological methods (X-ray and CT) (1). Although it is typically seen in children due to the flexibility of the spine, it may also occur in adults because of degenerative changes or vascular causes. Early diagnosis is critical for preventing secondary injury (2).

Case

A 23-year-old male patient was brought to our emergency department following an in-vehicle traffic accident (rear-seat passenger, vehicle rollover down an embankment). He had no known chronic disease, medication use, or previous surgical history. On physical examination, his vital signs at presentation were as follows: blood pressure 110/70 mmHg, heart rate 72 beats/min, temperature 36.8°C, and SpO_2 98%. Glasgow Coma Scale (GCS) score was 15, and the patient was oriented and cooperative. No laceration, hematoma, or obvious external traumatic lesion was detected on the body.

Neurological examination revealed motor function of 0/5 (plegia) in all four extremities and loss of sensory function in all dermatomes (quadriparesis). Deep tendon reflexes were absent.

Laboratory and Imaging Findings

Laboratory investigations were within normal limits. Computed tomography scans of the entire spine (cervical, thoracic, and lumbar) and direct radiographs demonstrated no fracture, dislocation, or significant osseous pathology.

Clinical Course

Because of the severe neurological deficit despite the absence of radiological findings, SCIWORA was considered as the preliminary diagnosis, and spinal immobilization was maintained. The patient was consulted to the Department of Neurosurgery and admitted for further evaluation (spinal MRI) and non-surgical management/follow-up.

Discussion

SCIWORA is a condition that is difficult to diagnose and may be misleading for clinicians. In the literature, it has been explained by mechanisms such as transient stretching of the spinal canal or disruption of vascular supply to the spinal cord (1,3). In our case, despite a high-energy trauma mechanism, the absence of external lesions and a completely normal CT scan could have been misleading. However, the quadriparesis identified on physical examination guided the diagnosis beyond the radiological findings. In these patients, a “normal CT” finding is not a sufficient criterion for mobilizing the patient (4).

Conclusion

In trauma patients, radiological examinations should not take precedence over physical examination findings. SCIWORA must always be considered in patients with neurological deficits despite normal imaging findings, and these patients should be followed under strict immobilization.

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Keywords:SCIWORA, Spinal Cord Injury, Trauma, Emergency Department.

9287

A case of isolated headache and possible subarachnoid hemorrhage: Sinus venous thrombosis

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Abstract

This case presentation aims to highlight the necessity of considering sinus venous thrombosis in the differential diagnosis of headache in emergency medicine practice. A 60-year-old female patient presented to the emergency department with a one-week history of headache that had worsened over the past two days. The patient reported no improvement in her headache despite the use of non-steroidal anti-inflammatory drugs. Vital signs were stable on presentation. . His pupils were isochoric, and light reflexes were bilaterally positive. Neurological examination revealed no lateralised findings. Cerebellar tests were normal. Cranial nerve examination was normal. Pathological reflexes were negative. Other systemic examinations were normal. Laboratory values showed no significant pathological findings. Non-contrast cranial tomography revealed a hyperdense appearance in the right transverse sinus tract. The tomography showed a suspicious image for bleeding. Magnetic resonance venography was performed. The magnetic resonance venography revealed loss of flow consistent with acute thrombosis in the right transverse sinus, sigmoid sinus, and right internal jugular vein. The patient was admitted to the neurology ward with a diagnosis of sinus vein thrombosis. Low-molecular-weight heparin therapy was initiated. No neurological deterioration was observed in the patient during twelve days of ward follow-up. The patient was discharged on oral anticoagulant therapy. Sinus venous thrombosis should be considered in patients with sudden, severe headache and a normal neurological examination.

Keywords: Headache, Magnetic resonance, Sinus vein thrombosis.

Introduction

Sinus vein thrombosis (SVT) is a rare but potentially life-threatening cerebrovascular disease characterised by thrombotic occlusion of cerebral veins and dural sinuses (1). It accounts for approximately 0.5–1% of all strokes and is more common in young adults (1,2). The clinical presentation is highly variable, ranging from isolated headache to focal neurological deficits and altered consciousness (1). Headache is the most common symptom of SVT and may be the sole finding in some cases (2,3). In some cases, it may mimic subarachnoid hemorrhage (SAH) with sudden onset or severe headache (2,4). In patients

presenting to the emergency department with sudden and severe headache, SAH is first ruled out, but SVT is often overlooked in the differential diagnosis. The treatment approaches for these two clinical conditions are fundamentally different. While anticoagulation is contraindicated in SAH, early anticoagulation therapy in SVT significantly improves prognosis (5-7). Therefore, accurate and timely diagnosis is vital to clinical outcomes. This case presentation aims to highlight the necessity of considering sinus venous thrombosis in the differential diagnosis of headache in emergency medicine practice.

Case Report

A 60-year-old female patient presented to the emergency department with a one-week history of headache that had worsened over the past two days. The patient reported no improvement in her headache despite the use of non-steroidal anti-inflammatory drugs. Her medical history included diabetes mellitus and asthma. She had no known drug allergies. There was no history of trauma. There was no cardiac history. She was not taking any anticoagulant medication. Vital signs were stable on presentation. Blood pressure was 130/70, pulse was 80, oxygen saturation was 98, and temperature was 36,5°C. The patient was alert, cooperative, and oriented. His pupils were isocoric, and light reflexes were bilaterally positive. Neurological examination revealed no lateralised findings. Cerebellar tests were normal. Cranial nerve examination was normal. Pathological reflexes were negative. Other systemic examinations were normal. Laboratory values showed no significant pathological findings. Non-contrast cranial computed tomography (CT) revealed a hyperdense appearance in the right transverse sinus tract (Figure 1). The tomography showed a suspicious image for bleeding. Magnetic resonance (MR) venography was performed. The MR venography revealed loss of flow consistent with acute thrombosis in the right transverse sinus, sigmoid sinus, and right internal jugular vein. (Figure 2). The patient was admitted to the neurology ward with a diagnosis of sinus vein thrombosis. Low-molecular-weight heparin therapy was initiated. No neurological deterioration was observed in the patient during twelve days of ward follow-up. The patient was discharged on oral anticoagulant therapy.

Discussion

SVT can present with numerous symptoms and findings (1,8). Headache is the most common symptom, and a prospective study has shown that headache is the sole finding in a significant proportion of patients (2). Similarly, a series of 17 cases reported that SVT can present with headache alone (3). In another series of 27 cases, headache was reported in 81.5% of cases (9). These data indicate that an isolated headache is an important presenting symptom for SVT. Subarachnoid haemorrhage should be ruled out in patients presenting with sudden and severe headache. Patients presenting with thunderclap headache should also be considered for sinus vein thrombosis in the differential diagnosis (4). While anticoagulation is contraindicated in the treatment of SAH, early anticoagulation improves prognosis in SVT

treatment, making the distinction between these two clinical conditions important (5). It has been reported that cranial CT may be normal in approximately 25% of cases (9). Clinical suspicion is of great importance for early diagnosis of SVT, and MR imaging (MRI) and MR venography (MRV) are considered the gold standard for diagnosis (1). Therefore, if clinical suspicion persists, evaluation with MRI and MRV should be performed. In our case, the diagnosis was confirmed by MRV, and an approach consistent with the literature was followed. In terms of treatment, low-molecular-weight heparin administration and concomitant oral anticoagulation are widely recommended in the literature (5-7). In our patient, clinical improvement was observed with early initiation of anticoagulation therapy.

Conclusion

SVT should be considered in patients with sudden, severe headache and a normal neurological examination.

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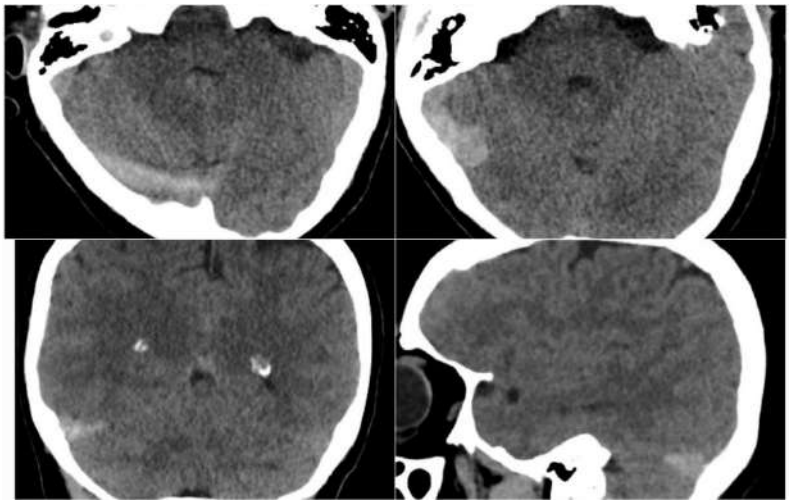

Figure 1. Brain computed tomography image of our patient

Figure 2. Magnetic resonance venography and brain image of our patient.

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Emergency management of pisiform dislocation: A case report

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ABSTRACT

This case report aims to emphasize that rare carpal injuries may occur in wrist trauma and that careful patient evaluation is essential to avoid missing such injuries. A 16-year-old male presented to the emergency department with pain and swelling in his right wrist after falling onto his hand. Physical examination revealed tenderness in the right wrist, while the neurovascular examination was normal. Two-view radiographs demonstrated pisiform bone subluxation and a non-displaced fracture of the scaphoid bone. Closed reduction was performed in the emergency department, followed by immobilization with a short arm splint. The patient was discharged without complications. Pisiform subluxation should be considered in the differential diagnosis of trauma patients presenting with wrist tenderness and swelling after a fall. Careful physical examination and radiological evaluation are crucial to prevent overlooking pisiform subluxation and concomitant injuries.

Keywords: Dislocation, Emergency, Pisiform.

INTRODUCTION

The pisiform bone is a sesamoid bone that develops within the tendon of the flexor carpi ulnaris muscle (1). Pisiform dislocation is a rare injury that is difficult to diagnose early and has been infrequently described in the literature, which increases the likelihood of missed diagnosis by healthcare providers (1,2). Although dislocations of carpal bones are relatively more common, dislocation of the pisiform bone is extremely rare, and there is still no consensus regarding the optimal treatment strategy (3). In this case report, we aim to highlight that rare carpal injuries may occur in wrist trauma and that careful evaluation of patients is necessary to prevent missed diagnoses.

CASE REPORT

A 16-year-old male with no known comorbidities presented to the emergency department with complaints of pain and swelling in his right wrist after falling onto his outstretched hand. On admission, his vital signs were as follows: body temperature 36.8°C, heart rate 92 beats/min, blood pressure 120/80 mmHg, respiratory rate 16 breaths/min, and oxygen saturation 99%.Physical examination revealed tenderness and painful joint

movements in the right wrist. The range of motion was full. Peripheral pulses were palpable, capillary refill time was normal, and no sensory or neurological deficits were detected.Two-view radiographs of the hand and wrist were obtained. Imaging revealed pisiform subluxation and a non-displaced fracture of the scaphoid bone (Figure 1). Closed reduction of the dislocation was successfully performed in the emergency department (Figure 2). No complications occurred following the procedure. The patient's pain improved and the swelling decreased significantly. The wrist was immobilized with a short arm splint, and the patient was discharged with a recommendation for follow-up in the orthopedic outpatient clinic.

DISCUSSION

Pisiform dislocation is a rare injury among wrist traumas. The pisiform bone is a sesamoid bone located within the tendon of the flexor carpi ulnaris muscle and contributes to the medial stability of the wrist through the pisohamate and pisometacarpal ligaments (4). Pisiform dislocation may occur due to a high-energy impact when the wrist is in extension or from a sudden traction force exerted by the flexor carpi ulnaris muscle. In our case, the 16-year-old patient developed a pisiform dislocation after falling onto his open right hand.

