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Case Report

Journal of Emergency Medicine Case Reports

# A Rare Complication of Acute Otitis Media: Brain Abscess Case Report

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## Abstract

While meningitis is defined as inflammation of the pia and arachnoid membranes,brain abscess is defined as suppurative infection occurring in the parenchymal tissue of the brain.The source of brain abscesses are usually middle ear infections, mastoiditis, frontal and nasal sinusitis. A 69-year-old female patient was brought to the emergency room by her relatives with a complaint of confusion.It was learned that the patient had pain in her right ear for 3 days and antibiotic treatment was started the day before due to infection in the eardrum.Infective blood parameters were high.A lesion containing air density was observed in the patient's brain CT scan in the right temporal fossa.Upon this radiological finding, a contrast-enhanced brain MRI was performed and a lesion containing air-fluid leveling was detected in the right temporal lobe.A tube was inserted into the eardrum of the patient by the ear, nose and throat department.She was transferred to intensive care for follow-up and treatment after the procedure. Acute otitis media is an infection that can progress with intracranial complications if not treated properly and if left untreated.It should not be forgotten that meningitis and brain abscess may also occur in a patient with altered consciousness diagnosed with acute otitis media.

**Keywords:** Acute otitis media, brain abscess, emergency department

## Introduction

Otitis media is defined as inflammation of the middle ear. Complications of otitis media arise when infectious agents and their toxic products spread beyond the pneumatized cavities of the temporal bone and the mucosal boundaries that surround these structures. Many of these complications are observed following subacute or chronic infections. Serious intracranial complications most commonly occur during acute exacerbations of chronic purulent otitis media, especially in cases associated with cholesteatoma. Posterior cranial fossa abscesses usually develop via spread through the lateral sinus or labyrinth. Cerebellar abscesses may also occur via direct extension from a perisinusal abscess. Most otogenic brain abscesses originate from venous thrombophlebitic lesions. Direct spread through the dura is rare, as the dura mater is highly resistant to infection. However, localized inflammation may lead to thrombophlebitis in adjacent cerebral or cerebellar veins, which may then rapidly extend into the white matter, an area with minimal resistance to infection. This process results in surface necrosis and subsequent abscess formation. The temporal lobe is the most frequently involved site, followed by the cerebellum.

Meningitis is defined as inflammation of the pia and arachnoid membranes that envelop the brain and spinal cord, while a brain abscess is defined as a suppurative infection within the brain parenchyma. The three most common pathogens responsible for purulent meningitis are Haemophilus influenzae, Streptococcus pneumoniae, and Neisseria meningitidis (1). The primary sources of brain abscesses are typically middle ear infections, mastoiditis, and frontal or nasal sinusitis (2). Brain abscesses may arise as sequelae of meningitis or may coexist with it. Although the incidence of brain abscesses has declined with widespread antibiotic use, their medical management remains challenging. Meningitis is the most frequently reported intracranial complication of acute otitis media, followed by brain abscess and lateral sinus thrombosis (3–5). Over the past 50 years, increased access to healthcare, improved social welfare, and more effective medical interventions have led to a tenfold decrease in mortality rates associated with otitis media complications (6).

This report aims to emphasize the importance of early recognition and intervention in similar clinical scenarios.

## Case Report

A 69-year-old woman was brought to the emergency department by her family with complaints of decreased responsiveness,

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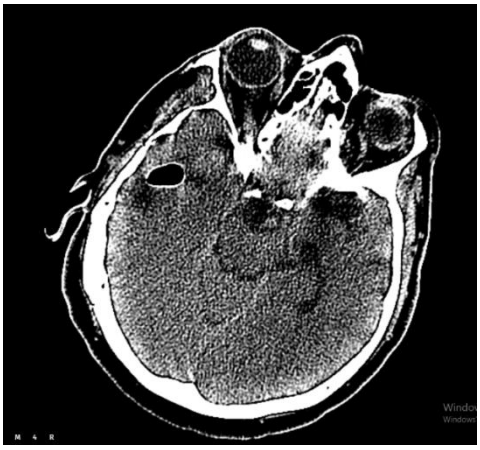

**Figure 1.** BT axial images

speech difficulties, confusion, and failure to recognize familiar people. She exhibited no focal neurological deficits such as hemiparesis, cranial nerve involvement, or seizures. History revealed that the patient had experienced right ear pain for three days and had started antibiotic treatment the previous day due to a diagnosed tympanic membrane infection. She had no known chronic illnesses or history of regular medication use. There was also no prior history of otological disease, tympanic membrane perforation, or otologic surgery.

On admission, her vital signs were as follows: body temperature 38 °C, blood pressure 140/100 mmHg, pulse 100/min, and $SpO_2$ 97%. On physical examination, the patient's general condition was moderate to poor, she was somnolent, disoriented, and uncooperative. Otoscopic examination of the right ear revealed a perforated tympanic membrane with purulent discharge. Laboratory investigations showed WBC: 18,870/mm³, CRP: 311 mg/L, AST: 147 U/L, ALT: 109 U/L, and ALP: 163 U/L.

A non-contrast brain CT scan revealed a lesion with air density in the right temporal fossa and a loss of aeration in the right mastoid air cells (Figure-1). Subsequent contrast-enhanced brain MRI demonstrated a lesion with air-fluid levels in the right temporal lobe, with no significant enhancement after contrast administration. There was also a marked loss of aeration in the middle ear and adjacent mastoid cells (Figure-2,3).

Lumbar puncture was performed after ophthalmologic evaluation, under the clinical suspicion of meningitis and brain abscess, taking into account the risk-benefit ratio. CSF microscopic examination revealed abundant leukocytes, and Streptococcus pneumoniae was isolated in the CSF culture.

No surgical intervention was recommended by the neurosurgery team. However, an ear tube was inserted by the otolaryngology department after a joint evaluation. As the abscess size was borderline for surgical indication, it was decided to proceed with close monitoring following tympanic membrane drainage. The patient was subsequently transferred to the intensive care unit (ICU) for further follow-up and medical treatment.

During ICU follow-up, the patient's condition deteriorated further and elective intubation was performed. After 10 days of intubation, she was successfully extubated, transferred to the general ward, and later discharged with full recovery.

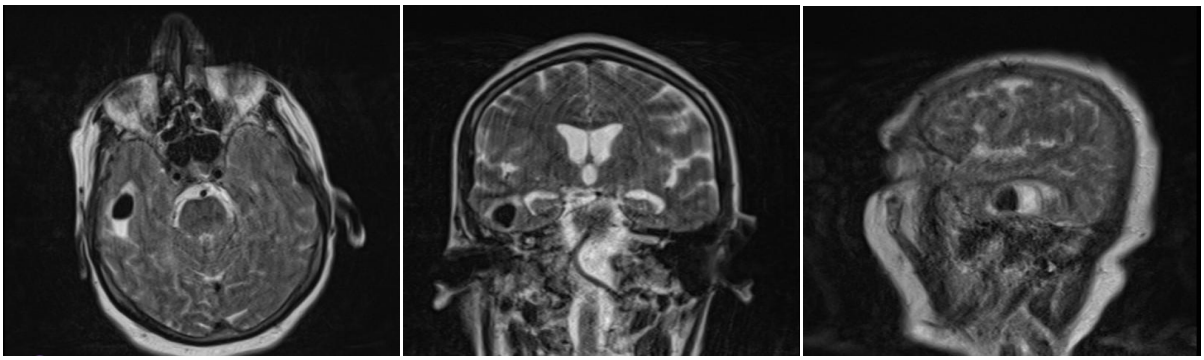

**Figure 2.** MR-T2 sequence axial, sagittal, coronal section images

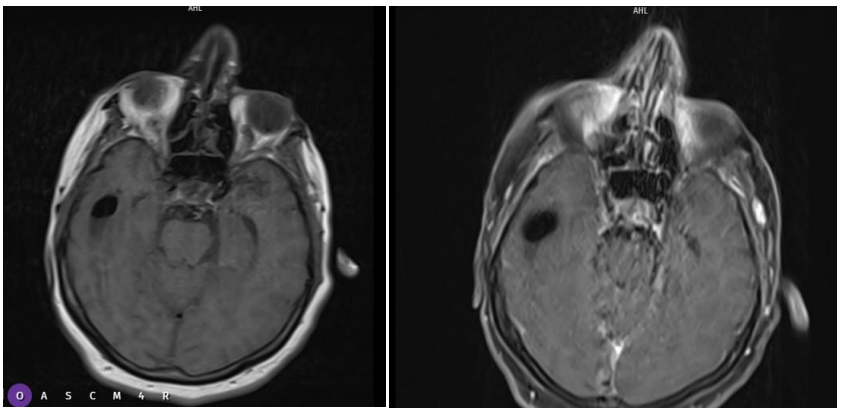

**Figure 3.** MR-T1 sequence non-contrast and contrast images

## Discussion

Meningitis is the most commonly reported intracranial complication of acute otitis media. In more advanced cases, severe complications such as brain abscess and lateral sinus thrombosis may also occur (3–5). In this report, we present a case of acute otitis media complicated by a brain abscess, confirmed by neuroimaging and CSF analysis following a decline in consciousness.

The leading pathogens of purulent meningitis include Haemophilus influenzae, Streptococcus pneumoniae, and Neisseria meningitidis. Purulent meningitis is characterized by inflammation of the leptomeninges and typically results from bacterial infections, posing significant risks if not promptly treated. Clinical manifestations may include high fever, severe headache, and vomiting. Antibiotic therapy is the mainstay of treatment, and early diagnosis is crucial to prevent permanent neurological damage (1,7). In our case, Streptococcus pneumoniae was identified in the CSF culture.

For brain abscess treatment, broad-spectrum antibiotics capable of penetrating the blood-brain barrier are required, and therapy is typically continued for 3 to 6 weeks after clinical and laboratory improvement. Surgical drainage can aid both in microbiological diagnosis and in reducing abscess size. The decision for surgery depends on abscess size and location. Combined medical and surgical treatment has shown favorable outcomes, particularly when the pathogen is identified. However, in small or multiple abscesses, medical management alone may be appropriate. If the abscess enlarges during follow-up, surgical reassessment is warranted (8).

In the present case, surgical drainage via craniotomy was not recommended. Instead, a tympanic membrane tube was inserted for drainage by the otolaryngology team. Antibiotic therapy was guided by culture sensitivity results.

Due to the inability to obtain further information regarding the patient's treatment course following emergency department management, our presentation of the post-emergency clinical and therapeutic process is limited.

## Conclusion

Acute otitis media is an infection that may progress to life-threatening intracranial complications if not promptly and adequately treated. In patients with altered mental status and a diagnosis of acute otitis media, clinicians should maintain a high index of suspicion for complications such as meningitis and brain abscess. Early diagnosis and multidisciplinary intervention are critical for improving clinical outcomes.

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Case Report

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# Spontaneous Retroperitoneal Hemorrhage with Psoas Hematoma in an Elderly Patient: A Diagnostic and Therapeutic Challenge

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## Abstract

Spontaneous retroperitoneal hemorrhage (SRH) is a rare and potentially life-threatening condition, often challenging to diagnose and manage, especially in elderly patients with multiple comorbidities. Psoas hematoma is a known complication that can be associated with anticoagulation therapy. A 93-year-old male patient with a history of atrial fibrillation, chronic heart failure, and anticoagulant use (apixaban) presented with complaints of dizziness following a fall in the bathroom. He was initially assessed in the neurology clinic for potential cognitive symptoms; however, while awaiting assessment, he experienced a syncopal episode, resulting in his transfer to the emergency room. The patient had a significant reduction in hemoglobin levels, necessitating additional examination. Imaging revealed an 8 cm left psoas hematoma with active extravasation. The patient received a blood transfusion, vitamin K, and therapeutic interventions, including interventional radiology, which effectively halted the hemorrhage. This case highlights the diagnostic complexity and therapeutic challenges posed by spontaneous retroperitoneal hemorrhage in elderly patients, especially those on anticoagulant therapy. Early recognition and appropriate intervention are crucial for patient recovery.

**Keywords:** Anticoagulant therapy, psoas hematoma, spontaneous retroperitoneal hemorrhage, transarterial embolization

## Introduction

Spontaneous retroperitoneal hemorrhage (SRH) is a rare and potentially fatal disease, especially in geriatric people. It may result from trauma, anticoagulant therapy, or other underlying vascular disorders. Psoas hematoma, a blood collection within the psoas muscle, is a common presentation in these cases. The risk of bleeding in elderly adults is heightened by anticoagulants, such as direct oral anticoagulants (DOACs) utilized for atrial fibrillation. Timely diagnosis and intervention are essential to avert morbidity and mortality. Previous studies highlight the role of early imaging and multidisciplinary management in addressing such cases effectively (1, 2). Moreover, new publications highlight that transarterial embolization and cautious therapy are crucial depending on the severity and evolution of the hematoma (3, 4).

## Case Report

A 93-year-old male patient was admitted to the emergency department after experiencing a syncopal episode. The patient had a history of atrial fibrillation and chronic heart failure and was receiving anticoagulant medication (apixaban). He presented to the neurology clinic the previous day with symptoms indicative of dementia, such as confusion and forgetfulness. While awaiting his neurology consultation, he suffered vertigo and subsequently lost consciousness. Upon assessment at the emergency department, his Glasgow Coma Scale (GCS) was 15, and his vital signs indicated hypotension and tachycardia (Blood pressure: 70/40 mmHg, Pulse: 110bpm, $SaO_2$: 90, Temperature: 36.2°C).