The literature reports that isolated pisiform dislocations are frequently misdiagnosed as wrist sprains and that delayed diagnosis may lead to persistent pain (2,5). Concomitant carpal bone injuries associated with pisiform subluxation are rare; however, they may occur due to complex trauma mechanisms. The presence of a concurrent scaphoid fracture suggests that the traumatic force was transmitted to both the ulnar and radial columns of the wrist. Triquetral fractures accompanying pisiform subluxation have also been reported in the literature when the traumatic force is transmitted unilaterally (6). Similarly, in our case, pisiform subluxation was accompanied by a non-displaced scaphoid fracture.

Closed reduction and immobilization are usually sufficient in the acute phase. Early intervention is important to prevent chronic instability and functional impairment. Surgical treatment may be required if reduction fails or if a locked dislocation develops (1).

In our case, closed reduction was successfully performed in the emergency department, followed by immobilization with a short arm splint, and the patient was discharged with a recommendation for orthopedic outpatient follow-up.

CONCLUSION

Pisiform subluxation should be considered in the differential diagnosis of trauma patients presenting with wrist tenderness and swelling after a fall. Careful physical examination and radiological evaluation are essential to prevent missing pisiform subluxation and associated injuries.

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Figure 1. Pisiform subluxation at initial presentation

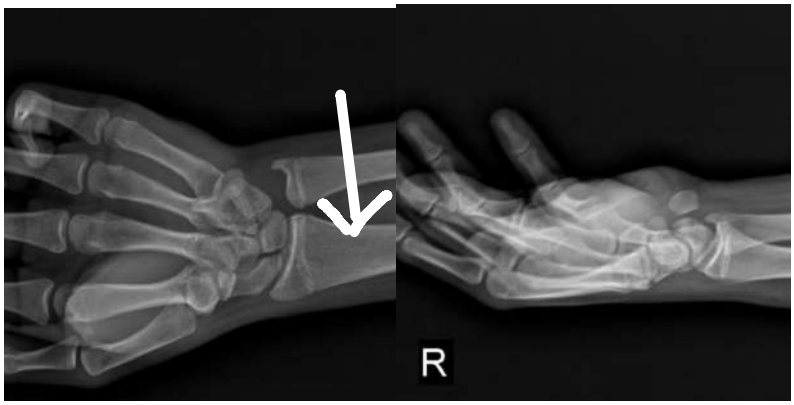

Figure 2. After closed reduction of the pisiform dislocation

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Predictors of Hospital Length of Stay in Patients with Pneumothorax: A Retrospective Study

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INTRODUCTION

Pneumothorax is a clinical condition characterized by the accumulation of air in the pleural space, resulting in partial or complete collapse of the lung, and may require urgent intervention. It may occur due to spontaneous, traumatic, or iatrogenic causes and presents with a wide clinical spectrum ranging from mild dyspnea to life-threatening conditions such as tension pneumothorax (1).

Length of hospital stay (LOS) is a key indicator of healthcare quality and cost-effectiveness. Identifying the factors that influence LOS in patients with pneumothorax enables early recognition of high-risk patients and optimization of treatment strategies (2). This study aims to evaluate the parameters affecting LOS in hospitalized patients with pneumothorax in light of the current literature.

MATERIALS AND METHODS

This study was conducted on the data of 90 patients who presented to the emergency department of a tertiary care center between 2024 and 2025 and were hospitalized in the thoracic surgery clinic with a diagnosis of pneumothorax. Patient characteristics including age, sex, presenting symptoms (dyspnea, chest pain, syncope, etc.), comorbidities, laterality, and type of pneumothorax (spontaneous, traumatic, iatrogenic) were recorded. In addition, the treatments administered (tube thoracostomy, surgery) and complications observed (infection, subcutaneous emphysema, prolonged air leak) were documented.

Statistical analyses were performed using SPSS software. The Mann–Whitney U test and Kruskal–Wallis test were used for data that did not follow a normal distribution. A multivariable linear regression model including age, sex, and clinical variables was constructed to assess independent predictors of LOS. A p-value of <0.05 was considered statistically significant

RESULTS

The baseline characteristics and analysis results of the 90 patients included in the study are presented below. The mean age of the patients was 54.26±20.81 years, with a median age of 58.5 (19–99) years. Of the patients, 70 (77.8%) were male and 20 (22.2%) were female. The most common presenting symptom was dyspnea (40.1%), while traumatic pneumothorax (61.1%) was the leading etiological type. Malignancy was present in 18.9% of the patients. Tube thoracostomy was performed in 72.2% of cases (Table-1). LOS ranged from 1 to 23 days, with a mean of 6.71±4.35 days and a median of 6.0 days. Patients who developed complications, particularly subcutaneous emphysema and infection, had significantly longer LOS ($p=0.044$ and $p<0.001$, respectively). LOS was also significantly prolonged in patients who underwent tube thoracostomy, had prolonged air leak, presented with dyspnea, or experienced recurrent pneumothorax (all $p<0.05$). In multivariable linear regression analysis, the model was statistically significant ($R^2=0.624$), explaining 62.4% of the variance in LOS. Independent predictors of increased LOS included the presence of complications ($\beta=0.404$; $p<0.001$), tube thoracostomy ($\beta=0.255$; $p<0.001$), prolonged air leak ($\beta=0.294$; $p<0.001$), and recurrence of pneumothorax ($\beta=0.236$; $p=0.003$). Age, sex, and presenting symptoms were not significantly associated with LOS ($p>0.05$).

DISCUSSION AND CONCLUSION

Our study demonstrated that prolonged air leak (PAL) and the development of infection are the two most critical factors contributing to increased length of hospital stay in the management of pneumothorax. In the literature, the development of PAL has been associated with conditions that impair lung parenchymal integrity, such as the presence of large bullae, low body mass index, active smoking, and underlying chronic obstructive pulmonary disease (2–4). Our findings also confirm that these complications significantly prolong hospitalization ($p<0.001$).

Regarding infection (e.g., empyema, pneumonia), the duration of chest tube placement is the most important risk factor. Additionally, overcrowding and procedural conditions in emergency departments may occasionally compromise optimal sterilization, thereby increasing the risk of infection. In traumatic cases, the presence of retained hemothorax also provides a favorable environment for bacterial growth (3).

Current approaches to antibiotic prophylaxis do not recommend routine use in spontaneous pneumothorax; however, in penetrating chest trauma, administration of first-generation cephalosporins within the first 24 hours has been shown to reduce the risk of empyema (1).

In patients hospitalized for pneumothorax, length of hospital stay is determined more by complications arising during the clinical course and interventional procedures (such as

tube thoracostomy) rather than demographic characteristics. To reduce LOS, early aggressive and multidisciplinary management is recommended, particularly in high-risk patients (elderly, traumatic cases, and those with a history of recurrence).

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Table 1. Basic Clinical and Demographic Characteristics of Patients

Variable	Category	n	%
Presenting Complaint	Dyspnea	36	40.1
	Pleuritic chest pain	21	23.3
	Cough	18	20.0
	Back pain	13	14.4
	Syncope	2	2.2
Comorbidities	None	49	54.4
	Malignancy	17	18.9
	Rib fracture	15	16.7
	Bulla	6	6.7
	Hemothorax	4	4.4
	Empyema	2	2.2
	Other (unspecified)	5	5.5
Type of Pneumothorax	Iatrogenic	20	22.2
	Spontaneous	55	61.1
	Traumatic	15	16.7
Side	Left	37	41.1
	Right	53	58.9
Location on CT	Apical	19	21.1
	Basal	23	25.6
	Lateral	31	34.4
	Total	17	18.9
Complications	None	79	87.8
	Infection	7	7.8
	Subcutaneous emphysema	4	4.4
Tube Thoracostomy	No	25	27.8
	Yes	65	72.2
Prolonged Air Leak	No	84	93.3
	Yes	6	6.7
Surgery due to Prolonged Air Leak	No	84	93.3
	Yes	6	6.7

Variable	Category	n	%
Recurrent Pneumothorax	No	80	88.9
	Second episode	9	10.0
	≥3 episodes	1	1.1

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Association Between Platelet-to-Lymphocyte Ratio and Mortality in Patients with Urosepsis

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Abstract

Objective: Urosepsis is a serious clinical condition associated with high mortality in patients requiring intensive care. In this study, we aimed to evaluate the relationship of the platelet/lymphocyte ratio (PLR), lymphocyte, and platelet values with mortality in urosepsis patients admitted to the intensive care unit from the emergency department.

Methods: This retrospective observational study included patients who presented to the emergency department between August 10, 2025, and January 10, 2026, and were admitted to the intensive care unit with a diagnosis of urosepsis. A total of 34 patients meeting the inclusion and exclusion criteria were enrolled in the study. Demographic characteristics, as well as lymphocyte ($\times 10^9$/L), platelet ($\times 10^9$/L), and PLR values at the time of admission, were analyzed. Statistical analyses were performed using SPSS 27 software.

Results: The mean age of the patients included in the study was calculated as 59.35±21.91 years, and the mortality rate was found to be 17.6%. PLR values were determined to be significantly higher in the deceased group compared to the surviving group ($p<0.001$). Although lymphocyte levels were lower in the deceased group, no statistical significance was found ($p=0.052$). No significant relationship was found between platelet values and mortality ($p=0.206$).

Conclusion: It can be considered that PLR may be associated with mortality in urosepsis patients and may serve as a practical marker for early risk stratification. However, this finding needs to be supported by larger, prospective studies.

Keywords: Urosepsis, platelet/lymphocyte ratio, PLR, mortality, sepsis, inflammation

INTRODUCTION

Urosepsis is a life-threatening clinical condition characterized by organ dysfunction resulting from a dysregulated host response to infections originating from the urinary tract [1]. As a significant component of the sepsis spectrum, urosepsis accounts for approximately 20–40% of all sepsis cases and is more frequently observed, particularly in the elderly population [2].

Urinary tract infections are among the most common bacterial infections worldwide and can rapidly progress to sepsis in complicated cases [3]. Urinary obstruction, catheter use, and accompanying comorbid diseases have been identified as significant risk factors for the development of urosepsis [4].

Gram-negative bacteria, primarily Escherichia coli, are most frequently responsible for the etiology of urosepsis [1]. Mortality rates increase significantly in patients requiring intensive care and can reach levels of 17–30% [5]. This situation underscores the importance of early diagnosis and prognosis determination in clinical management.

Sepsis continues to be a major cause of mortality on a global scale, leading to millions of deaths each year [6]. Therefore, identifying rapid and easily accessible biomarkers that can predict prognosis in urosepsis patients is of critical importance for clinical decision-making processes. In recent years, it has been shown that hematological parameters, particularly lymphocytes, platelets, and their derivative indices (e.g., platelet/lymphocyte ratio, PLR), may have prognostic value [5].

In this study, we aimed to evaluate the relationship of age, gender, lymphocyte, platelet, and PLR values with mortality in urosepsis patients admitted to the intensive care unit from the emergency department.

MATERIALS AND METHODS

Study Design and Patient Population

This study was planned as a retrospective observational study including patients who presented to the Emergency Department of Malatya Training and Research Hospital between August 10, 2025, and January 10, 2026, and were admitted to the intensive care unit with a diagnosis of urosepsis.

Within the scope of the study, a total of 41 patients were evaluated via the hospital information management system. Thirty-four patients meeting the inclusion and exclusion criteria were included in the study. Patients with missing data and cases whose diagnosis could not be clearly confirmed were excluded.

Inclusion and Exclusion Criteria

Patients aged 18 years and older who presented to the emergency department between August 10, 2025, and January 10, 2026, had a confirmed diagnosis of urosepsis based on clinical, laboratory, and/or imaging findings, and were admitted to the intensive care unit were included in the study.

The diagnosis of urosepsis was made by evaluating the presence of clinical findings related to urinary tract infection together with signs of systemic inflammatory response and organ dysfunction.