Following the recent fall in the restroom, a head computer tomography (CT) imaging was conducted, revealing no acute intracranial abnormalities. Nevertheless, more examination was necessary owing to his hypotension and tachycardia. His hemoglobin level declined by 5 g/dL since the last medical visit (The initial hemoglobin (Hb) value was 16.3g/dL, followed by 11.3g/dL). A CT scan of the abdomen revealed that the left psoas muscle enlarged compared to its symmetry, measuring 78x60 mm at its widest point in the axial plane. There is a heterogeneous appearance suggestive of hematoma within it, and active contrast extravasation is also observed in the left psoas muscle (Figure-1 and Figure-2).

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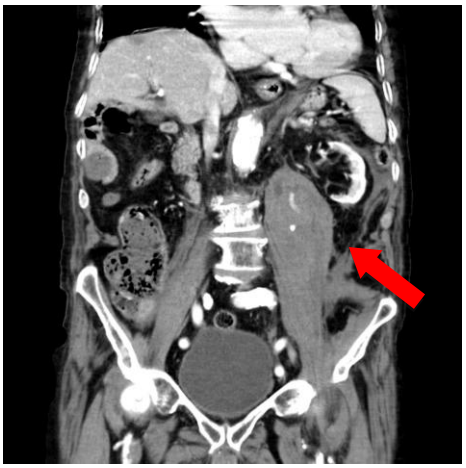

**Figure 1.** A CT scan of the abdomen showing an active contrast extravasation (marked with the arrow) on left psoas muscle, suggestive of ongoing bleeding

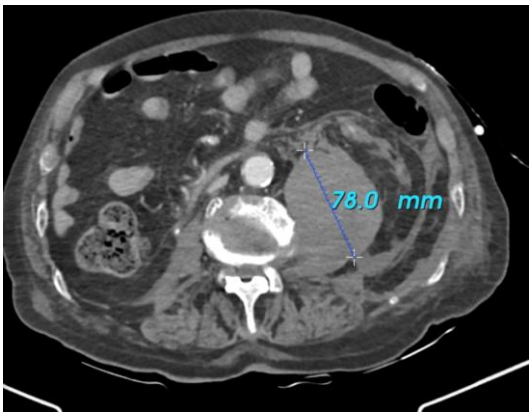


**Figure 2.** A coronal CT scan of the abdomen showing the left psoas muscle enlarged compared to its symmetry, measuring 78x60 mm at its widest point in the axial plane

Given the patient's anticoagulation therapy and the severity of the bleeding, the patient was administered 2 units of packed red blood cells, Vitamin K, and transferred to the intensive care unit (ICU) for closer monitoring and further management. He underwent interventional radiology, which confirmed cessation of the bleeding without requiring additional procedures.

Post-procedure, the patient developed Grey Turner's sign, an indication of retroperitoneal bleeding. He continued to receive supportive care, including blood transfusions and medical management. On day 2, his hemoglobin remained stable (The Hb trend was 12.7g/dL, 11.8g/dL, 10.8g/dL, 10.4g/dL, 10.8g/dL), and the patient was transferred to the general ward. The patient was then initiated on low-molecular-weight heparin and discharged after five days without any further problems.

## Discussion

A rare clinical condition known as spontaneous retroperitoneal hemorrhage (SRH) can cause nonspecific symptoms like tachycardia, hypotension, and abdominal pain, which sometimes causes delays in diagnosis. SRH is a condition that is difficult to diagnose due to nonspecific symptoms and can be fatal if early diagnosis and intervention are not achieved (5). The use of anticoagulant drugs, which raise the risk of bleeding, may make SRH in older individuals even more difficult. Because of its vascularity, the psoas muscle, which is situated in the retroperitoneal region, frequently becomes the location of hematoma formation. Recent research indicates that interventional radiology plays a critical role in limiting surgical intervention and attaining hemostasis (4, 6). Furthermore, a high rate of technical and clinical success has been demonstrated by transarterial embolization in the management of such instances (4).

This patient's anticoagulant treatment with apixaban (Eliquis®) most certainly contributed significantly to his vulnerability to bleeding. Initial abdominal CT imaging was essential for detecting the psoas hematoma and assessing the necessity for intervention. Interventional radiology has emerged as a crucial modality in the management of these situations, facilitating non-surgical hemostasis. The hazards linked to anticoagulant medication, encompassing dual antiplatelet therapy and Vitamin K antagonist therapy, are emphasized in earlier reports and reflect the findings in this instance (2, 7).

This case emphasizes the importance of considering SRH in elderly patients presenting with nonspecific symptoms and the need for prompt imaging and management. The development of Grey Turner's sign post-procedure is a classic but rare indicator of retroperitoneal bleeding. Close monitoring and appropriate blood product replacement are key components of treatment (1, 6).

## Conclusion

This case demonstrates the difficulty in identifying and managing spontaneous retroperitoneal hemorrhage in older patients, especially those receiving anticoagulation medication. To maximize patient outcomes, early detection via imaging and prompt interventions, such as blood transfusions and interventional radiology, are crucial. Additionally, this case emphasizes the significance of tailored management plans for senior citizens with numerous comorbidities. Further research into risk mitigation strategies, including novel anticoagulants with reduced bleeding profiles, could advance care standards for such patients.

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Case Report

Journal of Emergency Medicine Case Reports

# First Fatal Human Case of Beta-Cyfluthrin Intoxication in Türkiye: A Case Report

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## Abstract

Cyfluthrin, a type II synthetic pyrethroid, is extensively used in agricultural and veterinary practices. While generally considered to have low mammalian toxicity, significant exposure to concentrated formulations can lead to systemic toxicity and multiorgan failure. Human fatalities due to cyfluthrin are rare and not well-characterized in the literature. We report the case of a 67-year-old female shepherd with no known comorbidities who presented to the emergency department approximately 24 hours after dermal and possible inhalational exposure to a veterinary insecticide containing 25 g/L beta-cyfluthrin in emulsifiable concentrate form. She had applied the formulation directly to livestock without protective equipment. Within hours, she developed acute retrosternal chest pain, vomiting, and transient loss of consciousness. Upon arrival to the emergency department, she was hypotensive, bradycardic, and hypoxic. Laboratory analysis revealed severe metabolic acidosis, hyperglycemia, markedly elevated hepatic and cardiac enzymes, acute kidney injury, and coagulopathy. Imaging showed a moderate pericardial effusion without tamponade. Despite intensive supportive care, including high-dose sodium bicarbonate, N-acetylcysteine, and fluid resuscitation, her condition deteriorated, culminating in disseminated intravascular coagulation, shock, and death on ICU day 8. This case illustrates the potential lethality of beta-cyfluthrin exposure in humans and underscores the importance of early recognition, protective practices, and aggressive management in suspected pyrethroid poisoning.

**Keywords:** Cyfluthrin, insecticides, multiorgan failure, pesticide poisoning, rural population

## Introduction

Synthetic pyrethroids are a widely used class of insecticides derived from natural pyrethrins, known for their high efficacy against pests and relatively low mammalian toxicity. Among them, cyfluthrin, a type II pyrethroid containing a cyano group, is extensively applied in agricultural, veterinary, and public health settings for the control of a broad spectrum of insects (1,2). Its mechanism of action involves prolonged activation of voltage-gated sodium channels in nerve cells, resulting in sustained depolarization and repetitive neuronal firing, ultimately leading to paralysis in target species (3).

Despite their widespread perception as "safe" insecticides for humans, pyrethroids—particularly in their concentrated forms—may cause significant systemic toxicity through dermal, inhalational, or oral exposure. Human poisoning typically results in mild neurological and gastrointestinal symptoms; however, more severe presentations including seizures, pulmonary edema, shock, and multiorgan dysfunction have been reported, particularly following large or prolonged exposures to type II pyrethroids such as cyfluthrin and beta-cyfluthrin (4–6). The severity of toxicity depends on multiple factors including formulation concentration, route and duration of exposure, and individual susceptibility.

Beta-cyfluthrin is a more potent stereoisomer-enriched variant of cyfluthrin used in many commercial emulsifiable concentrate products for livestock and crop protection. In Türkiye, these formulations are commonly used in rural settings with limited awareness of personal protective measures. Literature on fatal human exposures to cyfluthrin is sparse, with most case reports describing full recovery after supportive care (5,6). A fatal case of human beta-cyfluthrin intoxication following dermal exposure has not been previously documented in the medical literature.

We present a rare and fatal case of presumed cyfluthrin intoxication in a shepherd who developed progressive lactic acidosis, ischemic hepatitis, coagulopathy, and ultimately death following unprotected dermal and inhalational exposure to a 25 g/L beta-cyfluthrin emulsifiable concentrate preparation. This report highlights the potential lethality of improperly handled pyrethroid insecticides and underscores the importance of early recognition and aggressive supportive care in similar presentations.

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## Case Report

A 67-year-old female shepherd with no known medical history was brought to the emergency department (ED) by ambulance after collapsing in a remote pasture area. According to family members, approximately 24 hours earlier, the patient had used an insecticidal veterinary preparation containing 25 g/L beta-cyfluthrin in emulsifiable concentrate form to treat her goats for fleas. The product was applied without gloves or respiratory protection in an open-air setting. She reportedly complained of retrosternal chest pain, fatigue, and vomiting several hours later, and then became increasingly confused and weak over the next day.

Upon presentation to the ED, her vital signs were: blood pressure 65/45 mmHg, heart rate 65 bpm, respiratory rate 22 breaths per minute, oxygen saturation 88% on room air, and temperature 36.5°C. She was pale, diaphoretic, and mildly lethargic, but oriented to person and place. Cardiac auscultation revealed muffled heart sounds; bilateral basal crackles were heard on lung examination. Her abdomen was soft and non-tender. No obvious dermal lesions or rashes were noted, though her hands and arms had a faint chemical odor. Neurological examination showed no focal deficits; GCS was 13 (E4V4M5).

Initial laboratory evaluation revealed severe metabolic acidosis (pH: 6.95, bicarbonate: 14 mmol/L), hyperglycemia (glucose: 420 mg/dL), elevated liver enzymes (AST: 2913 U/L, ALT: 2205 U/L), LDH >2726 U/L, and cardiac injury (troponin T: 62 ng/L). Renal function was impaired (creatinine: 2.86 mg/dL; urea: 148 mg/dL), and coagulation parameters were abnormal (INR: 1.51; fibrinogen: 244 mg/dL; D-dimer: 6400 ng/mL). Electrocardiography revealed sinus bradycardia. Transthoracic echocardiography demonstrated a 2–3 cm circumferential pericardial effusion without tamponade physiology. Chest radiograph and thoracoabdominal CT revealed mild bilateral pleural effusions and pericardial fluid, with no signs of pulmonary embolism.

Due to the lack of available toxicology screening for pyrethroids at our center, the diagnosis of cyfluthrin poisoning was made clinically, based on the exposure history, symptom profile, and exclusion of alternative causes. She was admitted to the intensive care unit with a presumptive diagnosis of pyrethroid-induced lactic acidosis, ischemic hepatitis, and developing multiorgan dysfunction syndrome. Initial therapy included high-dose intravenous sodium bicarbonate infusion, N-acetylcysteine, broad-spectrum antibiotics, and aggressive fluid resuscitation. Despite supportive care, she remained hypotensive, oliguric, and showed signs of progressive hepatic injury.

By ICU day 2, she developed disseminated intravascular coagulation with thrombocytopenia (platelets: 55 $\times 10^3/\mu L$), elevated D-dimer (>21,000 ng/mL), and worsening liver enzyme levels. Hemodialysis could not be initiated due to hemodynamic instability. Multidisciplinary consultations concluded that her deterioration was consistent with a systemic inflammatory response syndrome secondary to pesticide intoxication. On day 8, the patient experienced pulseless electrical activity arrest. Cardiopulmonary resuscitation and multiple doses of epinephrine failed to achieve return of spontaneous circulation. She was declared dead after 45 minutes of advanced life support.

## Discussion

Cyfluthrin is a widely used type II synthetic pyrethroid that targets voltage-gated sodium channels, resulting in repetitive neuronal firing and eventual paralysis in insects. While considered to have low mammalian toxicity due to hepatic detoxification pathways, significant dermal or inhalational exposure to concentrated formulations—such as emulsifiable concentrates (ECs)—may lead to serious systemic effects in humans (1,2).

In this case, exposure occurred during unsupervised outdoor application of a veterinary insecticide containing 25 g/L beta-cyfluthrin, a formulation commonly marketed in Türkiye under various trade names. The patient's symptomatology developed rapidly and progressed over the subsequent 24 hours, suggesting substantial percutaneous and/or inhalational absorption. Though gastrointestinal ingestion was not confirmed, nausea, vomiting, and central nervous system symptoms are consistent with systemic absorption and early toxicity (3,4).

Previous reports in the literature describe largely mild-to-moderate symptoms following cyfluthrin exposure—often paresthesia, dizziness, nausea, or tremors (1,3). However, more severe cases have been documented, including seizures, pulmonary edema, and shock, particularly after ingestion or exposure to high-concentration commercial products (5,6). Isbister et al. reported a series of concentrated pyrethroid exposures in which some patients developed metabolic acidosis and cardiovascular compromise (6). Similar to our case, these presentations required intensive care, but fatality was rare—highlighting the exceptional severity of our patient's outcome.

The development of pericardial effusion in this patient is particularly noteworthy, as it is infrequently reported in pyrethroid poisoning. Its etiology may involve toxin-induced pericardial inflammation or secondary capillary leak syndrome. The profound hepatic injury observed is consistent with ischemic hepatitis and has been attributed in animal models to mitochondrial dysfunction, oxidative stress, and direct hepatocellular apoptosis following cyfluthrin exposure (7). The metabolic acidosis (pH 6.95) was life-threatening and likely multifactorial-related to tissue hypoperfusion, lactic acidosis, and impaired clearance by the liver and kidneys.