Excluded from the study were:

Patients with missing clinical or laboratory data,

Cases where lymphocyte or platelet values could not be obtained,

Patients with a history of hematological disease, active malignancy, or immunosuppressive therapy,

And patients whose diagnosis of urosepsis could not be clearly confirmed.

In line with these criteria, 34 of the 41 evaluated patients were included in the study.

Data Collection and Variables

Demographic characteristics (age, gender) and clinical outcomes (mortality) of the patients were recorded. Lymphocyte count and platelet values at the time of admission were examined as laboratory parameters.

Lymphocyte count was evaluated in units of $\times 10^9$/L, and platelet count in $\times 10^9$/L. The platelet/lymphocyte ratio (PLR) was calculated by dividing the platelet count by the lymphocyte count.

PLR is an easily accessible hematological index reflecting systemic inflammation, which has emerged as a prognostic marker in critically ill patient populations in recent years [7,8].

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics.

The conformity of continuous variables to normal distribution was assessed using Kolmogorov–Smirnov and Shapiro–Wilk tests. It was determined that age, lymphocyte, and PLR values showed a normal distribution, while platelet values did not show a normal distribution.

Data with normal distribution were expressed as mean ± standard deviation, and data without normal distribution were expressed as median and interquartile range (IQR, 25%–75%).

For comparisons between groups, the independent samples t-test was used for variables with normal distribution, and the Mann–Whitney U test was used for variables without normal distribution.

Categorical variables were expressed as frequencies and percentages. A p-value of <0.05 was considered statistically significant.

RESULTS

The mean age of the total 34 patients included in the study was calculated as 59.35±21.91 years. We determined that 58.8% (n=20) of the patients were male and 41.2% (n=14) were female. The mortality rate was found to be 17.6% (n=6).

When we examined the laboratory parameters: the mean platelet (PLT) count was calculated as 284.70±108.38 $\times 10^9$/L, the mean lymphocyte count was 1.46±0.65 $\times 10^9$/L, and the mean platelet/lymphocyte ratio (PLR) was calculated as 214.69±67.47. Median values were found as 265.00 $\times 10^9$/L for platelets, 1.49 $\times 10^9$/L for lymphocytes, and 198.04 for PLR, respectively.

We assessed the normal distribution of data using Kolmogorov–Smirnov and Shapiro–Wilk tests. We found that age, lymphocyte, and PLR variables showed a normal distribution, while platelet values did not show a normal distribution.

When we compared the groups according to mortality: the mean age of deceased patients was calculated as 70.50±12.83 years, and the mean age of surviving patients was 56.96±22.87 years. No statistically significant difference was found between the groups in terms of age (p=0.173).

When we compared lymphocyte values: the mean lymphocyte level in the deceased group was 0.98±0.76 ×10⁹/L, and in the surviving group, it was 1.56±0.60 ×10⁹/L. Although the lymphocyte level was lower in the deceased group, this difference was not found to be statistically significant (p=0.052).

When we examined PLR values: the mean PLR value in the deceased group was calculated as 300.03±82.29, and in the surviving group as 196.40±48.42. The PLR value was determined to be statistically significantly higher in the deceased group (p<0.001).

In the analysis performed regarding platelet values: the mean rank for the deceased group was calculated as 12.83, and for the surviving group as 18.50. No statistically significant difference was found between the groups in terms of platelet values (p=0.206).

DISCUSSION

Urosepsis is the most severe clinical form of urinary tract infections, characterized by systemic inflammatory response and organ dysfunction, and is associated with significant mortality and morbidity, especially in patients requiring intensive care [1,2]. In recent years, studies aiming to identify simple and accessible biomarkers that can predict prognosis in urosepsis patients have been increasing.

In this study, we evaluated the relationship between demographic characteristics, hematological parameters, and mortality in urosepsis patients admitted to the intensive care unit from the emergency department. We found the mortality rate in our study to be 17.6%. Although the mean age was higher in the deceased group, we observed that this difference was not statistically significant. We noted that lymphocyte levels were lower in the deceased group, but this difference did not reach the threshold for statistical significance. In contrast, we determined that PLR values were significantly higher in the deceased group and showed a significant relationship with mortality. No significant difference was found between the groups in terms of platelet values.

The literature reports that mortality rates in urosepsis and sepsis patients vary within a wide range, and these rates increase markedly, especially in patients requiring intensive care [2,4]. It can be said that our findings are consistent with this range. Although the effect of age on sepsis mortality has been demonstrated in many studies, some studies have reported results that did not reach statistical significance, similar to our findings [9]. This situation is thought to be possibly related to patient numbers, population characteristics, and the distribution of comorbidities.

Our findings regarding lymphocyte levels appear to be partially consistent with studies reporting that lymphopenia may have prognostic significance as a reflection of immune dysfunction in sepsis [10]. However, the failure to reach statistical significance in our study may be related to the limited sample size or patient heterogeneity.

PLR, on the other hand, has attracted attention in recent years as a marker reflecting systemic inflammation and has been shown to have prognostic value in different patient groups [8]. Studies conducted in the sepsis population also report that high PLR values may be associated with poor prognosis [11]. In our study, the finding that PLR was significantly higher in the deceased group suggests that this parameter could be used as a prognostic marker in urosepsis patients.

Our failure to find a significant relationship between platelet count and mortality suggests that platelets alone may be limited as a prognostic marker, but may yield more meaningful results when evaluated together with other inflammatory parameters [11].

When the findings of our study are evaluated together, it can be said that easily calculable hematological indices, particularly PLR, may be useful for early risk stratification in urosepsis patients, but it may be more appropriate to evaluate these parameters in conjunction with clinical and other laboratory data rather than using them alone.

CONCLUSION

In this study, we found that PLR was significantly associated with mortality in patients hospitalized in the intensive care unit due to urosepsis, whereas lymphocyte and platelet values alone did not show a significant relationship. Our findings suggest that PLR may be a potential marker for early risk stratification in urosepsis patients. However, rather than using this parameter alone in clinical decision-making processes, evaluating it together with other clinical and laboratory findings may be more appropriate.

LIMITATIONS

The main limitations of our study are its retrospective design, being conducted at a single center, and its limited sample size. Furthermore, the fact that only laboratory parameters at the time of admission were evaluated and dynamic changes could not be analyzed are among the other important limitations of the study.

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Not Just an Adult Emergency: Traumatic Penile Fracture in a 4-Year-Old Boy

A Rare Genital Injury Secondary to Domestic Trauma: Pediatric Penile Fracture

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Introduction

Although penile trauma and traumatic penile fracture (disruption of the tunica albuginea integrity) are more commonly described in the adult population, they may also occur in pediatric patients and may require early diagnosis and prompt surgical intervention. Such injuries are generally associated with sexual activity or direct trauma; however, external devices or instrument-related injuries may rarely constitute the etiology (1,2). Ultrasonography (USG) is a rapid, non-invasive imaging modality that provides valuable information in the evaluation of penile trauma; nevertheless, surgical decision-making is primarily based on the integration of clinical findings and imaging results (3).

Case Presentation

Patient: A 4-year-old boy. Family history and mechanism of injury: According to the family, while the child was playing at home, a massage device present in the household was applied to the penis. The family recognized the incident and immediately brought the patient to the emergency department.

Clinical findings on admission: Physical examination revealed diffuse penile edema, extensive subcutaneous hematoma extending toward the superior aspect of the scrotum, and partial penile deformity. The family reported that the child had urinated forcefully approximately 1–2 hours before presentation and that no gross hematuria was observed. Due to the patient's age, subjective pain assessment could not be reliably obtained.

Imaging findings: Superficial scrotal and penile ultrasonography demonstrated a subcutaneous hematoma between the superior scrotal region and the penile root, with tissue thickness reaching approximately 12 mm. Edema and localized swelling were present within the suprapubic skin and subcutaneous tissues. Superficial penile ultrasonography further revealed disruption of the full-thickness continuity of the tunica albuginea in the midshaft region of the penis, raising suspicion for complete traumatic penile fracture.

Laboratory findings: Blood laboratory investigations obtained in the emergency department were unremarkable. No gross hematuria or accompanying urological symptoms were identified.

Management: A pediatric surgery consultation was obtained. The patient was admitted to the ward for surgical intervention and close follow-up. Two clinical examination photographs demonstrating the patient's condition were included in the report.

Case Photographs

Figures 1 and 2 demonstrate the patient's clinical examination findings. The images were included in the case presentation with parental consent.

Figure 1. Diffuse penile edema and subcutaneous hematoma extending along the scrotal border.

Figure 2. Close-up view of the glans penis demonstrating skin edema, hematoma, and deformity.

Discussion

This case demonstrates penile trauma resulting from an unusual mechanism in the pediatric age group. Disruption of the full-thickness integrity of the tunica albuginea (penile fracture) generally requires open surgical repair, and early surgical intervention improves both functional and cosmetic outcomes (2,4). Although ultrasonography is useful for identifying hematoma localization, subcutaneous edema, and potential tunical defects, advanced imaging modalities such as magnetic resonance imaging (MRI) or retrograde urethrography may occasionally be necessary to evaluate associated prostatic or urethral injuries (3,5).

In pediatric patients, the mechanism of trauma, physical examination findings, and family history play critical roles in the surgical decision-making process. The absence of gross hematuria decreases the likelihood of urethral injury; however, urinary patterns and possible dislocation should still be carefully evaluated during follow-up. In the present case, the decision by pediatric surgery to hospitalize the patient for operative management and observation was appropriate because of suspected tunical disruption and the risk of progressive hematoma and edema.

This case highlights the importance of early evaluation, appropriate imaging, and a multidisciplinary approach in pediatric genital trauma associated with domestic devices or household instruments.

Conclusion

Although penile trauma is rare in pediatric patients, it may result in significant functional and cosmetic consequences. Timely presentation facilitated by family awareness, appropriate imaging to determine the need for surgery, and simultaneous management involving pediatric surgery and urology are of critical importance.

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Keywords: *Penile trauma, pediatrics, hematoma, tunica albuginea*

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Acute bilateral anterior shoulder dislocation developing during sports activity

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Abstract

In this case report, we aimed to describe the diagnosis and management of bilateral shoulder dislocations that occurred during resistance exercise in a gymnasium. A male patient in his thirties presented to the emergency department with sudden pain and loss of movement in both shoulders as a result of the weight accelerating upwards uncontrollably during a lat pulldown exercise in the gymnasium. A prominent epaulet sign was observed in both shoulders. The loss of deltoid contour and the prominence of the acromion were remarkable. There was a severe limitation in both active and passive ranges of motion. Distal pulses were palpable, and motor and sensory examination findings were within normal limits; axillary nerve function was also preserved. As a result of clinical and radiological evaluation, the patient was diagnosed with bilateral anterior shoulder dislocation. Under monitoring, appropriate sedation and analgesia were administered. Bilateral closed reduction manoeuvres were performed successfully. Post-reduction pain decreased significantly, and the range of motion was regained. The post-reduction neurovascular examination was evaluated as normal. No complications developed. Shoulder immobilization was recommended for the patient, and he was referred to the orthopaedics outpatient clinic for follow-up. It should be kept in mind that sudden overloads during resistance exercises can, although rare, lead to bilateral anterior shoulder dislocation.

Key words: Bilateral shoulder dislocation, Anterior dislocation, Sports injuries.

Introduction

The shoulder joint is the most mobile joint in the body, and this mobility comes at the expense of stability. Ninety-five percent of shoulder dislocations are in the anterior direction (1). Bilateral dislocations are rare and mostly posterior (2). Bilateral anterior dislocation is an even rarer condition (3,4). In this case report, we aimed to describe the diagnosis and management of bilateral shoulder dislocations that occurred during resistance exercise in a gymnasium.