A major limitation was the absence of confirmatory toxicological analysis, including serum or urinary cyfluthrin or metabolite levels (e.g., 3-phenoxybenzoic acid). However, this diagnostic barrier is common in rural or resource-limited centers, where diagnosis is typically clinical. Additionally, product retrieval and chemical analysis were not possible due to lack of labeling and disposal before admission. The establishment of regional toxicology reference laboratories capable of detecting pyrethroid metabolites would greatly enhance diagnostic accuracy in such rural poisonings. This case highlights the critical need for training programs on pesticide safety among rural agricultural workers and livestock handlers.

## Conclusion

This case underscores that beta-cyfluthrin, while perceived as low-risk, may be fatal under certain exposure scenarios. The delayed presentation—approximately 24 hours post-exposure-likely contributed to diagnostic and therapeutic delay. Notably, no antidote exists for cyfluthrin poisoning, and management remains entirely supportive. Early recognition, organ function monitoring, and aggressive critical care are essential. Education on pesticide handling and mandatory labeling may reduce the risk of such fatalities in rural areas.

**Conflict of interest:** The authors declare no conflicts of interest.

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Case Report

Journal of Emergency Medicine Case Reports

# A Rare Cause of Hamman Syndrome A Singing-Induced Case Report

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## Abstract

Pneumomediastinum is a rare condition characterized by the presence of air in the mediastinal space. Hamman's syndrome, an affliction that arises from disparate pressures within the pulmonary interstitium and alveoli, often manifests subsequent to the Valsalva maneuver. The crepitation perceived during systolic contraction, a phenomenon known as Hamman's sign, constitutes a pivotal element in the diagnostic process. A 22-year-old male patient presented to the emergency department with atypical chest tightness and dyspnea that began after singing. Cardiac auscultation revealed crepitation in the precordial area. Chest X-ray revealed air densities around the trachea. Given the absence of esophageal or bronchial rupture, in conjunction with the presence of Hamman's sign, a diagnosis of Hamman's syndrome was established. The patient was discharged after undergoing follow-up. Since a serious condition such as Hamman syndrome may develop even in dyspnea that develops after daily activity, necessary investigation and treatment should be performed.

**Keywords:** Dyspnea, hamman's syndrome, pneumomediastinum

## Introduction

Pneumomediastinum (PM) is defined as the presence of air in the mediastinum. Pneumomediastinum is usually caused by various predisposing factors including trauma, acute asthma attack, strenuous physical activity, excessive vomiting or severe cough (1). However, spontaneous pneumomediastinum (SPM) occurring without a clear triggering factor is defined as Hamman syndrome. Hamman syndrome was first described by Louis Hamman in 1939 (2).

Hamman syndrome is a picture that develops due to the pressure difference between the pulmonary interstitium and alveoli, which usually develops as a result of valsalva maneuver. The air coming into the mediastinum is due to the leakage of air that occurs due to this pressure difference (3). Hamman Syndrome has been reported to be associated with certain lung diseases such as asthma, COPD, interstitial lung diseases and bronchiectasis (4). It has also been detected in situations such as coughing, sneezing, vomiting, defecation and birth (5). Hamman Syndrome associated with singing has never been reported in the literature before.

In pneumomediastinum, chest pain and dyspnea are the most common complaints. In addition to these complaints, swelling and compression effect on the neck may also be observed. Hamman syndrome is a self-limiting clinical picture with a generally good prognosis. No special treatment is needed in follow-up (6,7). Crepitation heard with systolic contraction during auscultation of the heart (Hamman's sign) plays an important role in the diagnosis of Hamman syndrome (8).

In this case report, we describe a patient diagnosed with Hamman syndrome that occurred after a concert.

## Case Report

The patient was a 22-year-old male who presented to the emergency department with atypical chest pain and persistent sensation in the throat. These symptoms began the day before his arrival, following his return home from a concert that had taken place during the night. Upon inquiry into his medical history, it was ascertained that he did not have any chronic diseases and was not taking any medication.

The vital parameters at the time of admission were as follows: body temperature 36.6°C, heart rate 83/min, blood pressure 121-79 mmHg, and saturation 98% (without oxygen). The electrocardiogram (ECG) of the

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patient showed sinus rhythm. The patient was found to be in good general condition, exhibiting consciousness, orientation, and cooperation. Physical examination revealed crepitation upon palpation of the skin in the supraclavicular region. Auscultation revealed crepitation in the upper thoracic region and in the precordial region during cardiac auscultation. Conventional laboratory tests did not reveal any pathological parameters.

Chest radiography was performed to rule out pneumothorax and pneumomediastinum, which were suspected based on physical examination and laboratory test results. The diagnosis of pneumomediastinum was made when air densities were observed around the trachea and there was no pneumothorax line in the lung parenchyma on chest radiography (Figure-1). Electrocardiography (ECG) was performed for other differential diagnoses for chest pain in the patient and it was seen that he had normal sinus rhythm. In blood tests, since there was no cardiac enzyme elevation and acute phase reactants were normal, pericarditis, myocardial infarction, and pneumonia were excluded. Since the patient did not have a history of trauma, trauma-related pathologies were not considered.

Following the diagnosis, the patient was administered nasal oxygen, broad-spectrum antibiotics, and analgesia, and subsequently admitted to the thoracic surgery service. Since the approach of our hospital's thoracic surgery clinic is prophylactic antibiotics in pneumomediastinum, the patient was started on prophylactic antibiotics. During subsequent follow-ups, no evidence of esophageal or bronchial rupture was observed. The patient was diagnosed with Hamman syndrome, a diagnosis that was made on the basis of the presence of Hamman's sign.

The patient was discharged after his complaints regressed and the pneumomediastinum regressed on control chest radiography. At the time of discharge, there was no pathology in the patient's vital parameters and his clinical complaints regressed.

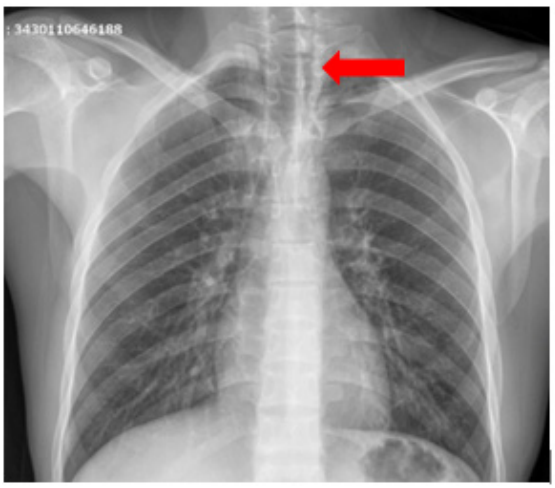

**Figure 1.** The figure presents a chest X-ray image of the patient. The red arrow indicates the presence of air densities surrounding the trachea

## Discussion

Pneumomediastinum has been observed in young and healthy individuals. However, delays in diagnosis can lead to potentially fatal complications, including mediastinitis. The absence of specific diagnostic findings necessitates clinical suspicion for its identification. Consequently, it is imperative for medical professionals to possess a comprehensive understanding of the clinical manifestations associated with pneumomediastinum. Our case underscores the significance of Hamman's sign, a rare clinical finding associated with pneumomediastinum. Informed consent was obtained from the patient.

The literature suggests that pneumomediastinum typically manifests between the ages of 20 and 25, predominantly in healthy, thin men (9). A case series published by Morgan et al. documented a mean age of 23 years among patients, with 72% of cases reported as male (10). The age and gender of our case were found to be compatible with the literature.

While the diagnosis of pneumomediastinum should first be suspected, imaging is absolutely necessary for a definitive diagnosis. Chest X-rays have been shown to have a sensitivity range of 91-95% and a specificity of 70% in diagnosing pneumomediastinum (13). However, a chest computed tomography (CT) scan may be useful for small pneumomediastinum that cannot be detected on X-ray (11,12). In the present case, chest radiography was sufficient for diagnosis.

The treatment of pneumomediastinum is typically approached in a conservative manner. However, the role of hospitalization, the administration of 100% oxygen supplementation, and prophylactic antibiotic administration remains a subject of debate (13). The treatment approach for pneumomediastinum is generally symptomatic, and the condition often resolves spontaneously. In cases of more severe pneumomediastinum, surgical intervention may be necessary, although it is a rare occurrence (14). There is an absence of compelling evidence in the extant literature to support the use of oxygen therapy or prophylactic antibiotic treatment in such cases. However, both were administered in the present case. Given the patient's reported pain, analgesic treatment was administered as a component of the symptomatic treatment.

In a study by Sadarangani et al., three factors contributing to pressure changes leading to pneumomediastinum were identified: increased alveolar pressure (Valsalva maneuver), decreased pleural pressure (Muller maneuver), and decreased interstitial pressure (hyperventilation). Raptopoulos et al. reported cases of pneumomediastinum developing during sports activity (15). Additionally, a case of pneumothorax resulting from shouting has been documented (16). A comprehensive literature review revealed 12 documented cases of pneumomediastinum resulting from shouting (17). However, a thorough examination of the extant

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literature revealed a paucity of cases documenting pneumomediastinum in association with singing. A single case of pneumoretrofaringeum associated with singing was documented (18). Our case represents a novel occurrence of Hamman syndrome in the context of singing, a finding that has not been previously documented in the extant literature.

## Conclusion

Shortness of breath that develops after a normal daily activity such as singing should not be taken lightly as it can cause Hamman Syndrome, which can be fatal.

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Case Report

Journal of Emergency Medicine Case Reports

# Deaths due to Bull Attack in Artvin: Case Series

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## Abstract

Aggressive and unpredictable behaviors of livestock, particularly bulls, present a significant risk of injury and death to farmers involved in animal husbandry. Given the cultural importance of bull breeding in Artvin, analyzing and evaluating the injuries that occur in this context is crucial. This study aims to present cases of bull-related injuries resulting in autopsy in Artvin, highlighting fatal injuries that may occur during animal handling. We retrospectively analyzed cases of individuals who died due to attacks by the cattle they raised among the autopsies performed at the Artvin Forensic Medicine Branch Directorate between 2016 and 2024. Four cases were identified where individuals succumbed to attacks by the bulls. In the autopsies, three of these cases exhibited fractures of the sternum, ribs, and spine, along with internal organ injuries. One individual died from head trauma and cerebral hemorrhage caused by a bull kick. For those engaged in bull breeding, the physical strength and behavioral responses of the animals can lead to head trauma, internal organ damage, and multiple bone fractures. Therefore, the use of protective equipment and its widespread adoption among all workers is essential for ensuring the safety of individuals involved in animal husbandry.

**Keywords:** Autopsy, bull, fatal injury

## Introduction

Attacks by animals such as bulls, horses, pigs, or dogs can cause high-energy trauma, leading to severe injury or death (1). Bull attacks are particularly common in Latin American countries due to the use of bulls in demonstrations (2). Additionally, aggressive and unpredictable animal behavior is observed in regions of Turkey where agriculture and animal husbandry are intensive (3-5). Such encounters can result in injuries from crushing, chewing, goring, or biting; these injuries can range from ecchymosis and lacerations on the skin to damage to internal organs and vessels, bleeding in body cavities, and limb amputations. Traumas to the head, chest, and abdomen are especially life-threatening (6-9). The bull statue at the entrance of Artvin city and the bull fighting festival, which strengthens social ties, illustrate that bull breeding has become a symbol of the city and is integratedin to its sociocultural structure (10, 11). Therefore, analyzing the fatal injuries caused by bull attacks in Artvin is of great importance. This study aims to present cases of bull-related injuries resulting in autopsy in Artvin, highlighting fatal injuries that may ocur during animal handling.

In this study, autopsies performed at the Artvin Forensic Medicine Branch Directorate from January 1, 2016, to December 31, 2024, were retrospectively analyzed. Information regarding the cases, including age, gender, crime scene investigation images, witness statements, external examinations, and autopsy findings, was obtained from archive records and the National Judicial Network Project (UYAP) system.

This study was presented at a meeting of the Ministry of Justice's Forensic Medicine Institute Education and Scientific Research Commission on March 18, 2025, and was approved under decision number 21589509/2025/349. The research was conducted in accordance with the principles of the Declaration of Helsinki. Additionally, the Artvin Coruh University Scientific Research and Publication Ethics Committee determined, in its decision dated March 9, 2025, and numbered E-18457941-050.99-171952, that approval for compliance with scientific research and publication ethics is not required for our study.

## Case Reports

### Case-1

In October 2020, an investigation was launched into the suspicious death of an 87-year-old man in a village in central Artvin. The man's body was discovered near the area

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where his bulls were grazing. An autopsy was performed at the Artvin Forensic Medicine Branch Directorate to determine the exact cause of death. The autopsy revealed a hemothorax, lacerations in both the lung and liver, and signs of internal hemorrhage in the thoracic and abdominal regions. Additionally, deformity was noted in the thoracic region, along with comminuted and hemorrhagic fractures in the sternum and ribs. Hemorrhagic fractures were also found in the 5th and 6th cervical vertebrae.

**Case-2**

In May 2022, the body of a 66-year-old man was found in a village in Artvin province. When the family did not receive any news from him, they went to the grazing area where the bulls were located, and there they discovered his body adjacent to the spot where the animals were. An autopsy was performed at the Artvin Forensic Medicine Branch. The autopsy revealed a laceration in the lung and subarachnoid hemorrhage (SAH) in the brain. Additionally, signs of internal hemorrhage in the thoracic region, comminuted and hemorrhagic fractures in the sternum and ribs, and a hemorrhagic fracture in the cervical spine (C7) were also found.

**Case-3**

In September 2024, an 84-year-old man living in a village in the district of Artvin was found dead by his neighbor on the farm where he raised bulls. According to reports, the neighbor went to the scene after hearing shouting and discovered the body. An autopsy was performed at the Artvin Forensic Medicine Branch Directorate to determine the cause of death. The findings revealed comminuted, hemorrhagic fractures in the sternum and ribs, signs of internal bleeding in the thoracic region, and a comminuted, hemorrhagic fracture in the cervical (C4) vertebra.

**Case-4**

In September 2024, a 68-year-old man sustained injuries in a village in the district of Artvin when he was kicked by an animal while tending to his bulls. Conscious after the incident, he was immediately taken to the district central hospital and then transferred to the state hospital in the city center. Brain tomography revealed collapse fractures in the frontal and parietal bones, as well as diffuse subarachnoid hemorrhage. Following these findings, the patient was admitted to the intensive care unit and intubated. Unfortunately, he died later that same day. The cause of death was determined to be skulldome fractures and cerebral hemorrhage.

The findings from all cases are presented in Table-1 and Figure-1.

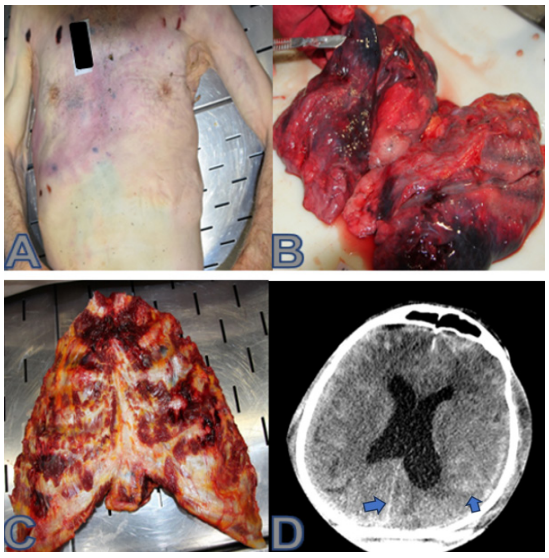


**Figure 1.** A) Hyperaemic deformed appearance of case 1 showing collapse of the chest. B) Contusion and haemorrhage areas in the lung of case 2. C) Multiple rib fractures of case 3 with separation and haemorrhage. D) Tomography image of diffuse subarachnoid haemorrhage in case 4.

## Discussion

Our study found four cases of death resulting from attacks by bulls raised on farms. Autopsies of the cases revealed fractures of the sternum, ribs and spine, as well as internal organ injuries in three of the cases. One case was found to have died as a result of head trauma and cerebral haemorrhage caused by a bull kick. Bull farming poses serious safety and health risks to live stock workers worldwide. The primary reason for these risks is the bulls' large size, sudden mobility, and unpredictable behavior (6). While kicking

**Table 1:** Information and findings of the cases

| Case | Age | Gender | Place of death | Season* | Injured Body Part | Organ injury | Bone fracture | Cause of Death |
|---|---|---|---|---|---|---|---|---|
| 1 | 87 | Male | Rural | Autumn | Chest-Neck | Lung, Liver | Sternum, multiple rib, C5-6 vertebral fracture | Internal Organ Damage and Internal Haemorrhage |
| **2** | 66 | Male | Rural | Spring | Head-Chest-Neck | Brain, Lung | Sternum, multiple rib, C7 vertebral fracture | Internal Organ Damage and Internal Haemorrhage |
| **3** | 84 | Male | Rural | Autumn | Neck | MedullaSpinalis | Sternum, multiple rib, C4 vertebral fracture | Complication due to neck (C4) fracture (Neurogenic Shock) |
| **4** | 68 | Male | Hospital | Autumn | Head | Brain | Frontal andparietal bone | Brain haemorrhage (Subarachnoid Haemorrhage) |

*; Winter: December, January, February, Spring: March, April, May, Summer: June, July, August, Autumn: September, October, November

is more common, head butting and crushing can result in more severe injuries. Additionally, cases of entrapment in confined spaces may occur during the transport of bulls to stables or transport vehicles (3). One study found that these injuries accounted for 0.02% of all traumas (12).

The fact that all of the patients in our study were male and in the advanced age group (meanage: 76.25 years) was found to be consistent with previous literature (4, 5). In a study, it was found that bull injuries were significantly more common in the male gender (198:18 ratio) (13). Activities such as shepherding, village guarding, hunting and outdoor sports, which have historically been attributed to men, bring about a more intense interaction of men with nature (4). This increases the risk of contact with domestic or wild animals and therefore leads to higher mortality rates in men from such contact.

In a study conducted in Poland, a total of 1,872 cases of animal-related injuries were analyzed, revealing that 98 (5.2%) of these cases resulted from bull attacks. It was also emphasized that 94% (n=92) of bull attacks occurred in rural areas, indicating a significant concentration in the geographical distribution of these injuries (1). Consistent with findings in the literature, the fact that all cases in our study occurred in rural areas supports the notion that these locations and certain working conditions constitute important risk factors. Accordingly, implementing necessary safety measures and increasing risk awareness in rural areas are critical to preventing such injuries.

In our study, we found that deaths occurred primarily during the autumn months (September-October), whichalignswiththeexistingliterature(8). Autumn is a time when both calving and milking begin, and dairy cows are gathered into tighter herds for these purposes (4). During this period, animals can experience increased stress, leading to more unpredic table behavior. Especially in autumn, animals gathering in large herds can cause them to exhibit increased stress and aggressive behaviour. In addition, increased work load during this period can cause workers to become tired and careless, increasing the risk of injury. Therefore, the seasonal distribution of injuries highlights the potential impact of agricultural activities, particularly livestock husbandry practices, on workers.

Injuries from bull attacks can occur in various ways and affect different parts of the body. Typical traumatic findings in the thoracic region include “flail chest” due to multiple rib fractures, as well as haemothorax, pneumothorax, and internal organ damage (5). Abdominal injuries are usually penetrating and may include intestinal perforations and severe organ damage, such as lacerations of the liver and spleen. The literature reports that the incidence of abdominal injuries varies between 4% and 64% (14, 15). Additionally, some studies have noted simultaneous diaphragm and jejunum perforations (16). Craniofacial injuries typically result from crushing or kicking blows, manifesting as spinal or skull fractures, as well as subdural, subarachnoid, and extradural haemorrhages, along with brain contusions and lacerations (17). In our study, most cases revealed comminuted fractures of the sternum and vertebrae, accompanied by chest and abdominal visceral injuries. In one case, diffuse subarachnoid haemorrhage developed following a blow to the head. These findings indicate that bull breeding can lead to serious and potentially fatal trauma. At this point, it is of utmost importance to maintain high-level safety measures and conduct regular risk assessments when working with bulls. In addition, providing animal behaviour training that includes topics such as reading animal body language and recognising signs of stres and aggression, informing employees about the use of protective equipment, and implementing emergency scenarios are important for improving safety.

## Conclusion

The physical force and behavioral responses of animals, such as bulls, can cause fatal injuries to individuals engaged in cattle breeding. These injuries often result in head trauma, internal organ damage, and multiple bone fractures. Therefore, it is vital to implement measures such as theuse of protective equipment for the safety of those involved in animal husbandry and to ensure this equipment is distributed among all employees. Additionally, strengthening emergency medical response facilities in rural areas is an essential requirement.

In cases of suspicious deaths in rural areas, particularly those involving multiple, separate rib and vertebral fractures, the possibility of animal intervention should be assessed in relation to the cause of death. Additionally, injuries that may have developed in the internal organs should be examined in detail. In such instances, it is essential to evaluate the findings at the scene of the incident, information obtained from local residents, hospital records, and autopsy results collectively. This comprehensive approach can lead to a more accurate understanding of the cause of death and the mechanism of injury.

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Case Report

Journal of Emergency Medicine Case Reports

# The Spot-on Purple Nuisance-Purple Urine Bag Syndrome

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## Abstract

Purple Urine Bag Syndrome (PUBS) is a rare and often overlooked condition, marked by the purple discoloration of urine, primarily seen in patients with urinary catheterization. We present two cases of PUBS, involving elderly, bed-bound patients with permanent urethra catheters who presented to the Emergency Department with urinary tract infections. Both cases were managed effectively with broad-spectrum antibiotics. The condition arises due to bacterial production of phosphatase and sulfatase, leading to the formation of purple pigment in the urine. While the slight of purple urine may be alarming, PUBS often responds well to prompt diagnosis and proper treatment, leading to favourable outcomes.

**Keywords:** Prolonged Bladder Catheterisation, Purple Urine, Purple Urine Bag Syndrome, Urinary Tract Infection

## Introduction

Purple Urine Bag Syndrome (PUBS) is a rare but significant complication associated with urinary tract infection, primarily occurring in patients with urinary catheterization. This syndrome is characterised by the striking purple discoloration of urine, which can serve as a distinctive indicator for medical professionals. Despite its diagnostic potential, PUBS remains under-recognized, often leading to unnecessary anxiety to the caregivers and families. This is particularly critical in elderly patients with multiple comorbidities, who may not exhibit typical symptoms of urinary tract infection, and may be unable to effectively communicate their discomfort or symptoms. Thus, increasing awareness and understanding of PUBS is essential to ensure timely diagnosis and appropriate management, ultimately alleviating distress for both patients and their caregivers.

## Case Reports

### Case-1

A 67 years old gentleman, with medical history of hypertension, degenerative spine disease, advanced chronic obstructive pulmonary disease, and congestive heart failure presented to the Emergency Department (ED) after being bedbound for eight years and was dependent on a urinary catheter for past eight months due toneurogenic bladder. For the past ten days, he experienced purple discoloration in his urine bag (Figure-1) and reported coffee ground vomitus

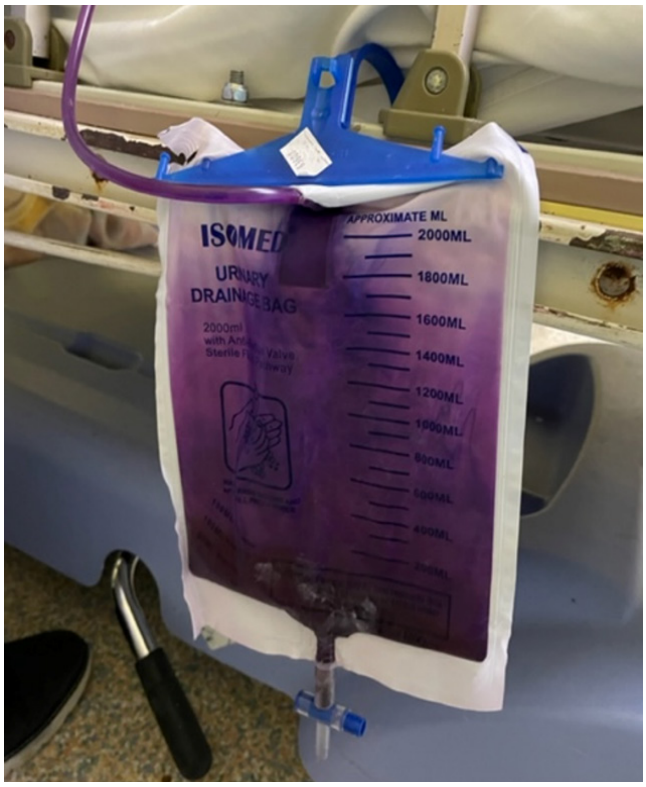


**Figure 1.** Shows purple discoloration of urine in the urine bag

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for three days. He denied any urinary symptom. Urinalysis revealed a pH of 9, leucocyte 3+ and erythrocyte 3+. Physical examination reviewed grade four sacral sore, measuring 5cmx5cm with necrotic edge with tendon exposed, while other physical examinations were unremarkable. His blood investigation reviewed hyperchloremic non anion gap metabolic acidosis (pH 7.252, bicarbonate 11mmol/L, base excess -16.9mmol/L, anion gap 8, delta ratio 0.3); leucocytosis (white cell count 23.5 x$10^9$/L); severe anaemia (haemoglobin 5g/dL) with worsening kidney function (urea 29.2 mmol/L, creatinine 334.6mmol/L). He was treated for severe anaemia, PUBS with urinary tract infection and infected sacral sore.His urinary catheter was changed, transfusion done and he was admitted for broad spectrum antibiotics infusion.

**Case-2**

An 84-year-old female with a history of hypertension, diabetes mellitus, and ischemic stroke had presented to the ED with atwo day history of shortness of breath, diarrhea, and vomiting, along with a striking purple urine in her urinary bag. She had been bedbound for the past two months due to recurrent ischemic strokes and required a Ryle's tube and urinary catheter, both changed biweekly. Upon examination, she appeared lethargic, tachypnoeic, with dry mucosa. Her blood pressure 76/61 mmHg, pulse rate 140 bpm with weak pulse volume, and oxygen saturation 88% on room air, which improved to 100% under a high-flow mask. She was afebrile with a random blood glucose of 9.2 mmol/L. Physical examination revealed generalized crepitations over the lungs, a soft but palpable bladder mass in the lower abdomen, and a grade 1 sacral sore, while the urinary bag contained purple-stained urine (Figure-2). During catheter exchange, foul-smelling pus was drained along with purple urine, leading to resolution of the bladder mass.

Blood investigations revealed leucocytosis (white cell count 16.5 x10^9/L), metabolic/lactic acidosis, and type 1 respiratory failure (pH 7.140, bicarbonate 13 mmol/L, base excess -13.9 mmol/L, PaO2 63 mmHg, PaCO2 20.3 mmHg, lactate 12.8 mmol/L), hyponatremia (sodium 123 mmol/L), and acute kidney injury (urea 9.4 mmol/L, creatinine 163 mmol/L). Urinalysis showed positive nitrites and leukocytes at 3+. An electrocardiogram revealed atrial fibrillation with a rapid ventricular response, and a chest X-ray showed consolidation in the right middle and lower zones.