Case Report

A male patient in his thirties presented to the emergency department with sudden pain and loss of movement in both shoulders as a result of the weight accelerating upwards uncontrollably during a lat pulldown exercise in the gymnasium. A prominent epaulet sign was observed in both shoulders (Figure 1). The loss of deltoid contour and the prominence of the acromion were remarkable. There was a severe limitation in both active and passive ranges of motion. Distal pulses were palpable, and motor and sensory examination findings were within normal limits; axillary nerve function was also preserved. As a result of clinical and radiological evaluation, the patient was diagnosed with bilateral anterior shoulder dislocation (Figure 2). Under monitoring, appropriate sedation and analgesia were administered. Bilateral closed reduction manoeuvres were performed successfully (Figure 3). Post-reduction pain decreased significantly, and the range of motion was regained. The post-reduction neurovascular examination was evaluated as normal. No complications developed. Shoulder immobilization was recommended for the patient, and he was referred to the orthopaedics outpatient clinic for follow-up.

Discussion

Shoulder dislocations are mostly unilateral (1); cases with bilateral dislocation are generally of the posterior type due to seizures or electric shock (2). Bilateral anterior dislocation, on the other hand, is associated with synchronized traumatic strain (3,4). In this case, the mechanism was evaluated as the shoulders being forced into sudden hyperextension and abduction. This situation may have placed excessive load on the anterior capsule and ligament complex, causing the humeral head to displace anteriorly. The fundamental approach in the emergency department requires a detailed neurovascular evaluation prior to reduction (5,6). Appropriate analgesia and sedation must be provided before the procedure, and traumatic and forceful manoeuvres should be avoided. Following reduction, the neurovascular examination must be repeated, and control radiographs should be obtained to exclude additional pathology or associated fractures. Early and accurate intervention is critical to

reducing potential complications and the risk of chronic instability that may develop in the subsequent period.

Conclusion

It should be kept in mind that sudden overloads during resistance exercises can, although rare, lead to bilateral anterior shoulder dislocation.

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Figure 1. Clinical anterior view of bilateral anterior shoulder dislocation (epaulet sign)

Figure 2. Chest X-ray showing anterior glenohumeral dislocation in both the right and left shoulders

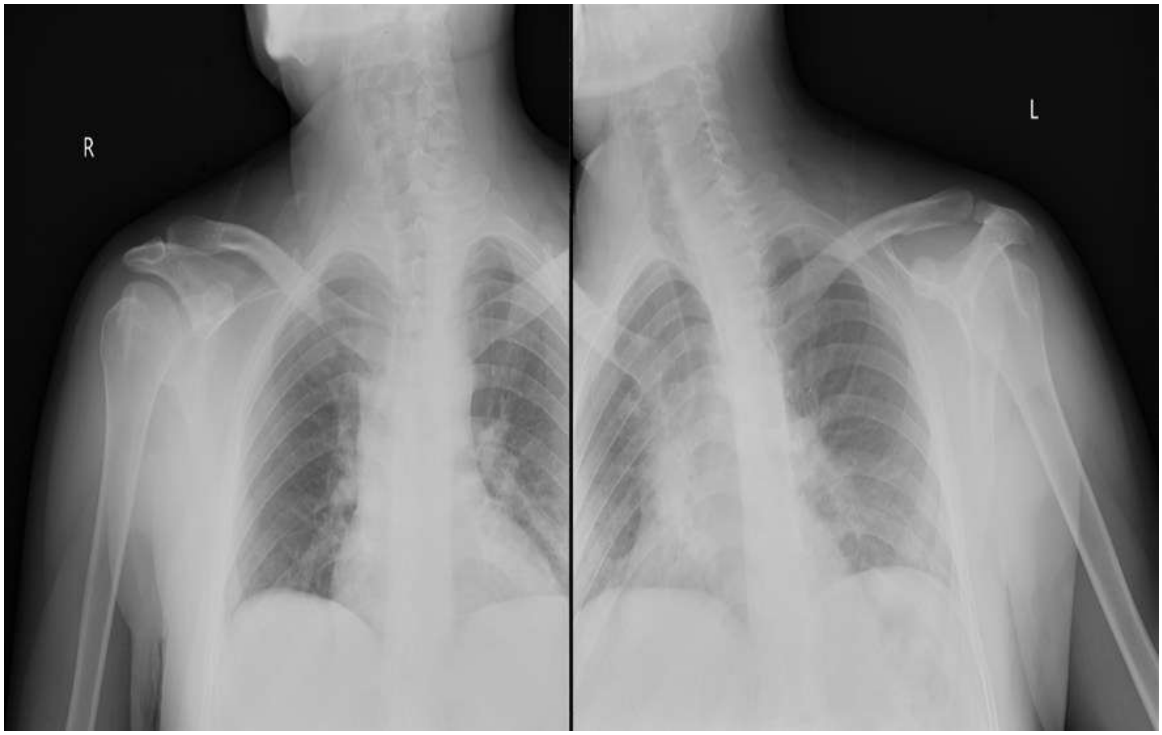

Figure 3. Chest X-ray images after reduction of the right and left shoulders

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Pulmonary embolism presenting with right upper quadrant pain: A case report

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Abstract

In this report, we present a case of pulmonary embolism presenting with right upper quadrant abdominal pain and emphasise the importance of considering atypical presentations in the differential diagnosis. A 73-year-old female patient presented to the emergency department with complaints of right upper quadrant abdominal pain and cough that had persisted for two days. At the time of admission, her vital signs were as follows: oxygen saturation 95%, heart rate 83 beats/min, temperature 36.3 °C, and respiratory rate 15 breaths/min. On physical examination, rales were detected in the right hemithorax. Abdominal examination revealed tenderness in the right upper quadrant, and Murphy's sign was positive. Laboratory investigations revealed an elevated C-reactive protein level of 128 mg/L, whereas other laboratory parameters were within normal limits. Hepatobiliary system and abdominal ultrasonography findings were reported as normal. Due to the persistence of symptoms, contrast-enhanced abdominal computed tomography was performed. Although no intra-abdominal pathology was detected, an appearance compatible with a Hampton hump was observed in the basal lung sections included within the imaging field. Based on this finding, pulmonary embolism was suspected and pulmonary computed tomography (CT) angiography was planned. Pulmonary CT angiography demonstrated filling defects consistent with emboli in the pulmonary artery branches of the right middle and lower lobes. The patient was admitted to the Department of Chest Diseases. Treatment was initiated with low-molecular-weight heparin and acetylsalicylic acid. Venous Doppler ultrasonography of the lower extremities did not reveal deep vein thrombosis. Echocardiography demonstrated normal right heart chambers. After six days of hospitalisation, the patient was discharged with oral warfarin therapy. In patients presenting with right upper quadrant abdominal pain, pulmonary embolism should be considered in the differential diagnosis after hepatobiliary pathologies have been excluded.

Keywords: Hampton hump, Murphy sign, Pulmonary embolism.

Introduction

Pulmonary embolism is one of the most serious forms of venous thromboembolism and represents a significant cause of emergency department admissions (1). The most common presenting symptoms are dyspnoea, pleuritic chest pain, and haemoptysis; however, the disease may also manifest with atypical clinical findings (1).

These atypical presentations include syncope, isolated tachycardia, back pain and, more rarely, abdominal pain. In patients presenting with right upper quadrant pain, hepatobiliary pathologies are usually considered first in the differential diagnosis, which may delay the diagnosis of pulmonary embolism (1-3).

In this report, we present a case of pulmonary embolism presenting with right upper quadrant abdominal pain and emphasise the importance of considering atypical presentations in the differential diagnosis.

Case Report

A 73-year-old female patient presented to the emergency department with complaints of right upper quadrant abdominal pain and cough that had persisted for two days. Her medical history was notable for aortic valve insufficiency. At the time of admission, her vital signs were as follows: oxygen saturation 95%, heart rate 83 beats/min, temperature 36.3 °C, and respiratory rate 15 breaths/min. Her Glasgow Coma Scale score was 15, and she was oriented and cooperative. Electrocardiography demonstrated normal sinus rhythm. On physical examination, rales were detected in the right hemithorax. Abdominal examination revealed tenderness in the right upper quadrant, and Murphy's sign was positive. Laboratory investigations revealed an elevated C-reactive protein level of 128 mg/L, whereas other laboratory parameters were within normal limits. Hepatobiliary system and abdominal ultrasonography findings were reported as normal. Due to the persistence of symptoms, contrast-enhanced abdominal computed tomography was performed. Although no intra-abdominal pathology was detected, an appearance compatible with a Hampton hump was observed in the basal lung sections included within the imaging field. Based on this finding, pulmonary embolism was suspected and pulmonary computed tomography (CT) angiography was planned. Pulmonary CT angiography demonstrated filling defects consistent with emboli in the pulmonary artery branches of the right middle and lower lobes. The patient was admitted to the Department of Chest Diseases. Treatment was initiated with low-molecular-weight heparin and acetylsalicylic acid. Venous Doppler ultrasonography of the lower extremities did not reveal deep vein thrombosis. Echocardiography demonstrated normal right heart chambers. After six days of hospitalisation, the patient was discharged with oral warfarin therapy.

Discussion

Pulmonary embolism is one of the most challenging clinical conditions to diagnose because patients may present with a wide variety of symptoms and clinical findings. Although dyspnoea and chest pain are the most common presenting symptoms, abdominal pain is considered a rare and atypical presentation.

Several case reports in the literature have described pulmonary embolism presenting with right upper quadrant abdominal pain (1–3). Rehman et al. reported a case of pulmonary embolism presenting with abdominal pain and emphasised that pulmonary embolism should be considered in the differential diagnosis (1). Similarly, Toker et al. described a rare case of pulmonary embolism presenting with right upper quadrant pain (2). Another case reported by Gantner demonstrated that pulmonary embolism may mimic an acute abdomen (3). Additional reports have also highlighted that the diagnosis of pulmonary embolism may be delayed in patients presenting primarily with abdominal pain (4).

Radiological findings may provide important diagnostic clues for pulmonary embolism. Chest radiography may be completely normal; however, findings such as atelectasis, elevation of the hemidiaphragm, consolidation mimicking pneumonia, and pleural effusion may also be observed. The Westermark sign and Hampton's hump are classic radiological findings that support the diagnosis of pulmonary embolism (5).

In the present case, hepatobiliary pathologies were excluded, and the identification of a Hampton hump in the lung sections on abdominal imaging accelerated the diagnostic process. The absence of deep vein thrombosis on lower extremity Doppler ultrasonography also demonstrates that pulmonary embolism may occur without an identifiable peripheral thrombus source.

Conclusion

In patients presenting with right upper quadrant abdominal pain, pulmonary embolism should be considered in the differential diagnosis after hepatobiliary pathologies have been excluded.

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RISK STRATIFICATION AND TREATMENT APPROACHES IN ACUTE PULMONARY EMBOLISM: A FOCUS ON RIGHT HEART DYSFUNCTION

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ABSTRACT

Background

Acute pulmonary embolism (PE) remains one of the leading causes of cardiovascular mortality despite remarkable advances in diagnostic imaging and therapeutic strategies. Because clinical presentation ranges from asymptomatic disease to obstructive shock, accurate early risk stratification is essential. Increasing evidence indicates that right ventricular (RV) dysfunction, rather than thrombus burden alone, is the primary determinant of short-term prognosis.

Objective

This narrative review summarizes current evidence regarding risk stratification in acute PE, emphasizing the clinical importance of RV dysfunction and its implications for contemporary therapeutic decision-making.

Methods

A narrative review was performed using PubMed, Scopus, and Web of Science databases. Publications between 2019 and 2025 addressing pulmonary embolism, right ventricular dysfunction, computed tomography, echocardiography, biomarkers, and current international guidelines were evaluated. Priority was given to randomized controlled trials, systematic reviews, and the latest ESC and CHEST guideline recommendations.