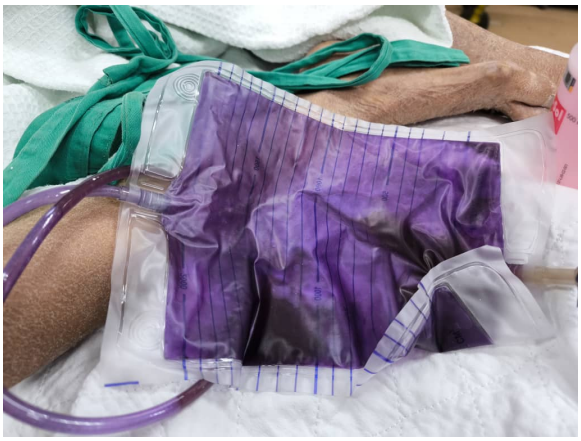

**Figure 2.** Shows purple discoloration of urine in the urine bag

The patient was diagnosed with PUBS associated with catheter-associated urinary tract infection and orthostatic pneumonia in septic shock. She was started on inotropic support after fluid boluses and broad-spectrum antibiotics.

## Discussion

PUBS is an uncommon condition, first reported in 1978, and it is the only known cause of purple coloured urine (1). PUBS usually involves female gender, constipation, alkaline urine, bed-ridden, and institutionalised patients with long-term urinary catheter (2-5).

A study done in 2005 revealed that the purple discoloration of urine originated not from the intraurethral portion of the catheter itself but rather from the urine bag and tubing connected to it (2). The purple discoloration is caused by a combination ofindigo, which contributes its blue colour and indirubin, which contributes its red colour. Indigo and Indirubin are both metabolites of the dietary protein, tryptophan. Tryptophan is initially metabolised in the gastrointestinal tract by gut bacteria into indole, which is then conjugated by liver into indoxyl sulphate. Normally, indoxyl sulphate is colourless and excreted in the urine, but in the presence of bacteria colonisers in the urinary catheter, that produce indoxyl sulphatase and phosphatase enzymes, indoxyl sulphate is converted to indoxyl and indirubin, resulting in the distinctive purple hue (2-7). Interestingly, this phenomenon is more likely to occur in alkaline urine (6).

Besides, PUBS is not only limited to urinary catheterisation. Recently, in January 2022, a case reported in India, where purple pleural fluid was drained after four days of chest tube insertion (8). This was the first reporting of PUBS outside urinary tract.

Despite its striking presentation, PUBS is usually benign and asymptomatic (7). PUBS are associated with urinary tract infection (UTI) caused by bacteria that produces sulphatase and phosphatase. The bacteria are mainly the Enterobacter species, like Escherichia coli, Klebsiella pneumoniae, Proteus, Enterococcus species, Pseudomonas aeruginosa, Providencia stuartii and so on (9). Urinalysis and cultures can be done to confirm UTI. All patients should have their urinary catheters replaced, along with appropriate antibiotics therapy. Asymptomatic patients should not be treated aggressively. Instead, control of urological sanitisation, decrease catheterisation duration, good catheter care and management of predisposing factor for PUBS like constipation should be the mainstay of treatment (2, 4, 5).

## Conclusion

PUBS is a reflection of UTI in catheterised patients, typically resulting from suboptimal catheter care and poor urogenital hygiene. To effectively manage PUBS, it is crucial to treat the UTI, replace the catheter and maintain good urogenital hygiene. Additionally, proper education on PUBS prevention should be given to both patients and relatives to minimize the risk of recurring episodes.

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Case Report

Journal of Emergency Medicine Case Reports

# Ocular Anticholinergic Toxicity from Datura arborea: A Reversible Mimic of Third Nerve Pals

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## Abstract

Unilateral fixed mydriasis is frequently associated with neurologic emergencies such as third cranial nerve palsy. However, rare toxicologic mimics-particularly those caused by topical exposure to anticholinergic alkaloids-may present similarly and lead to unnecessary neuroimaging. Datura arborea, an ornamental plant of the Solanaceae family, contains potent tropane alkaloids capable of inducing transient pharmacologic mydriasis via muscarinic receptorblockade. A 42-year-old previously healthy male developed sudden-onset blurred near vision and photophobia in the left eye shortly after accidental ocular contact with a flowering plant branch. Examination revealed left-sided dilated, nonreactive pupil with preserved extraocular movements, intact lid position, and impaired accommodation. Neurologic and fundoscopic examinations were unremarkable. A retrieved plant specimen was identified as Datura arborea by a consulting botanist. Pharmacologic testing with 1% pilocarpine produced no miosis, confirming muscarinic antagonism. The patient was managed conservatively with ocular irrigation, lubrication, and photoprotection. Symptoms resolved completely within 48 hours without recurrence. Topical exposure to Datura arborea can produce unilateral pharmacologic mydriasis that closely mimics third nerve palsy. Early recognition, targeted exposure history, and bedside pupillary testing can guide appropriate management and help avoid unnecessary neurologic evaluation.

**Keywords:** Anisocoria, anticholinergic, datura arborea

## Introduction

Acute-onset anisocoria, particularly when unilateral and accompanied by impaired pupillary reactivity, is frequently viewed as a potential indicator of life-threatening neurologic pathology, including third cranial nevre palsy, posterior communicating artery aneurysm, or transtentorial herniation (1). In emergency settings, such presentations often prompt urgent neuroimaging and specialist consultation. However, not all cases of fixed mydriasis are neurogenic in origin. Pharmacologic mydriasis, although benign and reversible, remains an under recognized diagnostic mimic that can result in unnecessary investigations and patient anxiety.

A range of pharmacologic agents and environmental exposures may lead to nonreactive pupil dilation. Among these, certain ornamental plants of the *Solanaceae* family-particularly *Datura arborea* (syn. *Brugmansia arborea*), commonly known as Angel's Trumpet-contain high concentrations of tropane alkaloids such as atropine, scopolamine, and hyoscyamine (2,3). These compounds are potent competitive antagonists of muscarinic receptors. When introduced topically into the eye, they block M3 receptors in the iris sphincter and ciliary muscle, resulting in unopposed sympathetic activity, loss of pupillary constriction, and accommodation paralysis (4,5). Clinically, this manifests as a fixed, dilated pupil with blurred nearvision-features that may closely resemble neuro-ophthalmologic emergencies.

Here in, we describe a case of unilateral pharmacologic mydriasis in an adult male following inadvertent ocular contact with *Datura arborea*. This case underscores the diagnostic value of detailed environmental history, the utility of bedside pharmacologic testing, and the importance of recognizing toxicologic mimics of neurologic syndromes.

## Case Report

A 42-year-old previously healthy male presented to the emergency department (ED) with sudden-onset blurred vision and photophobia in the left eye. The symptoms began approximately one hour after a low-hanging plant branchs truck the leftside of his face while he was playing soccer in a public park. He denied direct trauma to the globe,

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foreign body sensation, eyedrops or medications, diplopia, headache, or systemic complaints. Review of systems was negative for fever, dry mouth, confusion, urinary retention, or tachycardia.

On examination, the patient's vital signs were stable. The left pupil was dilated to approximately 7 mm, round, and unresponsive to both direct and consensual light (Figure-1). The right pupil measured 3 mm and was briskly reactive. Extraocular movements were full and symmetric bilaterally, and there was no ptosis. Visual acuity was 20/20 in the right eye and 20/80 for near vision in the left eye. The patient reported difficulty with near focusing, consistent with accommodation loss. Slit-lamp examination revealed clear corneas without epithelial staining, and deep, quiet anterior chambers. Mild bilateral conjunctival hyperemia was present. Intraocular pressures were 16 mmHg bilaterally. Fundoscopic evaluation showed sharp optic disc margins without signs of papilledema, hemorrhage, or retinal abnormalities. The remainder of the neurologic examination, including cranial nerve, motor, and cerebellar testing, was unremarkable.

While still in the ED, the patient's relatives returned with a fresh sample of the branch involved in the incident. The plant featured large, pendulous, trumpet-shaped yellow flowers. In consultation with a local plant taxonomist, the specimen was conclusively identified as *Datura arborea*-a toxic plant well-known for its anticholinergic properties.

Given the absence of neurologic signs and the suspicion of anticholinergic ocular exposure, a bedside pharmacologic test was performed. One percent pilocarpine was instilled in to the affected eye. After 30 minutes, the left pupil remained fully dilated and nonreactive, confirming the diagnosis of pharmacologic mydriasis due to muscarinic receptor blockade. Neurology and ophthalmology services were consulted, and both agreed that no further imaging or intervention was required.

Management consisted of ocular irrigation, preservative-free artificial tears, and photoprotection with sunglasses. The patient was observed for several hours and discharged without patient follow-up. At 48-hour telephone reassessment, the patient reported completere solution of symptoms, including restoration of near vision and normalization of pupillary size and reactivity.

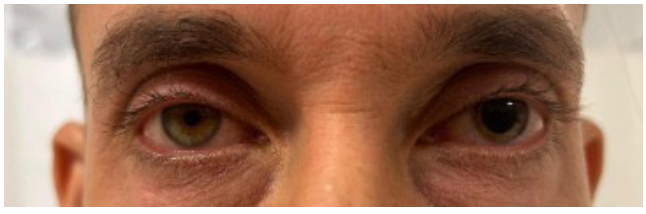

**Figure 1.** The left pupil was dilated to approximately 7 mm

## Discussion

This case highlights a benign but diagnostically challenging form of anisocoria resulting from direct ocular exposure to *Datura arborea*. The pathophysiologic mechanism is well-established: tropane alkaloids within the plant competitively antagonize muscarinic M3 receptors in the iris sphincter and ciliary muscle, leading to mydriasis and cycloplegia (4-6). The resulting fixed pupil and loss of near focus can mimic serious neuro-ophthalmologic disorders such as third nevre palsy.

Importantly, distinguishing pharmacologic mydriasis from neurologic causes is essential to avoid unnecessary diagnostic procedures. In this case, the absence of ptosis or ophthalmoplegia, preserved visual acuity, normal intraocular pressures, and a clear exposure history all pointed toward a toxicologic etiology. The pilocarpine test is a simple but powerful diagnostic tool: 1% pilocarpine causes nomiosis in pharmacologic blockade, where as denervated pupils (as seen in Adie syndrome or third nerve palsy) typically constrict due to supersensitivity (6).

The literature contains multiple case reports of similar presentations. Have lius and Asman described a series of seven adults with unilateral mydriasis following exposure to *Datura suaveolens*, all of whom recovered within 72 hours without specific therapy (7). Pediatric cases involving both *Datura* and *Brugmansia* species have also been reported, often in the context of outdoor play or unintentional plant contact (8,9). Direct instillation of plant sap in to the eye has occasionally been associated with corneal epithelial toxicity and transient tachycardia, although such complications are rare with simple surface exposure (10).

The current case reinforces the importance of detailed environmental history-taking in cases of isolated anisocoria. Early identification of the toxicologic cause permitted appropriate reassurance, avoided unnecessary neuroimaging, and ensured prompt symptomatic management. From a public health perspective, increased awareness of ornamental plant toxicity may help reduce similar presentations.

## Conclusion

Unilateral pharmacologic mydriasis resulting from *Datura arborea* exposure represents a reversible and benign condition that can closely mimic emergent neurologic disorders. Recognition of this clinical entity relies on careful history-taking, exclusion of neurologic deficits, and bedside pilocarpine testing. Awareness of such toxicologic mimics is essential for emergency physicians and ophthalmologist stop revent diagnostic delays, patient distress, and costly interventions.

**Conflict of interest:** None

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Case Report

Journal of Emergency Medicine Case Reports

# Small Round Blue Cell Sarcoma of the Left Humerus in a Pregnant Patient: Clinical Course of a Rare Aggressive Soft Tissue Tumor

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## Abstract

Small round blue cell tumors (SRBCT) represent a heterogeneous group of aggressive tumors with overlapping histological features, often requiring complex immunohistochemical and molecular studies for accurate diagnosis. Their occurrence during pregnancy poses significant diagnostic and therapeutic challenges, particularly when located in rare anatomical sites such as the humerus. We present the case of a 21-year-old pregnant woman at 31 weeks of gestation who was admitted with progressive pain and swelling in the left upper extremity. MRI revealed a large, intramedullary soft tissue mass in the mid-diaphysis of the left humerus with extension to adjacent musculature and neurovascular structures. Initial histopathological evaluation identified a poorly differentiated SRBCT. Despite extensive immunohistochemical work-up, a definitive diagnosis, necessitating advanced molecular analysis, remained elusive. The patient underwent cesarean delivery at 33 weeks due to obstetric indications. Subsequent PET-CT imaging demonstrated multiple hypermetabolic lesions consistent with metastatic disease. Chemotherapy with the VAC regimen was initiated following postpartum stabilization. This case underscores the complexity of diagnosing SRBCT during pregnancy and highlights the importance of multidisciplinary collaboration. Early recognition, thorough histopathological and molecular evaluation, and individualized oncologic and obstetric management are critical for optimizing maternal and fetal outcomes.

**Keywords:** Humerus, perinatal oncology, pregnancy, small round blue cell tumor, soft tissue sarcoma

## Introduction

Small Round Blue Cell Sarcoma, particularly Ewing's sarcoma, presents significant clinical management challenges in pregnant patients due to its rarity and the complexities of treatment during gestation. Diagnosis often occurs late, with nonspecific symptoms, as evidenced by patients presenting with fractures or localized masses (1, 2). Management typically involves a multidisciplinary approach, including preoperative chemotherapy and surgical intervention, with considerations for fetal safety (2-4). Prognostic factors indicate that maternal survival rates can be favorable, with one-year survival at 100% for bone sarcomas, although complications such as metastasis and local compression symptoms are common (4). Case studies highlight the necessity for individualized treatment plans, balancing maternal health and fetal outcomes, as seen in cases where cesarean sections were performed to mitigate risks(1). Overall, the prognosis remains variable, necessitating further research to optimize management strategies for this unique patient population (4).

## Case Report

A 21-year-old primigravida woman at 31 weeks of gestation presented with progressive pain and swelling in the left upper extremity. Her medical history was unremarkable, and there was no known family history of malignancy or genetic disorders. The pregnancy proceeded without complications until the emergence of musculoskeletal symptoms. Due to breech presentation and arrest of labor, a cesarean section was performed at 33 weeks of gestation. The patient reported no history of tobacco, alcohol, or substance use.

The initial complaint involved localized pain and swelling in the mid-portion of the left arm. Physical examination revealed soft tissue fullness, marked tenderness over the humerus, and restricted range of motion due to pain. No constitutional symptoms such as fever, weight loss, or fatigue were noted.

Magnetic resonance imaging demonstrated a destructive, intramedullary mass in the mid-diaphysis of the left humerus, measuring up to 85×50 mm. The lesion exhibited cortical disruption and extension into surrounding musculature and

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adjacent neurovascular structures (Figure-1). Tru-cut biopsy revealed features consistent with an undifferentiated small round blue cell sarcoma. Immunohistochemical analysis showed diffuse positivity for vimentin, actin, S100, synaptophysin, and a Ki-67 proliferation index of 35%. Markers including CD99, desmin, myogenin, and others were negative. A definitive diagnosis could not be established despite comprehensive immunophenotyping, and further molecular testing (FISH for CIC and EWSR1 rearrangements) was recommended.

Postpartum fluorodeoxyglucose (FDG) PET-CT imaging (Figure-2) revealed multiple hypermetabolic metastatic lesions in the lungs, thoracic and cervical vertebrae (including T10 and sacrum), and contralateral humerus. Following delivery, the patient was initiated on chemotherapy with a VAC regimen (vincristine, actinomycin D, cyclophosphamide).

As expected, the tumor exhibited high metabolic activity and locally invasive behavior. Unexpectedly, disseminated metastatic disease was present at the time of diagnosis, suggesting a more rapid progression possibly influenced by diagnostic delays associated with pregnancy. The prognosis is currently guarded, owing to the tumor's aggressive biological nature, undifferentiated histopathology, and the extent of metastatic involvement. The patient remains under multidisciplinary oncological care, with molecular analysis ongoing to refine therapeutic planning.

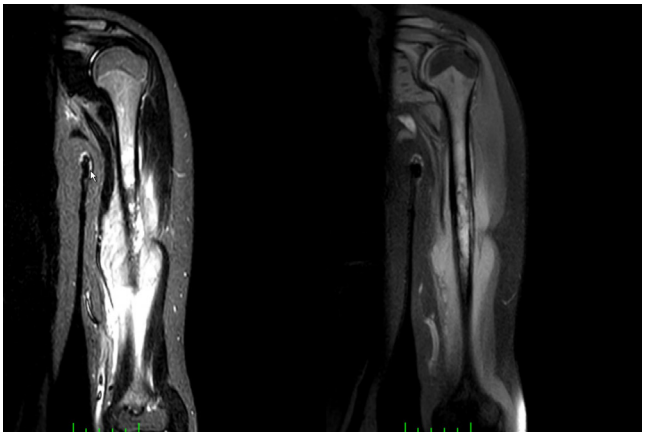

**Figure 1.** The lesion exhibited cortical disruption and extension into surrounding musculature and adjacent neurovascular structures

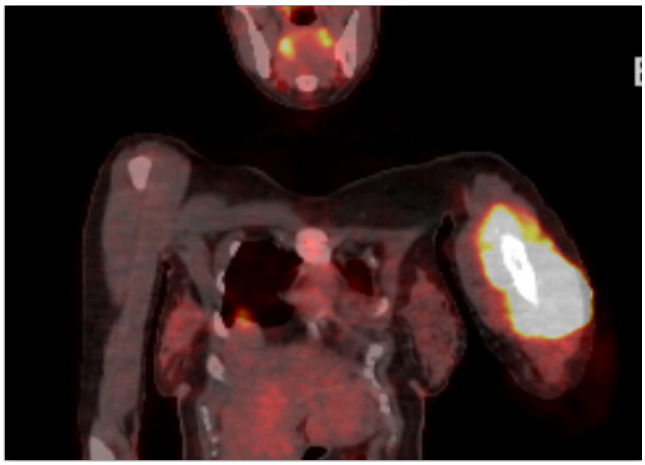

**Figure 2.** Postpartum fluorodeoxyglucose (FDG) PET-CT imaging

## Discussion

This case report presents a 21-year-old pregnant female patient who presented with pain in the left arm, specifically the shoulder region. Upon initial assessment, an MRI revealed a mass-like lesion located in the humeral midshaft, which was associated with an intramedullary extension. The lesion appeared to involve adjacent muscle structures, particularly the medial head of the triceps and the brachialis, with significant diffusion restriction. Further radiologic studies, including a CT and PET scan, confirmed the suspicion of metastasis, highlighting the presence of a hypermetabolic lesion in the left humerus, suggesting a primary tumor with metastatic spread (Figure 2). Additionally, the biopsy report raised concerns about a small blue round cell tumor, with possibly Ewing's sarcoma being a differential diagnosis.

The unique aspect of this case is the diagnosis in a pregnant patient, which is uncommon for soft tissue sarcomas like Ewing's sarcoma, particularly during the third trimester. The diagnostic process involved multiple imaging modalities, including MRI, CT, PET/CT, and histopathology, contributing to a multi-dimensional view of the lesion (Figure 1,2). Of particular note is the failure to achieve a definitive diagnosis from immunohistochemistry, with initial results pointing to the possibility of a neurogenic or myogenic sarcoma. However, including specialized methods such as FISH (Fluorescence in situ Hybridization) and additional genetic and molecular testing is crucial to reaching a more conclusive diagnosis (5). The pathology report indicated that the tumor exhibited aggressive local infiltration into surrounding muscular tissues, which could result in further complications if left untreated. This local infiltration, coupled with metastases seen in the lungs and vertebrae, presents a serious concern regarding disease progression and necessitates urgent management (6).

One of the significant challenges in this case was the complex interplay between the tumor's progression and the pregnancy. Ewing's sarcoma, while a relatively rare diagnosis in young adults, becomes particularly challenging to manage during pregnancy due to the limited options for chemotherapy and radiation therapy (7). Given the patient's status as 31 weeks pregnant, the oncological management needed to be balanced with the safety of both the mother and the fetus. The presence of a primary lesion with suspected metastasis further complicates this situation, as delaying chemotherapy could increase the risk of widespread disease progression, while immediate treatment might endanger the pregnancy.

The use of chemotherapy in pregnant women is controversial, particularly when the drugs used in traditional chemotherapy regimens (such as VAC, Vincristine, Dactinomycin, and Cyclophosphamide) are teratogenic in nature (8). In this case, chemotherapy was initiated with appropriate pre-treatment precautions, including anesthesia and careful monitoring, highlighting the importance

of multidisciplinary coordination between oncology, obstetrics, and anesthesiology teams. Another notable point is the recommendation for further molecular and cytogenetic testing, which is essential for confirming the diagnosis of Ewing's sarcoma and evaluating possible genetic mutations, such as EWSR1 gene translocations, which are characteristic of this tumor type (9). These genetic markers would help refine the diagnosis, determine prognosis, and guide treatment decisions (10). Moreover, such testing is imperative to distinguish between other small round blue cell tumors, such as neuroblastoma or lymphoma, which may have overlapping clinical and histopathological features.

Integrating molecular genetics into clinical practice is increasingly critical in treating cancers, especially in rare or unusual cases like this one. Given that conventional histopathology and immunohistochemistry could not definitively identify the tumor subtype, relying on genetic sequencing could provide the clarity needed to proceed with more targeted therapies. The patient's prognosis is heavily dependent on the stage of the disease at diagnosis, with metastatic involvement of the vertebrae and lungs raising concerns about a poor prognosis. Despite this, prompt initiation of chemotherapy, which is effective in treating Ewing's sarcoma, offers a chance for disease control. The patient's young age is a positive prognostic factor, as younger individuals tend to tolerate chemotherapy better and may experience more favorable responses. Furthermore, ongoing follow-up and monitoring through imaging and molecular markers will be necessary to assess the tumor's response to chemotherapy and to detect any potential recurrence or progression, particularly considering the presence of metastases. This case also brings forth ethical and psychological concerns regarding the management of a pregnant cancer patient. The psychological burden on the patient, who faces not only the threat to her own life but also the health of her unborn child, must be addressed. Offering appropriate counseling and psychological support is crucial in ensuring the patient's well-being and compliance with the proposed treatment regimen. In terms of ethics, the decision to proceed with chemotherapy, which poses a risk to fetal development, requires careful consideration. This decision-making process must involve the patient, her family, and a multidisciplinary team, with clear communication regarding the potential risks and benefits. The healthcare team must also ensure that the patient fully understands the implications of the treatment options available to her and the fetus.

## Conclusion

This case emphasizes the complexity of diagnosing and managing soft tissue sarcomas, particularly Ewing's sarcoma, in pregnant patients. The use of advanced imaging, genetic testing, and chemotherapy offers promising avenues for treatment, but careful coordination among healthcare providers and consideration of the ethical implications are crucial for optimizing outcomes. Further research into safer treatment protocols for pregnant patients with cancer is needed to improve the prognosis for both the mother and the fetus. This case also highlights the need for continuous advancements in molecular diagnostics to refine the classification and treatment of rare tumors like Ewing's sarcoma.

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Case Report

Journal of Emergency Medicine Case Reports

# The Result of a Strange Family Habit: Carbon Monoxide Poisoning

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## Abstract

Carbon monoxide poisoning usually occurs in closed environments through systems used for heating or hot water supply. Rarely, it can also result from the use of barbecues or hookahs in closed environments due to people's habits. In this case report, we aimed to present a case of carbon monoxide poisoning caused by burning a samovar inside the house. Three people from the same family applied to the emergency room with complaints of nausea, vomiting, headache, and dizziness. It was learned from history that the patients lit a samovar for tea in a closed environment and slept in the same environment. The patients' COHb levels in the emergency room were 15.9, 13.6, and 10.1, respectively. The patients were given high-flow normobaric oxygen therapy (13 lt/min). The patients were discharged without any problems in their follow-ups. Carbon monoxide poisoning can also occur, albeit rarely, through fuel sources such as hookah or samovar in closed spaces. Preventing such cases requires effective public health information strategies.

**Keywords:** Carboxyhemoglobin, CO poisoning, emergency medicine, samovar

## Introduction

Carbonmonoxide (CO) poisoning is caused by inadequate combustion of carbon-containing substances. Since CO is a colourless, odourless and tastelessgas, it often goes unnoticed until symptoms appear (1). The affinity of CO gastohaemoglobin (Hb) is 250 times higher than oxygen. During poisoning, it binds to Hband forms carboxyhaemoglobin (COHb) and hypoxaemic hypoxia occurs (2). As a result, many symptoms and signs may occur in patients, including nausea, vomiting, headache, dizziness, syncope, arrhythmia and death (3).

CO poisoning is frequently seen as a result of inhalation of exhaust gases and industrial waste gases in the United States of America, while in our country it is frequently caused by exposure to waste gases of heating and hot water supply systems (4,5). Water heater, combi boiler, solid fuel and natural gas stoves are the most common causes of CO poisoning in our country (6). In addition, CO poisoning can also be seen as a result of exposure to exhaust or hookah smoke indoors. In this case report, we aimed to present CO poisoning in a family who burnt samovar indoors.

## Case Reports

Three patients (50-year-old male, 47-year-old female and 15-year-old female) were admitted to the emergency department with complaints of nausea, vomiting, headache and dizziness. It was learnt from the history of these patients that they lit a samovar for tea in a closed environment. After drinking tea, they slept in the same environment and in the morning, the 47-year-old female patient wokeup with severe headache, nausea and vomiting and presented to the hospital. Since the other two patients also had symptoms and based on the history, investigations were requested with a prediagnosis of CO poisoning. The general condition of all three patients were moderate, conscious, orientated and co-operative. Vital signs were within normal limits. Systemic examination was unremarkable. Cranial CT imaging was not performed because the neurologic examination was unremarkable and the patients responded to symptomatic treatment. The COHb levels of the patients at the emergency department were 15.9, 13.6 and 10.1, respectively. Other laboratory and clinical characteristics are given in Table-1. High-flow normobaric oxygen therapy (13 lt/min) was given

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**Table 1:** Clinical and laboratory characteristics of patients

| Variables | Patient 1 | Patient 2 | Patient 3 |
|---|---|---|---|
| Years, years | 50 | 47 | 15 |
| Sex | Male | Female | Female |
| Arrival complaint | Nausea, dizziness | Headache, Nausea, Vomiting | Headache, Nausea |
| SBP/DBP, (mm/Hg) | 135/87 | 121/82 | 112/72 |
| Pulse, beat / min | 84 | 64 | 71 |
| Respiratory rate, /min | 13 | 14 | 14 |
| Fever, $^0$C | 36.7 | 36.3 | 36.5 |
| Arrival | | | |
| COHb level, % | 15.9 | 13.6 | 10.1 |
| Lactate, mmol/L | 0.9 | 1.9 | 1 |
| Creatine, mg / dL | 0.79 | 0.6 | 0.66 |
| Troponin, ng / L | 2.1 | 3.7 | 1.4 |
| ALT, U / L | 12 | 53 | 11 |
| AST, U / L | 20 | 47 | 20 |
| Glucose, mg / dL | 146 | 111 | 95 |
| LastCOHblevel, % | 4.3 | 3.2 | 1.4 |

SBP/DBP: Systolic blood pressure/Diastolic blood pressure, COHb: Carboxyhemoglobin, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase.

to the patients. In addition, hydration and symptomatic treatment are given. The patients were discharged from the emergency intensive care unit because they had no complaints and COHb levels were within normal limits.