Results

Current evidence demonstrates that RV dysfunction is the strongest predictor of early clinical deterioration and mortality in acute PE. Multimodal assessment combining computed tomography pulmonary angiography, echocardiography, cardiac biomarkers, and clinical evaluation provides superior risk stratification compared with clinical scoring systems alone. While anticoagulation remains the cornerstone of therapy, patients with intermediate-high and high-risk PE may benefit from advanced reperfusion strategies, including catheter-directed interventions and mechanical thrombectomy, depending on their hemodynamic status.

Conclusion

Contemporary management of acute PE should prioritize assessment of right ventricular function and individualized treatment strategies. A multimodal diagnostic approach facilitates early identification of high-risk patients and supports timely therapeutic intervention.

Keywords: Pulmonary embolism; Right ventricular dysfunction; Risk stratification; Anticoagulation; Mechanical thrombectomy

1. INTRODUCTION

Acute pulmonary embolism (PE) is the third leading cause of cardiovascular death after myocardial infarction and stroke, accounting for significant morbidity and mortality worldwide. Advances in multidetector computed tomography pulmonary angiography (CTPA), high-sensitivity cardiac biomarkers, and catheter-based therapies have substantially improved diagnosis and treatment over the last decade. Nevertheless, early mortality remains considerable, particularly among patients presenting with right ventricular (RV) dysfunction or hemodynamic instability [1,2].

The prognosis of acute PE is determined not only by thrombus burden but also by the cardiovascular response to pulmonary arterial obstruction. Acute increases in pulmonary vascular resistance impose pressure overload on the right ventricle, leading to RV dilatation, reduced left ventricular preload, impaired systemic perfusion, and, in severe cases, cardiogenic shock. Consequently, contemporary international guidelines emphasize RV dysfunction as the cornerstone of risk stratification[3,4].

Current management relies on integrating clinical findings, imaging, laboratory biomarkers, and validated prognostic scores to classify patients into low-, intermediate-, and high-risk categories. This individualized approach guides therapeutic decisions ranging from anticoagulation alone to advanced reperfusion therapies.

The objective of this review is to summarize current evidence regarding the role of RV dysfunction in risk stratification and to discuss contemporary treatment strategies for acute pulmonary embolism.

2. Methods

This study was designed as a narrative review of the contemporary literature addressing risk stratification and treatment strategies in acute pulmonary embolism (PE), with particular emphasis on right ventricular (RV) dysfunction. A comprehensive literature search was performed using PubMed, Scopus, and Web of Science databases. Articles published between January 2019 and June 2025 were screened using the keywords pulmonary embolism, right ventricular dysfunction, risk stratification, computed tomography pulmonary angiography, echocardiography, cardiac biomarkers, catheter-directed therapy, and mechanical thrombectomy. Current international guidelines, including the European Society of Cardiology (ESC) and the American College of Chest Physicians (CHEST), together with randomized controlled trials, systematic reviews, and high-quality observational studies, were prioritized. Studies focusing on chronic thromboembolic pulmonary hypertension, pediatric populations, and case reports were excluded.

3. Results

Recent evidence consistently demonstrates that right ventricular dysfunction is the most important predictor of early adverse outcomes in acute pulmonary embolism. Although anatomical clot burden contributes to disease severity, several studies have shown that RV dysfunction correlates more closely with short-term mortality, intensive care unit admission, and hemodynamic deterioration.

Computed tomography pulmonary angiography (CTPA) has become the first-line imaging modality because it simultaneously confirms the diagnosis and provides prognostic information. Among CT

findings, the RV/LV diameter ratio greater than 0.9–1.0 is the most reproducible indicator of RV dysfunction and has been associated with increased mortality and the need for advanced therapies. Additional findings, including interventricular septal bowing and contrast reflux into the inferior vena cava, further support the presence of significant RV strain [5,6].

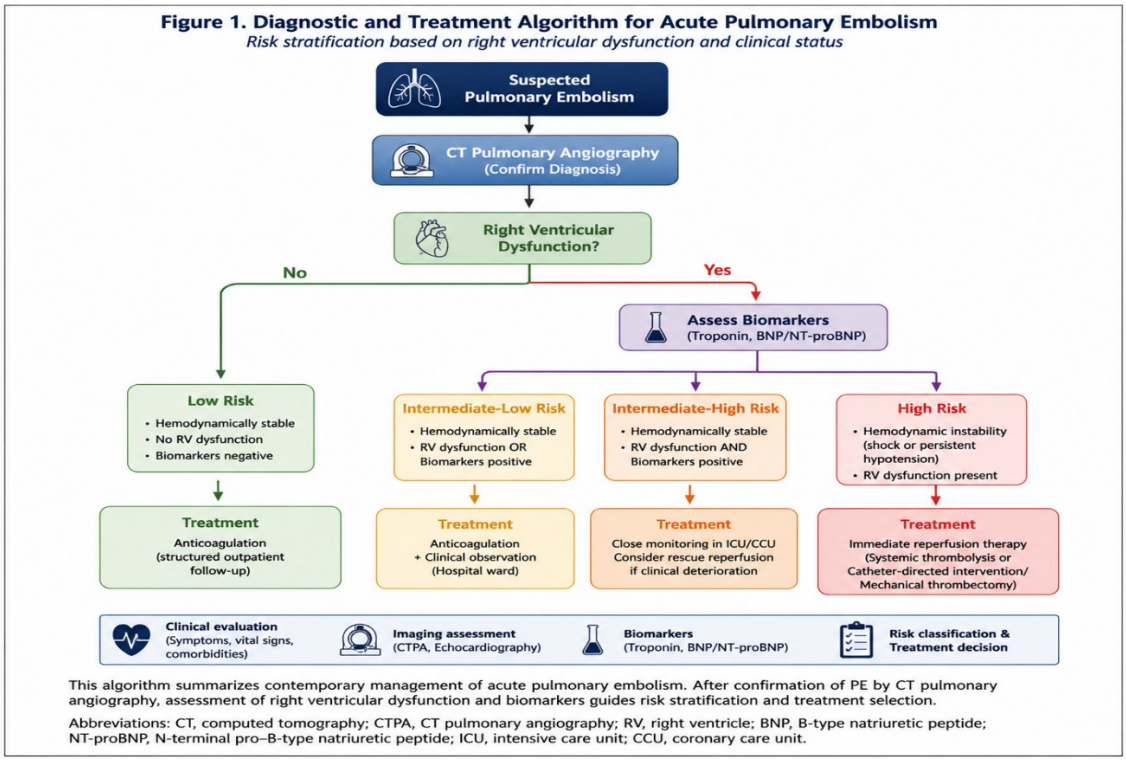
Echocardiography remains the reference method for functional assessment of the right ventricle. Parameters such as RV dilatation, reduced tricuspid annular plane systolic excursion (TAPSE), McConnell's sign, and elevated pulmonary artery systolic pressure identify patients at increased risk for clinical deterioration. Nevertheless, echocardiography is operator dependent and may not always be immediately available in emergency settings [Table 1].

Cardiac biomarkers complement imaging findings by reflecting myocardial injury and ventricular overload. Elevated cardiac troponin indicates RV ischemia, whereas BNP or NT-proBNP reflects ventricular wall stress. Combined positivity of imaging and biomarkers identifies patients with intermediate-high-risk PE who require close monitoring because of the increased likelihood of hemodynamic collapse.

Current guideline recommendations advocate risk-adapted management. Anticoagulation remains the cornerstone of treatment in all hemodynamically stable patients. Systemic thrombolysis is reserved primarily for high-risk PE or rescue therapy in patients with clinical deterioration. Catheter-directed thrombolysis and mechanical thrombectomy have emerged as promising alternatives for selected patients with intermediate-high and high-risk PE, offering rapid improvement in RV function while potentially reducing bleeding complications associated with systemic thrombolysis [Figure 1][7,8].

Table 1. Contemporary Risk Stratification and Initial Management of Acute Pulmonary Embolism

Risk Group	Hemodynamic Status	RV Dysfunction	Cardiac Biomarkers	Recommended Initial Treatment
Low Risk	Stable	Absent	Negative	Anticoagulation
Intermediate-Low	Stable	Present or Positive Biomarkers	One positive	Anticoagulation + Clinical Observation
Intermediate-High	Stable	Present	Positive	Close Monitoring ± Rescue Reperfusion
High Risk	Shock / Persistent Hypotension	Usually Present	Usually Positive	Immediate Reperfusion Therapy



4. Discussion

Accurate risk stratification remains the cornerstone of contemporary pulmonary embolism (PE) management. Although several clinical prediction models, including the Pulmonary Embolism Severity Index (PESI) and simplified PESI, have demonstrated prognostic value, current evidence suggests that these scores should not be used in isolation. Instead, integration of clinical findings with imaging and cardiac biomarkers provides a more reliable assessment of disease severity and early clinical deterioration.

Among all prognostic indicators, right ventricular (RV) dysfunction has consistently emerged as the strongest predictor of adverse outcomes. Acute pressure overload caused by pulmonary arterial obstruction initiates a cascade of RV dilatation, impaired ventricular contractility, decreased left ventricular filling, and reduced cardiac output. This pathophysiological mechanism explains why RV dysfunction correlates more closely with mortality than thrombus burden alone. Consequently, routine evaluation of RV function should be considered an essential component of the initial diagnostic work-up in every patient with acute PE [8,9].

Recent advances in multidetector CT have substantially changed the diagnostic approach to PE. Beyond confirming pulmonary arterial thrombi, CT pulmonary angiography provides valuable prognostic information through assessment of the RV/LV diameter ratio and other markers of RV strain. Because CT is rapidly available in most emergency departments, it has become the preferred first-line modality for simultaneous diagnosis and early risk assessment [10]. Echocardiography remains indispensable in patients with hemodynamic instability or inconclusive CT findings, providing direct functional assessment of the right ventricle.

Treatment strategies have also evolved considerably during the last decade. While anticoagulation continues to represent the foundation of therapy, growing evidence supports individualized treatment according to patient risk profile. Mechanical thrombectomy and catheter-directed interventions have demonstrated encouraging improvements in RV function and hemodynamic parameters, particularly in intermediate-high and high-risk patients. Nevertheless, current evidence remains insufficient to

recommend routine use of these techniques in all patients. Careful patient selection and multidisciplinary decision-making remain essential [11,12].

Overall, contemporary PE management has shifted from a thrombus-centered approach toward a physiology-based strategy focused on RV performance and early identification of patients at risk for deterioration. Future prospective studies are needed to better define the role of catheter-based reperfusion therapies and refine individualized treatment algorithms[12].

5. Conclusion

Acute pulmonary embolism remains a major cardiovascular emergency requiring prompt diagnosis and appropriate risk stratification. Current evidence consistently identifies right ventricular dysfunction as the principal determinant of early prognosis and the most important factor guiding therapeutic decisions. A multimodal assessment combining clinical evaluation, CT pulmonary angiography, echocardiography, and cardiac biomarkers allows more accurate identification of patients at increased risk for adverse outcomes than clinical prediction scores alone.

Anticoagulation remains the standard treatment for most patients, whereas advanced reperfusion therapies should be reserved for carefully selected intermediate-high and high-risk patients. Contemporary management should therefore emphasize individualized treatment strategies based on RV function rather than thrombus burden alone. Continued research is required to optimize patient selection for catheter-based interventions and improve long-term clinical outcomes.

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Neutrophil-to-Lymphocyte Ratio as a Prognostic Marker in Acute Exacerbation of Chronic Obstructive Pulmonary Disease

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Structured Abstract

Background

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is a major cause of hospitalization and mortality worldwide. Early identification of patients at increased risk for adverse outcomes remains challenging. Among readily available inflammatory biomarkers, the neutrophil-to-lymphocyte ratio (NLR) has emerged as a simple and cost-effective prognostic indicator .

Objective

To summarize current evidence regarding the prognostic value of NLR in patients hospitalized with AECOPD and its potential role in risk stratification and clinical decision-making.

Methods

A narrative review was performed using PubMed, Scopus, and Web of Science databases. Studies published between 2019 and 2025 investigating NLR in AECOPD were reviewed. Priority was given to systematic reviews, meta-analyses, prospective studies, and the latest GOLD recommendations .

Results

Current evidence indicates that elevated NLR is associated with increased disease severity, longer hospital stay, intensive care unit (ICU) admission, need for non-invasive or invasive mechanical ventilation, and higher short-term mortality. NLR demonstrates superior prognostic performance compared with several conventional inflammatory markers and may complement routine clinical assessment .

Conclusion

NLR is an inexpensive, rapidly available biomarker that may improve early risk stratification in AECOPD. Integration of NLR with clinical evaluation and conventional biomarkers could facilitate timely identification of high-risk patients.

Keywords: COPD; Acute exacerbation; Neutrophil-to-lymphocyte ratio; Prognosis; Biomarker

1. Introduction

Chronic obstructive pulmonary disease (COPD) is one of the leading causes of morbidity and mortality worldwide and is responsible for a substantial healthcare burden (1,2). Acute exacerbations accelerate lung function decline, reduce quality of life, and account for most COPD-related hospital admissions and healthcare costs (2,3). Despite advances in pharmacological treatment and supportive care, predicting disease severity at hospital admission remains difficult.

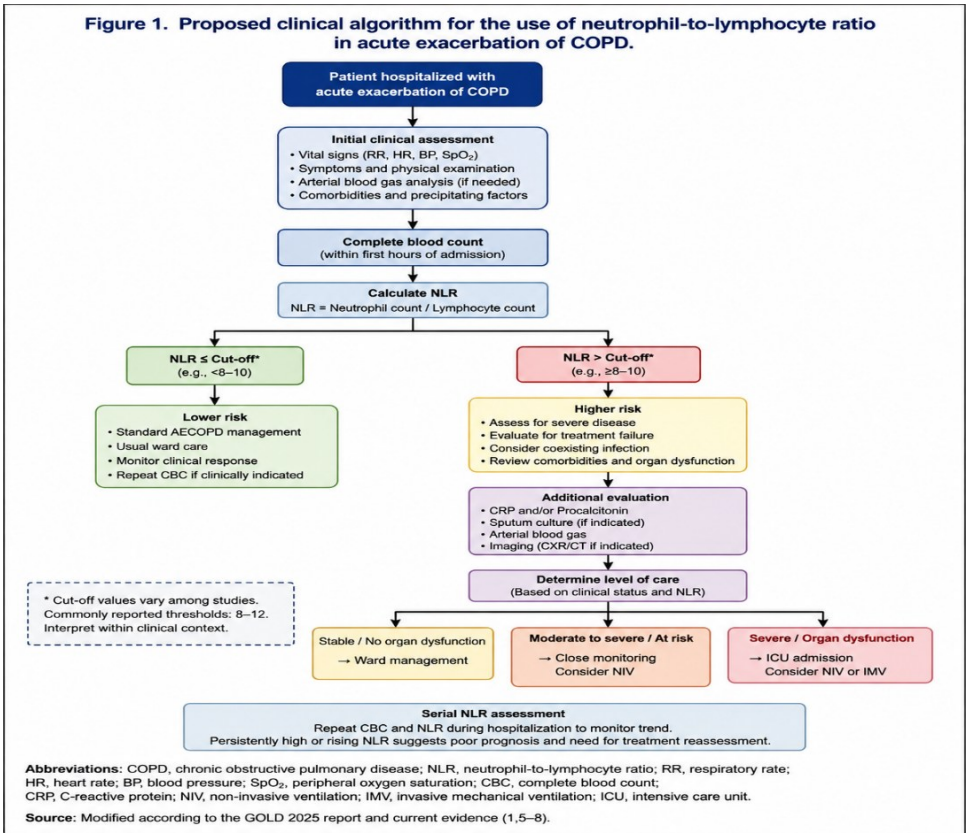
Systemic inflammation plays a central role in the pathogenesis of AECOPD. During exacerbations, activation of neutrophils together with relative lymphocyte suppression results in an elevated neutrophil-to-lymphocyte ratio (NLR), which reflects both innate immune activation and physiological stress (4,5). Because NLR is derived from a routine complete blood count, it is inexpensive, reproducible, and immediately available in virtually all healthcare settings.

Recent studies have demonstrated that elevated NLR correlates with ICU admission, mechanical ventilation, prolonged hospitalization, and mortality, suggesting that it may serve as a practical prognostic biomarker in AECOPD (5–8). This review summarizes current evidence regarding the clinical utility of NLR and its role in the early risk assessment of hospitalized patients with AECOPD.

2. Methods

A narrative review of the literature was conducted using PubMed, Scopus, and Web of Science databases. The search included publications from January 2019 through June 2025 using the keywords *COPD,* acute exacerbation, neutrophil-to-lymphocyte ratio, inflammatory biomarkers, prognosis, and mortality. Randomized trials, systematic reviews, meta-analyses, observational cohort studies, and the latest GOLD recommendations were preferentially included. Case reports, editorials, and studies with insufficient methodological quality were excluded.

Figure 1. Proposed clinical algorithm for the use of the neutrophil-to-lymphocyte ratio in acute exacerbation of COPD



3. Results

The available evidence consistently indicates that the neutrophil-to-lymphocyte ratio (NLR) is higher during acute exacerbations of chronic obstructive pulmonary disease (AECOPD) than during clinically stable periods. This increase reflects simultaneous neutrophil-dominant inflammatory activation and relative lymphopenia associated with physiological stress. In a 2022 systematic review and meta-analysis including 15 studies and 10,038 patients, admission NLR was significantly associated with mortality, mechanical ventilation, intensive care unit transfer, prolonged hospitalization, pulmonary hypertension, or composite adverse outcomes. The pooled sensitivity, specificity, and area under the receiver operating characteristic curve were 0.71, 0.73, and 0.78, respectively (2).

Higher NLR values have also been associated with the severity of acute respiratory failure. Hospitalized patients with elevated admission NLR more frequently required intensive care, non-invasive ventilation, invasive mechanical ventilation, or vasopressor support than patients with lower values. A 2024 retrospective study identified an NLR cut-off of approximately 11 for predicting a composite of ICU admission, invasive mechanical ventilation, and in-hospital mortality (3). However, reported thresholds differ substantially among cohorts, limiting the adoption of a universal clinical cut-off.

NLR may provide prognostic information extending beyond the index hospitalization. In a cohort of 503 hospitalized patients with AECOPD, elevated NLR was associated with adverse outcomes within 90 days, particularly COPD-related mortality and readmission (4). A subsequent study evaluating NLR kinetics found that patients with persistently low NLR during hospitalization had fewer adverse hospital outcomes and lower six-month mortality, whereas persistently elevated or increasing values identified a higher-risk phenotype (5). These observations suggest that serial measurement may be more informative than a single admission value.

NLR correlates with conventional inflammatory markers but reflects a different biological dimension. C-reactive protein (CRP) primarily indicates the magnitude of systemic inflammation, while procalcitonin is more useful when bacterial infection and antibiotic stewardship are considered. In a 2024 study, NLR was significantly higher in AECOPD than in stable COPD and showed a moderate positive correlation with CRP. Combining NLR with other leukocyte-derived indices improved diagnostic sensitivity, although the incremental prognostic value requires further validation (6).

The principal findings and clinical implications of frequently used inflammatory biomarkers are summarized in Table 1. Overall, NLR appears most useful as an adjunctive risk marker rather than as an isolated determinant of ICU admission or ventilatory support.

Table 1. Clinical significance of inflammatory biomarkers in acute exacerbation of COPD

Biomarker	Principal clinical implication	Advantages	Major limitations
NLR	Associated with adverse outcomes, ICU admission, ventilatory support, prolonged hospitalization, and mortality	Inexpensive, rapidly available, derived from routine blood count	Non-specific; affected by infection, corticosteroids, malignancy, stress, and comorbid disease
CRP	Reflects systemic inflammatory burden and may support identification of infectious exacerbations	Widely available and familiar in routine practice	Limited specificity; may remain elevated because of non-pulmonary inflammation
Procalcitonin	Supports assessment of bacterial infection and antibiotic de-escalation in selected patients	More closely related to bacterial infection than NLR or CRP	Not primarily validated as a stand-alone prognostic marker; affected by renal dysfunction and severe systemic inflammation
Blood eosinophils	May identify corticosteroid-responsive exacerbations and type 2 inflammatory phenotype	Supports individualized corticosteroid use	Variable during infection and corticosteroid exposure; threshold values remain context-dependent

4. Discussion

The findings of this narrative review support NLR as a practical marker of systemic inflammatory stress in patients hospitalized with AECOPD. Its main advantage is accessibility: NLR requires no additional laboratory test, cost, or processing time because it is calculated directly from the complete blood count. The consistency of its association with mortality, ICU admission, mechanical ventilation, and length of stay makes it attractive for early bedside risk assessment (2–5).

Nevertheless, NLR should not be interpreted as a disease-specific biomarker. Neutrophilia may result from bacterial infection, systemic corticosteroid exposure, smoking, physiological stress, or concomitant cardiovascular disease, while lymphopenia may reflect age, malnutrition, malignancy, or immune dysfunction. Therefore, a high NLR cannot distinguish an infectious exacerbation from other causes of clinical deterioration. It should be interpreted together with respiratory rate, oxygenation, blood gas analysis, mental status, hemodynamic findings, CRP, procalcitonin, and relevant comorbidities.

Another limitation is the absence of a universally accepted NLR threshold. Published cut-offs vary according to study population, disease severity, outcome definition, timing of blood sampling, and corticosteroid administration. Although values around have frequently identified patients at increased risk, adopting a single threshold without local validation could result in inappropriate escalation or false reassurance (8). The available meta-analytic evidence demonstrates moderate—not definitive—discriminative performance, with an area under the curve of approximately 0.78 (9,10).

Serial NLR measurements may partly overcome these limitations. A persistently elevated or increasing NLR may indicate ongoing inflammation, treatment failure, or evolving organ dysfunction, whereas a declining value may accompany clinical improvement. However, prospective studies are required to determine whether NLR-guided management improves outcomes beyond established clinical assessment.

Accordingly, the proposed pathway illustrated in Figure 1 positions NLR as an adjunctive marker. It may identify patients requiring more intensive observation, but decisions regarding ICU transfer, non-invasive ventilation, intubation, or antibiotic treatment should not be based on NLR alone.

5. Conclusion

The neutrophil-to-lymphocyte ratio is an inexpensive, rapidly available, and potentially useful prognostic biomarker in acute exacerbation of COPD. Elevated admission values are associated with adverse outcomes, including ICU admission, ventilatory support, prolonged hospitalization, readmission, and mortality. Serial measurement may provide additional information regarding clinical trajectory.

However, NLR lacks disease specificity, and reported cut-off values remain heterogeneous. It should therefore be incorporated into a multimodal assessment rather than used as an independent treatment trigger. Future prospective, multicenter studies should establish standardized thresholds and determine whether NLR-guided risk stratification provides incremental benefit beyond conventional clinical and laboratory parameters.

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Individualized Treatment Strategies in Intermediate-Risk Pulmonary Embolism: A Clinical Perspective

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Abstract

Background

Intermediate-risk pulmonary embolism (PE) comprises a heterogeneous population of hemodynamically stable patients with variable degrees of right ventricular (RV) dysfunction and myocardial injury. Although anticoagulation remains the standard initial treatment, a subgroup may experience early hemodynamic deterioration and require rescue reperfusion. The increasing availability of catheter-directed thrombolysis and mechanical thrombectomy has expanded therapeutic options but has also complicated treatment selection .

Objective

To review contemporary evidence regarding individualized management strategies for intermediate-risk PE and to identify clinical, imaging, and laboratory findings that may guide escalation beyond anticoagulation.