## Discussion

CO poisoning is an important cause of mortality and morbidity especially in developing countries. In an autopsy study conducted in Turkey, CO poisoning was found to be the most common cause of death due to poisoning (7). Undoubtedly, there are many studies on CO poisoning in the literature. However, what distinguishes our case from other studies in the literature is the CO poisoning caused by burning a samovar with wood in a closed environment. Samovar is a material used for boiling water in wood fire tobrew tea. Since it releases a lot of smoke and CO gas into the environment, it should be used outdoors. In the province where the case was observed, samovar tea is popular and consumed a lote specially in summer months. However, as in our case, family members burned the samovar in a closed environment instead of an open environment and slept at night without ventilating the environment well. As a result, they were exposed to the dense smoke in the environment and had CO poisoning.

Most CO poisoning occurs in households. CO poisoning occurs as a result of old, poorly maintained or poorly maintained systems used in homes or improper installation (8,9). In order to prevent these poisonings and prevent unwanted deaths, preventive practices should be implemented and public awareness should be raised (10). As a result of raising public awareness and consciousness, incidents such as the one in our case report will not occur.

One of the family members, the mother, wokeup because she had a severe headache, nausea and vomiting and wokeup the other family members. Thus, a family tragedy was avoided. In CO poisoning, various clinical findings ocur depending on the COHb level and the severity of these findings varies according to the level of CO to which the patient was exposed and the duration of exposure (2). It may present with mild symptoms such as fatigue, nausea and vomiting, or serious neurological and cardiovascular findings or death may ocur (11). CO poisoning is also called the silent killer because it causes death without any symptoms and mistand usually in sleep (12). What saves the lives of the patients are the nonspecific symptoms related to intoxication and these symptoms usually wake the patients up from sleep. In our case, it was these nonspecific symptoms of the mother that saved the lives of the family members.

## Conclusion

In many countries, including ours, CO poisoning continues to be a major public health problem that can arise from a variety of sources and can have potentially fatal consequences. While the majority of cases are associated with common sources such as faulty heating systems or poor ventilation, lessfrequent but notable examples have been reported due to the use of hookahs or samovars in closed spaces. Given the preventable nature of such incidents, it is essential to implement comprehensive public education campaigns aimed at raising awareness of the risks of CO poisoning, especially in traditional or cultural practices involving indoor fuel burning.

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Case Report

Journal of Emergency Medicine Case Reports

# An Unusual Cause of Hypertension: Bilateral Accessory Renal Arteries

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## Abstract

Hypertension in childhood is a significant risk factor for the subsequent development of vascular diseases in later life. It is recommended that all children with hypertension undergo investigation for potential secondary causes. The bilateral accessory renal artery represents a rare etiological factor in the development of renovascular hypertension. We presented a hypertensive adolescent patient who was admitted to the pediatric emergency department with complaints of dizziness and dyspnea and was diagnosed with bilateral accessory renal arteries, which was a rare condition. In renal color Doppler ultrasonography, measurements of both renal arteries at the renal hilum and interlobar levels were identified as significant for diagnosing stenosis. CT angiography revealed right renal and additional accessory renal arteries. A multidisciplinary approach and individualized patient management are critical for the diagnosis and treatment of renal vascular diseases. In this case, a rare variation, bilateral accessory renal artery, was diagnosed during the investigation of hypertension etiology. It is recommended that renal CT imaging is utilized for the precise diagnosis of renal artery abnormalities in hypertensive children.

**Keywords:** Accessory renal artery, childhood, secondary hypertension

## Introduction

Secondary hypertension is more prevalent in children than in adults, with a reported prevalence of 1–3%. The most frequent cause is underlying renal disease. Renovascular hypertension (RVH) refers to elevated blood pressure secondary to renal artery stenosis (1). In contrast, renal vascular anomalies such as accessory renal arteries are rarely encountered. These anomalies are usually asymptomatic and are often detected incidentally during imaging (2).

## Case Report

A 13-year-old boy presented to the pediatric emergency department with complaints of dizziness and shortness of breath. On physical examination, sitting blood pressure measurements were 140/90 mmHg in the right arm and 130/90 mmHg in the left arm. His body weight was 81 kg (above the 97th percentile, +3.03 SD), height 168 cm (93rd percentile, +1.47 SD), and body mass index (BMI) was 28.7 kg/m² (above the 95th percentile, +2.26 SD). Other systemic findings on physical examination were unremarkable. The patient's personal medical history was unremarkable. However, a positive family history of hypertension was noted in the father and paternal grandmother. Complete blood count, serum and urine biochemistry, urine culture, and arterial blood gas analyses were within normal limits. Plasma renin activity was 18.4 uIU/mL (reference range: 5.3-99.1), while aldosterone level was elevated at 53.8 ng/dL. Both electrocardiogram and transthoracic echocardiography revealed normal findings.Renal Doppler ultrasonography revealed increased peak systolic and end-diastolic velocities, with resistive index values ranging from 0.46 to 0.49 in both renal arteries at the hilum and interlobar levels-findings that were suggestive of stenosis. However, captopril-enhanced renal scintigraphy showed no evidence of renovascular disease in either kidney.Renal computed tomographic (CT) angiography was performed due to persistently elevated blood pressure values above the 95th percentile during follow-up after initiation of enalapril. CT angiography revealed the presence of the right renal artery and two accessory renal arteries supplying the upper and lower poles of the right kidney, respectively. On the left side, both the main renal artery and an accessory renal artery were seen to originate separately from the abdominal aorta (Figure 1, 2). These vessels converged at the renal hilum. The accessory renal artery on the left had a smaller diameter, but there was no evidence of stenosis. During follow-up, the patient was

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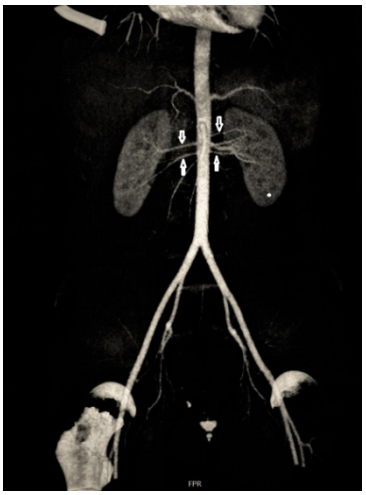

**Figure 1.** Three-dimensional computed tomographic angiography showing bilateral renal and accessory renal arteries. Two accessory arteries are observed on the right, supplying the upper and lower poles of the kidney. On the left, an accessory renal artery originates separately from the abdominal aorta and joins the main renal artery at the hilum. Arrows indicate the accessory vessels.

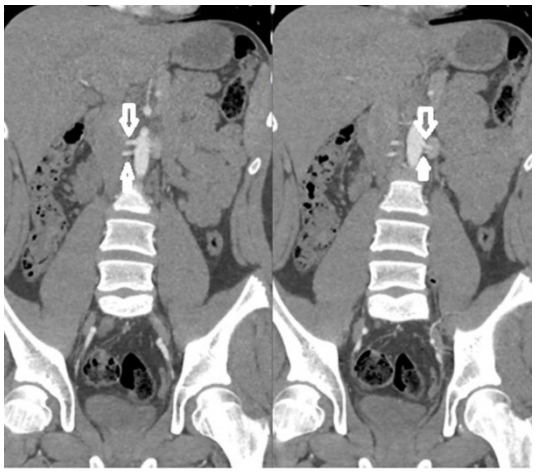

**Figure 2.** Coronal CT angiography images demonstrating the origins of the main and accessory renal arteries from the abdominal aorta. Both kidneys show separate vascular supplies, with accessory arteries visible on each side. Arrows indicate the vascular origins.

advised to follow lifestyle modifications, including dietary salt restriction and increased physicalactivity. However, blood pressure remained elevated despite these non-pharmacological interventions. Amlodipine was initiated at a dose of 0.05 mg/kg/day and gradually increased to the maximum tolerated dose. As hypertension persisted and echocardiography revealed left ventricular hypertrophy along with bilateral hypertensive retinopathy, enalapril was added to the treatment regimen. Subsequently, blood pressure progressively decreased and was maintained below the 90th percentile for age and height during out patient follow-up.

## Discussion

Hypertension represents a significant public health issue in children, paralleling its impact in adults. It is therefore imperative that hypertension in childhood is diagnosed and treated as early as possible, given the potential for complications to arise in adulthood. In children, blood pressure measurement is recommended at each examination from the age of three onwards. Furthermore, in the presence of associated risk factors, routine blood pressure measurement is recommended prior to the age of three (3).

Prior research has demonstrated that the presence of a unilateral accessory renal artery is observed in 20–36.1% of hypertensive adults. Nevertheless, a study reported a prevalence of 2-14.7% for the bilateral accessory renal artery. It is established that the presence of an accessory renal artery does not influence the diameter of the main renal artery (4). Similarly, the diameter of the main renal arteries was maintained in our patient.

In cases of renal artery stenosis, hypertension results from the disruption of blood flow in the renal artery or its associated branches. In the context of an ischemic kidney, the production of excess renin leads to an abnormal release of aldosterone. Hypertension is linked to the stimulation of the renin-angiotensin system and inadequate perfusion resulting from sodium and volume retention. This is the primary cause of elevated blood pressure in renovascular hypertension. Several studies have suggested that accessory renal arteries may contribute to hypertension due to increased renal vascular resistance or abnormal flow patterns. In some cases, these vessels are associated with segmental ischemia that may stimulate renin secretion and activate the renin-angiotensin-aldosterone system (RAAS) even in the absence of overt stenosis. Although the exact mechanism remains debated, accessory renal arteries should be considered potential contributors to pediatric hypertension when no other cause is identified (4,5).

The use of captopril renal scintigraphy is an effective diagnostic tool for the identification of RVH. A normal result does not preclude the possibility of RVH, and an abnormal result may be attributable to parenchymal lesions. In this case, the results of the captopril renal scintigraphy did not corroborate the diagnosis.

Computed tomography angiography provides a non-invasive method of imaging the anatomical structure and defining pathological findings in renal pathologies. Consequently, a diagnosis of bilateral accessory renal artery was made on the basis of CT angiography in our patient.

Renal artery variations are classified into two principal groups: early division and extra renal arteries (ERA).

Segmental branching of the main renal arteries from proximal of renal hilum level is called as early division. ERA is divided into two groups as hilar (accessory) and polar (aberrant) arteries. While the hilar arteries enter the kidneys from the hilum with the main renal artery, polar arteries enter the kidneys directly from the capsule outside the hilum (6).

Occlusive diseases of the renal arteries are characterized by poor prognosis in children. Endovascular techniques, surgery and medical approaches are used for the treatments of the renovascular diseases. Success rate is reported as 79% with these methods (6). A multidisciplinary approach involving nephrology, interventional radiology, and surgery, as well as individualized patient management, is critical for accurate diagnosis and effective treatment.

In addition to renovascular anomalies, the role of obesity should also be considered in the pathogenesis of hypertension. The patient in this case had a body mass index above the 95th percentile, meeting the criteria for obesity. Obesity is known to contribute to elevated blood pressure through mechanisms such as increased sympathetic nervous system activity, sodium retention, and vascular dysfunction. Therefore, it is possible that both obesity and bilateral accessory renal arteries acted synergistically in the development of hypertension in this patient (7, 8).

## Conclusion

In our case, bilateral accessory renal artery was found while investigating the causes of hypertension as a rare variation. Although unilateral accessory renal artery has been reported in the literature, the bilateral accessory renal artery is rarely seen in children. Therefore, we suggest that renal CT angiography be considered for the accurate diagnosis of renal artery abnormalities in hypertensive children.

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Case Report

Journal of Emergency Medicine Case Reports

# From a Traditional Treatment to a Major Amputation: A Zootherapy-Induced Necrotizing Soft Tissue Infection

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## Abstract

Necrotizing soft tissue infections (NSTIs) are rapidly progressive and often life-threatening clinical conditions. If not diagnosed early, they can lead to serious outcomes such as sepsis, vascular complications, and limb loss. This case presents a necrotizing soft tissue infection (NSTI) that is rarely reported in the literature in Turkey; it developed after a zootherapy intervention, was complicated by arterial occlusion, and resulted in an above-knee major amputation. A 90-year-old male patient did not adhere to the recommended medical treatment after an ankle sprain and instead applied raw sheep lung to the injured area for six days. Following this traditional zootherapy attempt, he returned with widespread erythema, edema, foul-smelling discharge, and signs of systemic inflammation. His Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score was calculated as 8, and he was managed under the presumptive diagnosis of NSTI. In addition to antibiotic therapy, wound care was administered; however, on the eighth day, an acute arterial occlusion developed, and the patient was referred to a tertiary care center. Despite all treatments, no clinical improvement was achieved and an above-knee amputation was performed. Traditional practices such as zootherapy can cause rapidly progressing infections and irreversible clinical outcomes, especially in elderly individuals with comorbid conditions. In this context, increasing healthcare professionals' sensitivity to traditional treatment practices and raising public awareness about the potential risks of these interventions are of great importance for early intervention and the prevention of complications.

**Keywords:** Amputation, necrotizing soft tissue infection, traditional treatment, zootherapy

## Introduction

In communities where health literacy is low or access to healthcare services is limited, non-evidence-based traditional treatment methods are frequently used (1). Many of these interventions, applied after traumatic injuries and lacking sterile conditions, can lead to serious complications such as soft tissue infections and tissue necrosis (2).

Necrotizing soft tissue infections (NSTIs) are severe infections that spread rapidly through the skin, subcutaneous tissue, fascia and, in some cases, muscle, and despite their rarity, they carry high mortality rates. The disease often begins with nonspecific clinical findings such as fever, localized pain, and erythema; this can delay diagnosis and result in delayed treatment. NSTIs are associated with serious complications such as rapid clinical deterioration, sepsis, multiorgan failure, and limb loss. Therefore, early diagnosis, prompt initiation of broad-spectrum antibiotic therapy, and urgent surgical debridement are crucial for improving prognosis (3).