Methods

A narrative review was conducted using PubMed, Scopus, Web of Science, and current international guideline documents. Studies evaluating anticoagulation, systemic thrombolysis, catheter-directed thrombolysis, mechanical thrombectomy, RV dysfunction, cardiac biomarkers, and clinical deterioration in intermediate-risk PE were considered. Randomized trials, prospective device studies, systematic reviews, and major guideline recommendations were prioritized .

Results

Intermediate-low-risk patients generally achieve favorable outcomes with anticoagulation and clinical observation. Intermediate-high-risk patients, characterized by concomitant RV dysfunction and positive cardiac biomarkers, have a greater risk of early decompensation and require monitored care. Routine systemic thrombolysis reduces hemodynamic collapse but increases major bleeding and intracranial hemorrhage. Catheter-directed thrombolysis and mechanical thrombectomy improve surrogate hemodynamic outcomes, including RV/LV ratio, but definitive mortality benefits remain incompletely established .

Conclusion

Management of intermediate-risk PE should be individualized according to hemodynamic trajectory, RV function, myocardial injury, bleeding risk, and local expertise. Advanced reperfusion should not be applied routinely but may be appropriate in selected patients with clinical deterioration or a high-risk physiological profile.

Keywords: Pulmonary embolism; Intermediate risk; Right ventricular dysfunction; Thrombolysis; Mechanical thrombectomy

1. Introduction

Intermediate-risk pulmonary embolism represents one of the most challenging categories in acute PE management. These patients are normotensive at presentation but show evidence of RV pressure overload, myocardial injury, or both. The clinical course is highly variable: most remain stable with anticoagulation alone, whereas a smaller subgroup develops progressive respiratory failure, hypotension, obstructive shock, or cardiac arrest (1,2).

The European risk classification separates intermediate-risk PE into intermediate-low and intermediate-high categories. Intermediate-low-risk patients have either imaging evidence of RV dysfunction or elevated cardiac biomarkers, whereas intermediate-high-risk patients have both findings despite preserved systemic blood pressure (3). This distinction is clinically important because patients with combined RV dysfunction and myocardial injury have a higher probability of early deterioration and require closer monitoring.

Anticoagulation remains the foundation of therapy. However, systemic thrombolysis, catheter-directed thrombolysis, and catheter-based mechanical thrombectomy have been investigated as strategies to achieve faster RV unloading and prevent hemodynamic collapse. The principal clinical challenge is balancing the potential benefit of early reperfusion against the risk of major bleeding and procedural

complications. Therefore, treatment decisions should be based on dynamic physiological assessment rather than the risk category alone.

This review summarizes current evidence regarding individualized treatment strategies for intermediate-risk PE and proposes a practical framework for selecting patients for anticoagulation, monitored observation, or advanced reperfusion therapy.

2. Methods

A narrative literature review was performed using PubMed, Scopus, and Web of Science. Publications addressing intermediate-risk PE, RV dysfunction, cardiac biomarkers, systemic thrombolysis, catheter-directed thrombolysis, and mechanical thrombectomy were reviewed. Priority was given to international guidelines, randomized controlled trials, prospective multicenter studies, and systematic reviews. The PEITHO, FLARE, EXTRACT-PE, PEERLESS, and contemporary catheter-based therapy studies were evaluated for efficacy, safety, and clinical applicability (4–8). Studies limited to chronic thromboembolic pulmonary hypertension, isolated case reports, and pediatric populations were excluded.

3. Results

Current evidence supports therapeutic anticoagulation as the initial treatment for most hemodynamically stable patients with intermediate-risk PE (1,2). Intermediate-low-risk patients generally require standard anticoagulation and inpatient observation, whereas intermediate-high-risk patients should be managed in a monitored setting because deterioration may occur during the first 24–72 hours.

The PEITHO trial evaluated systemic tenecteplase plus heparin in normotensive patients with RV dysfunction and positive troponin. Thrombolysis reduced the composite outcome of death or hemodynamic decompensation but substantially increased extracranial major bleeding and hemorrhagic stroke (3). These findings established that routine full-dose systemic thrombolysis is not justified in unselected intermediate-risk patients. Current practice therefore reserves systemic thrombolysis primarily for rescue treatment when hemodynamic deterioration occurs despite anticoagulation.

Catheter-directed thrombolysis aims to deliver lower thrombolytic doses directly into the pulmonary arteries. Early trials demonstrated rapid reductions in RV/LV ratio and pulmonary artery pressure, with lower total thrombolytic exposure than systemic therapy. However, most studies were small, single-arm, or focused on short-term surrogate endpoints rather than mortality (4,5).

Mechanical thrombectomy offers immediate thrombus extraction without thrombolytic exposure. In the FLARE study, FlowTriever thrombectomy significantly reduced the RV/LV ratio with a low major bleeding rate in intermediate-risk PE (6). The EXTRACT-PE trial similarly demonstrated improvement in RV/LV ratio using aspiration thrombectomy (7). More recent randomized comparisons, including PEERLESS, suggest that large-bore mechanical thrombectomy may reduce clinical deterioration, intensive care utilization, and rescue treatment compared with catheter-directed thrombolysis in selected patients requiring intervention, although mortality differences remain uncertain (8).

The most clinically relevant factors supporting escalation beyond anticoagulation include worsening tachycardia, increasing oxygen requirement, borderline systolic blood pressure, elevated lactate, progressive RV dysfunction, rising troponin, syncope, severe hypoxemia, and limited cardiopulmonary reserve. Conversely, advanced age, prior intracranial hemorrhage, recent surgery, active bleeding, severe thrombocytopenia, and frailty increase the procedural or thrombolytic risk. Individualized treatment therefore requires simultaneous assessment of deterioration risk and bleeding risk. The proposed management strategy is summarized in Table 1.

Table 1. Individualized management of intermediate-risk pulmonary embolism

Clinical category	Key findings	Recommended management
Intermediate-low risk	Hemodynamically stable; RV dysfunction **or** positive biomarker	Therapeutic anticoagulation; ward observation; reassessment if symptoms worsen
Intermediate-high risk without deterioration	Hemodynamically stable; RV dysfunction **and** positive biomarker	Anticoagulation; monitored unit or ICU-level surveillance; early multidisciplinary review
Intermediate-high risk with warning signs	Increasing oxygen need, tachycardia, elevated lactate, borderline blood pressure, progressive RV dysfunction	Consider catheter-directed therapy or mechanical thrombectomy according to bleeding risk and expertise
Hemodynamic deterioration	Persistent hypotension, vasopressor requirement, obstructive shock	Rescue reperfusion: systemic thrombolysis, catheter-based intervention, or surgical embolectomy

4. Discussion

The central principle in intermediate-risk PE is that risk classification represents a starting point rather than a definitive treatment decision. Patients classified within the same category may have substantially different physiological reserve, bleeding risk, clot distribution, and probability of deterioration. Therefore, individualized therapy must incorporate dynamic clinical changes in addition to baseline imaging and biomarker findings.

Routine systemic thrombolysis cannot be recommended because its reduction in hemodynamic collapse is offset by major bleeding and intracranial hemorrhage (3). Nevertheless, the PEITHO findings should not be interpreted as evidence against all reperfusion. Instead, they support selective use in patients whose risk of decompensation exceeds their bleeding risk.

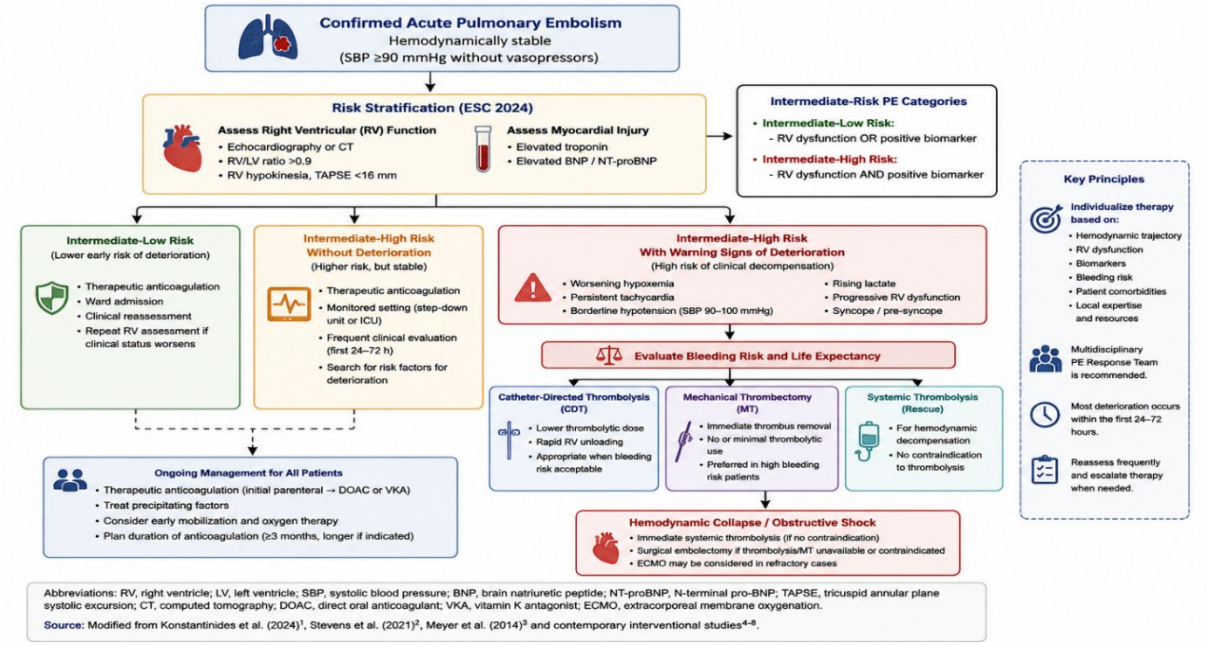
Catheter-based therapies have changed the therapeutic landscape. Mechanical thrombectomy is particularly attractive in patients with high bleeding risk because it avoids thrombolytic exposure. Catheter-directed thrombolysis may be suitable when thrombus is accessible and bleeding risk is acceptable. However, improvements in RV/LV ratio and pulmonary artery pressure are surrogate endpoints, and the relationship between these changes and long-term survival remains uncertain (9,10).

A multidisciplinary Pulmonary Embolism Response Team may improve treatment consistency by integrating pulmonology, cardiology, radiology, vascular surgery, emergency medicine, and intensive care expertise. Such teams are particularly valuable when the optimal strategy is uncertain or when local interventional resources influence treatment feasibility.

Accordingly, anticoagulation remains appropriate for most patients, while advanced reperfusion should be reserved for patients with clinical deterioration or a constellation of high-risk physiological features. The treatment pathway is illustrated in Figure 1.

Figure 1. Treatment Algorithm for Intermediate-Risk Pulmonary Embolism

An individualized, stepwise clinical approach



Abbreviations: RV, right ventricle; LV, left ventricle; SBP, systolic blood pressure; BNP, brain natriuretic peptide; NT-proBNP, N-terminal pro-BNP; TAPSE, tricuspid annular plane systolic excursion; CT, computed tomography; DOAC, direct oral anticoagulant; VKA, vitamin K antagonist; ECMO, extracorporeal membrane oxygenation.

Source: Modified from Konstantinides et al. (2024)[1], Stevens et al. (2021)[2], Meyer et al. (2014)[3] and contemporary interventional studies[4–8].

5. Conclusion

Intermediate-risk pulmonary embolism requires individualized, dynamic management. Anticoagulation is sufficient for most intermediate-low-risk and clinically stable intermediate-high-risk patients. Routine systemic thrombolysis should be avoided because of major bleeding risk. Catheter-directed thrombolysis and mechanical thrombectomy may be considered in selected patients with worsening clinical status, severe RV strain, or limited cardiopulmonary reserve.

Treatment decisions should integrate hemodynamic trajectory, RV function, cardiac biomarkers, lactate, oxygen requirement, bleeding risk, comorbidities, and institutional expertise. Future randomized studies should determine whether catheter-based strategies improve survival and long-term functional outcomes beyond short-term hemodynamic recovery.

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Clinical Utility of CT-Based Right Ventricular Dysfunction in Risk Stratification of Acute Pulmonary Embolism

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Abstract

Background

Computed tomography pulmonary angiography (CTPA) has evolved beyond its diagnostic role and now provides valuable prognostic information in patients with acute pulmonary embolism (PE). CT-derived markers of right ventricular (RV) dysfunction have emerged as important indicators of disease severity and adverse clinical outcomes .

Objective

To review the current evidence regarding CT-based assessment of RV dysfunction and its role in risk stratification and therapeutic decision-making in acute PE.

Methods

A narrative review was conducted using PubMed, Scopus, and Web of Science. Publications between 2019 and 2025 evaluating CT-derived RV parameters, RV/LV ratio, interventricular septal bowing, inferior vena cava reflux, and current ESC recommendations were reviewed. International guidelines, systematic reviews, and major clinical studies were prioritized .

Results

CT-derived RV dysfunction is consistently associated with increased mortality, hemodynamic deterioration, ICU admission, and the need for reperfusion therapy. The RV/LV diameter ratio remains the most extensively validated imaging marker. Additional CT findings, including interventricular septal bowing, contrast reflux into the inferior vena cava, pulmonary artery enlargement, and right atrial enlargement, improve prognostic assessment when interpreted together. CT therefore provides simultaneous diagnostic confirmation and early risk stratification in a single examination .

Conclusion

CT-based evaluation of RV dysfunction has become an integral component of contemporary PE management. Integration of CT findings with clinical assessment and cardiac biomarkers improves early identification of high-risk patients and supports individualized treatment strategies.

Keywords: Pulmonary embolism; Computed tomography; Right ventricular dysfunction; Risk stratification; RV/LV ratio

1. Introduction

Acute pulmonary embolism (PE) remains a major cause of cardiovascular morbidity and mortality worldwide (1). Although computed tomography pulmonary angiography (CTPA) was originally introduced as a diagnostic tool for detecting pulmonary arterial thrombi, advances in multidetector CT technology have considerably expanded its clinical utility. Contemporary CTPA not only confirms the diagnosis of PE but also provides detailed anatomical and functional information regarding right ventricular (RV) performance, allowing simultaneous assessment of disease severity and prognosis (1,2).

Acute obstruction of the pulmonary arterial circulation increases RV afterload, resulting in RV dilatation, interventricular septal displacement, impaired left ventricular filling, and reduced cardiac output. These hemodynamic changes are readily detectable on CT and correlate closely with short-term mortality and clinical deterioration (2–4). Among the available CT parameters, the RV/LV diameter ratio has become the most widely accepted imaging marker of RV dysfunction and is incorporated into contemporary risk assessment algorithms (1).

Beyond RV/LV ratio measurement, additional CT findings—including interventricular septal bowing, reflux of contrast into the inferior vena cava (IVC), right atrial enlargement, and pulmonary artery dilatation—provide complementary prognostic information (4–7). This review summarizes current evidence regarding CT-derived markers of RV dysfunction and discusses their role in individualized risk stratification and treatment planning in acute PE.

2. Methods

A narrative review was performed using PubMed, Scopus, and Web of Science databases. Studies published between January 2019 and June 2025 evaluating CT-derived RV dysfunction in acute PE were reviewed. Search terms included pulmonary embolism, computed tomography pulmonary angiography, right ventricular dysfunction, RV/LV ratio, interventricular septal bowing, inferior vena cava reflux, and risk stratification. Current ESC guidelines, systematic reviews, meta-analyses, prospective observational studies, and landmark clinical investigations were preferentially included, whereas case reports and studies with limited methodological quality were excluded.

3. Results

Computed tomography pulmonary angiography (CTPA) has become the cornerstone of acute pulmonary embolism (PE) diagnosis while simultaneously providing valuable prognostic information through assessment of right ventricular (RV) morphology. Increasing evidence indicates that CT-derived markers of RV dysfunction predict adverse clinical outcomes independently of thrombus burden and should be incorporated into routine risk stratification (1–3).

The RV/LV diameter ratio remains the most extensively investigated CT parameter. Measurements are obtained on axial or reconstructed four-chamber images by comparing the maximal transverse diameters of both ventricles. An RV/LV ratio ≥0.9 has consistently been associated with increased mortality, early hemodynamic deterioration, intensive care unit admission, and the need for reperfusion therapy (1,2). Several studies have demonstrated that CT-

derived RV/LV ratio has prognostic performance comparable to transthoracic echocardiography, particularly in emergency settings where immediate echocardiography may not be available (4).

Additional CT findings further improve risk assessment. Interventricular septal bowing reflects acute RV pressure overload and impaired left ventricular filling. Contrast reflux into the inferior vena cava (IVC) and hepatic veins indicates elevated right atrial pressure and severe RV dysfunction. Although these findings are less sensitive than the RV/LV ratio, their presence strongly suggests advanced hemodynamic compromise and should prompt closer clinical observation (5).

Morphological assessment of the pulmonary circulation also provides prognostic information. Enlargement of the main pulmonary artery, right atrial enlargement, and increased clot burden correlate with RV strain; however, clot burden alone has demonstrated weaker prognostic value than functional RV parameters. Current evidence therefore supports emphasizing RV performance rather than thrombus extent when estimating patient risk (2,6).

Recent developments in artificial intelligence (AI) have introduced automated CT analysis capable of measuring RV/LV ratio, ventricular volumes, and clot characteristics with minimal operator variability. Preliminary studies suggest that AI-assisted quantification may shorten interpretation time and improve reproducibility in emergency departments, although prospective validation remains necessary before widespread clinical implementation (7). The principal CT findings associated with RV dysfunction are summarized in **Table 1**.

Table 1. CT-derived markers of right ventricular dysfunction in acute pulmonary embolism

CT Finding	Pathophysiological significance	Clinical implication
RV/LV ratio ≥0.9	RV dilatation due to pressure overload	Increased mortality and hemodynamic deterioration
Interventricular septal bowing	Elevated RV pressure	Severe RV dysfunction
Contrast reflux into IVC/hepatic veins	Increased right atrial pressure	High-risk physiology
Right atrial enlargement	RV volume overload	Advanced RV dysfunction
Main pulmonary artery enlargement	Increased pulmonary vascular resistance	Suggests pulmonary hypertension
Decreased LV cavity size	Reduced preload	Low cardiac output

4. Discussion

CT pulmonary angiography has evolved from a purely diagnostic imaging technique into a comprehensive tool for prognostic assessment in acute PE. Unlike echocardiography, which evaluates cardiac function dynamically, CT provides standardized anatomical measurements that can be obtained immediately after diagnosis without requiring additional imaging. This dual diagnostic and prognostic capability makes CTPA particularly valuable in emergency practice (8,9).

Among CT-derived variables, the RV/LV ratio remains the most robust and reproducible marker of RV dysfunction. Its widespread validation across multiple cohorts has led to its incorporation into international guidelines and contemporary risk stratification algorithms (10). Nevertheless, RV/LV ratio should not be interpreted in isolation.

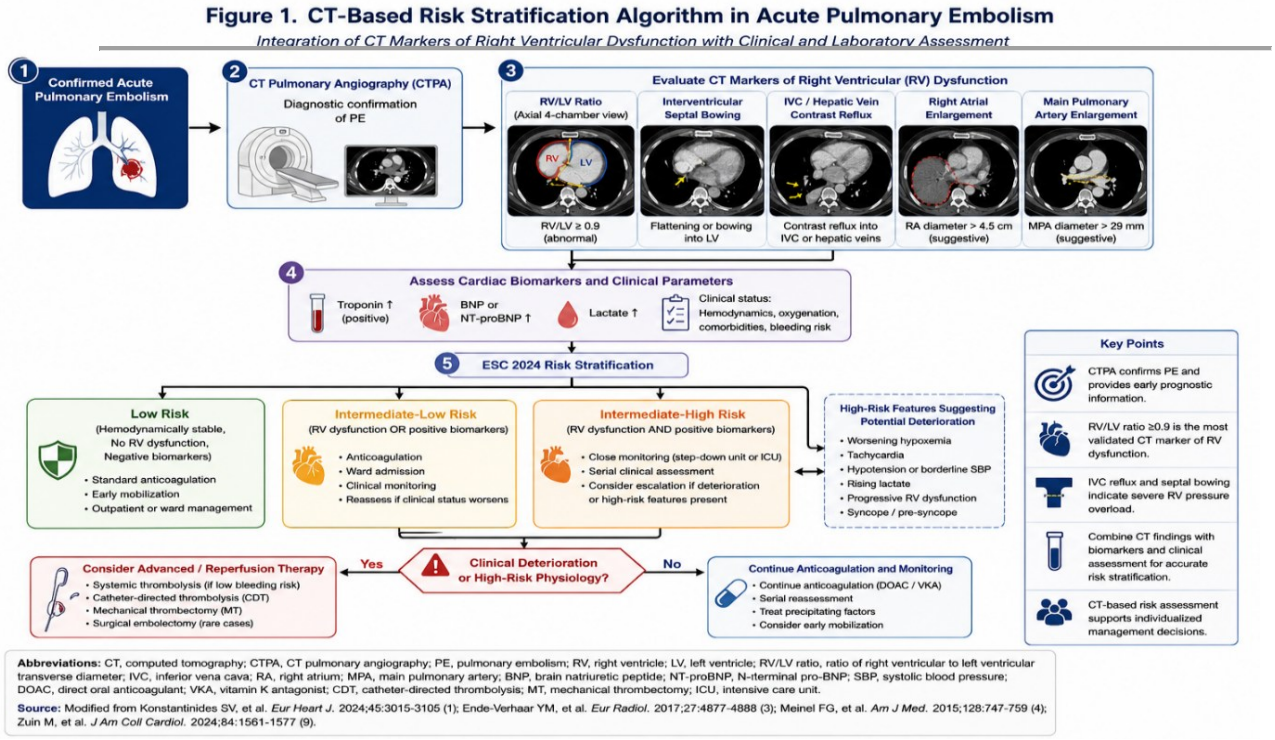
Combining this measurement with interventricular septal bowing, IVC reflux, cardiac biomarkers, lactate concentration, and clinical findings substantially improves prediction of early deterioration.

An important limitation of CT-based assessment is that it provides a static anatomical image rather than continuous functional evaluation. Factors such as chronic pulmonary hypertension, pre-existing RV enlargement, timing of contrast acquisition, and image reconstruction technique may influence measurements. Consequently, CT findings should always be interpreted within the overall clinical context.

Artificial intelligence represents one of the most promising future developments in CT-based PE assessment. Automated quantification of RV dimensions, ventricular volumes, and clot burden has the potential to standardize reporting and reduce interobserver variability. As these technologies become integrated into routine radiology workflows, CT-derived risk stratification may become faster, more objective, and more reproducible.

Overall, current evidence supports routine evaluation of CT-derived RV dysfunction in every patient with acute PE. Rather than serving solely as a diagnostic examination, CTPA should be considered a comprehensive risk stratification tool capable of influencing early therapeutic decision-making.

Figure 1 :CT-Based Risk Stratification Algorithm in Acute Pulmonary Embolism



5. Conclusion

CT-derived assessment of right ventricular dysfunction plays a central role in the contemporary management of acute pulmonary embolism. Among available imaging parameters, the RV/LV diameter ratio remains the most reliable predictor of adverse outcomes. Additional CT findings, including interventricular septal bowing, contrast reflux into the inferior vena cava, and right atrial enlargement, provide complementary prognostic information.

Integration of CT findings with clinical evaluation and cardiac biomarkers enables more accurate identification of patients at risk for hemodynamic deterioration and supports individualized treatment strategies. Future studies should focus on validating AI-assisted CT analysis and determining its impact on clinical outcomes and therapeutic decision-making.

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