Zootherapy is a traditional practice defined as the direct application of animal organs, tissues, or secretions to the body for therapeutic purposes. Although these methods have been used for centuries in some regions, their lack of scientific validity and failure to adhere to sterilization practices can set the stage for serious infections(4). Practices such as applying animal organs directly to injured tissue are rarely reported in the literature, but such interventions can increase the risk of secondary bacterial contamination in affected tissues (5). Particularly in elderly individuals with comorbid diseases, these infections may follow a refractory course and can lead to severe outcomes such as sepsis, vascular complications, or even amputation (6). The potential for non-sterile traditional practices to cause serious infections has also been demonstrated by cases such as necrotizing fasciitis after wet cupping therapy (7)and soft tissue infections following catgut acupuncture using animal-derived threads (8).

In this case, we present a rare and severe clinical scenario of widespread cellulitis, soft tissue necrosis, arterial occlusion, and ultimately a major amputation that developed following

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the application of non-sterile sheep lung tissue after an ankle injury. The case highlights the devastating complications that zootherapy, a practice outside of modern medicine, can cause, especially in elderly individuals with comorbid conditions. To our knowledge, this case is one of the few reported in Turkey of a necrotizing soft tissue infection arising from an animal tissue–based zootherapy practice, and it provides a unique contribution to the literature.

## Case Report

A 90-year-old male patient presented to the emergency department after an ankle sprain. Physical examination and radiological evaluation revealed no bone pathology (Figure-1). No open wound was observed at the trauma site, but a minimal superficial abrasion was present on the skin. A plaster splint was applied, and elevation and cold compresses were recommended. However, after discharge, the patient did not follow medical advice: he removed the splint and wrapped raw sheep lung directly around the injured area, continuing this practice for six days. This traditional remedy, believed to relieve pain and swelling, falls under the concept of zootherapy, wherein animal tissues are used for healing.

Six days later, the patient presented again to the emergency department with widespread erythema, edema, increased local warmth, foul-smelling discharge, and signs of systemic inflammation in the left foot and lower leg (Figure-2,3). His medical history included hypertension, type 1 diabetes mellitus, chronic obstructive pulmonary disease (COPD), and coronary artery disease (CAD). Laboratory findings were as follows: C-reactive protein (CRP) 180.4 mg/L, white blood cell count (WBC) 19.65×10³/µL, glucose 244 mg/dL, creatinine 1.07 mg/dL, sodium 132 mmol/L, and hemoglobin 14.5 g/dL. Based on these results, the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score was calculated to be 8. The combination of a widespread inflammatory response following raw animal tissue contact, a history of diabetes, rapidly worsening local infection signs, and a high LRINEC score pointed to a strong possibility of NSTI. In light of the clinical and laboratory findings, along with the history of zootherapy, management was initiated under the presumptive diagnosis of NSTI.

CT angiography of the lower extremity was performed to evaluate potential vascular complications. Imaging revealed diffuse atherosclerotic plaques at the level of the terminal aorta and left iliac artery, approximately 50–60% stenosis in the left iliac arterial system, and significant subcutaneous edema in the left lower leg and foot. Consultations were obtained from the infectious diseases and cardiovascular surgery departments. The cardiovascular surgery team did not recommend any interventional procedure and opted for medical management. The patient was admitted to the infectious diseases department for further treatment.

During hospitalization, only blood cultures were obtained; no samples were taken from the wound site, a significant shortcoming in the management. The lack of wound culture prevented definitive identification of the causative microorganism and limited targeted therapy. The blood culture grew coagulase-negative staphylococci, which was interpreted as contamination from skin flora. Empiric broad-spectrum antibiotic therapy with meropenem, tigecycline, and amikacin was initiated. Simultaneously, wound care was performed by the plastic surgery team.

On the eighth day of hospitalization, physical examination revealed coolness, pallor, and loss of peripheral pulses in the left foot. A repeat CT angiography showed maintained flow in the posterior tibial artery but no flow in the anterior tibial and dorsalis pedis arteries. The findings were consistent with an acute arterial occlusion (Figure-4). The patient was transferred to a higher-level center for advanced wound care and possible revascularization procedures.

At the referral center, despite all medical and interventional treatments, no clinical improvement was achieved. With progressive ischemia and tissue necrosis, an above-knee amputation of the left lower extremity was performed. However, because the amputation was performed at another institution, the microbiological or histopathological examination results of the removed tissues were not accessible to us. Therefore, the histological nature of necrosis could not be determined.

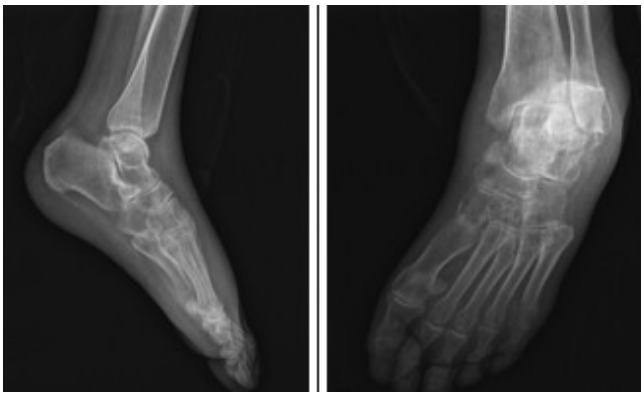

**Figure 1.** Radiological image with no bone pathology

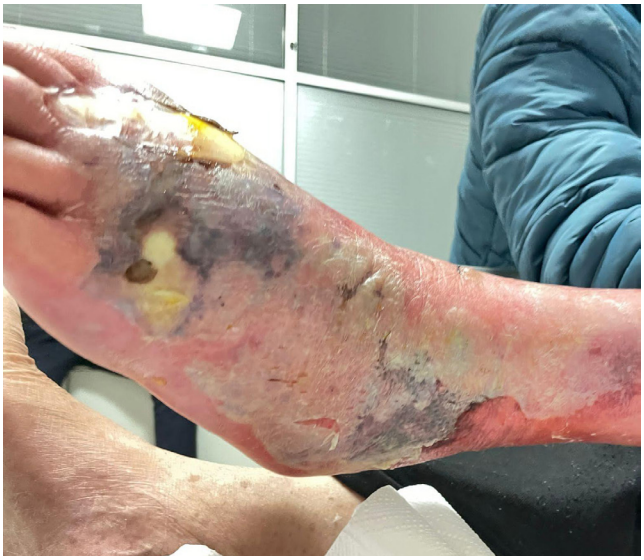

**Figure 2.** Plantar view of the affected foot showing extensive erythema, necrotic changes, and purulent foci

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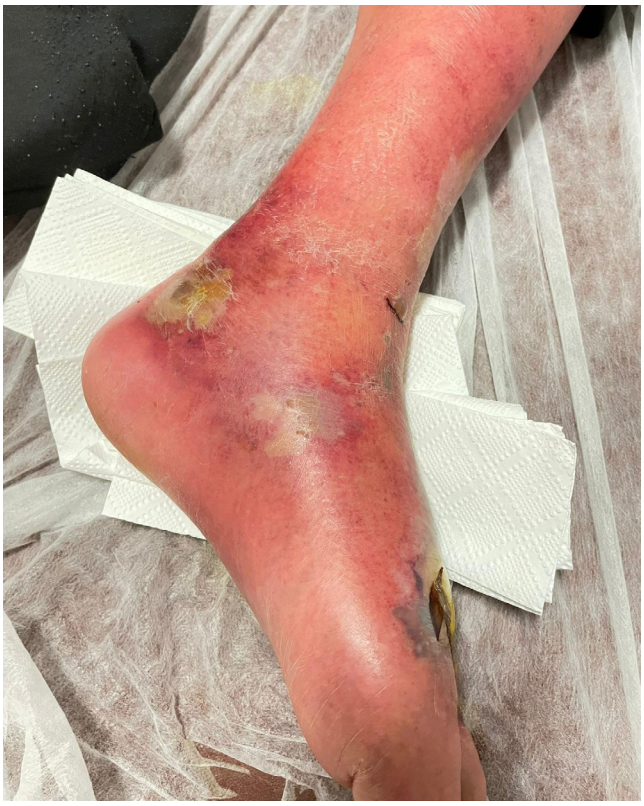

**Figure 3.** Lateral view of the same foot demonstrating diffuse edema, ecchymosis, and ulcerated lesions

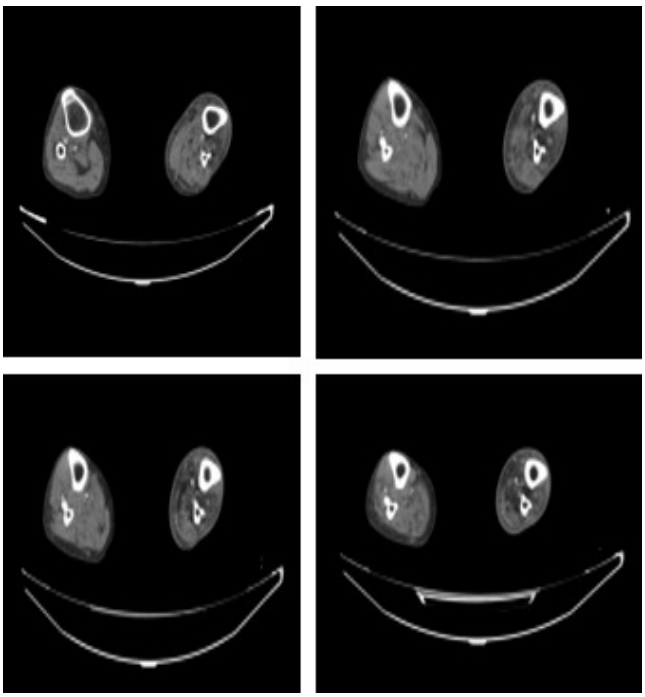

**Figure 4.** CT angiography with acute arterial occlusion

## Discussion

Traditional practices like zootherapy, involving the use of animal products for treatment, persist especially in rural communities. However, because such practices lack sterile conditions, they can predispose individuals to infectious complications. According to the World Health Organization's report on traditional and complementary medicine, the uncontrolled use of animal tissue–based interventions can lead to severe allergic reactions, toxicity, and life-threatening infections (1). This case illustrates a rare clinical scenario characterized by a rapidly progressive NSTI that developed after the traditional application of raw sheep lung.

Although the skin integrity appeared to be intact after the trauma, the presence of minimal dermabrasion and direct contact with raw animal tissue created a suitable biological environment for a severe infection to develop. The literature has reported that such traditional practices can result in complications like granulomas, cellulitis, abscesses, and, albeit rarely, necrotizing fasciitis (7,8). Furthermore, comorbid conditions such as diabetes, advanced age, and peripheral vascular disease are defined risk factors for the development of NSTI (2,9). The LRINEC score is a laboratory scoring system widely used in the diagnosis of NSTI, comprising six parameters (C-reactive protein, leukocyte count, hemoglobin, sodium, creatinine, and glucose levels). A score of ≥6 indicates significant risk, and a score ≥8 indicates a high-risk level (10). In this patient, the presence of these predisposing factors, along with a high LRINEC score of 8, strongly supported the likelihood of NSTI.

Although the CT angiography did not detect typical radiological signs of necrotizing fasciitis, such as gas accumulation or fluid collections along the fascial planes, this is not sufficient by itself to rule out NSTI. The literature indicates that imaging modalities play an adjunct role in NSTI diagnosis; however, particularly in the early stage, these findings may not always be detectable (11,12). Therefore, in cases suspected of NSTI, clinical evaluation, the presence of comorbidities, and laboratory-based scoring systems like LRINEC are prioritized as key determinants in the diagnostic process. Indeed, in the present case, despite inconclusive radiological findings, the patient's clinical course, associated risk factors, and high LRINEC score prompted treatment under the presumptive diagnosis of NSTI, and the subsequent severe vascular complications and invasive nature of the infection validated this approach.

The fact that only blood culture was obtained in this case can be considered a significant limitation. Not performing deep tissue sampling or obtaining a wound culture hindered precise identification of the pathogen and made it more difficult to guide antibiotic selection. Current literature shows that in patients with suspected NSTI, obtaining a wound culture early in the course both increases diagnostic clarity and allows for targeted therapy (13,14). Despite this deficiency, the empiric broad-spectrum antibiotic regimen initiated in our patient appears to have been appropriate and in line with combinations recommended in the literature (2,15).

The arterial occlusion observed in our patient is one of the serious vascular complications that can develop during NSTI. This occlusion typically arises from the inflammatory reaction in infected tissue, endothelial damage, and local edema compressing the arterial lumen. Although the main mechanism of vascular occlusion in NSTI is inflammation and pressure from surrounding tissues, on rare occasions septic thrombophlebitis

or direct microbial invasion of the arterial wall can occur. On CT angiography, arterial narrowing or occlusion is usually associated with local vascular edema and perivascular inflammation. This situation impairs distal tissue perfusion, contributing to ischemia and necrosis (2,12,16). In our patient, the inflammation and local edema caused by the infection compressed the arterial lumen, leading to an arterial occlusion. This occlusion compromised distal perfusion and led to further progression of ischemia and necrosis. The ensuing vascular insufficiency and spread of infection rendered conservative treatment and revascularization attempts ineffective; consequently, an above-knee amputation was required. This sequence of events demonstrates that vascular complications in NSTI play a critical role in disease progression and treatment success.

This case draws attention to the severe outcomes that can result from traditional treatment methods like zootherapy. Diagnosing serious infections such as NSTI can be difficult. Especially in regions where traditional practices are prevalent, an incomplete or misleading patient history can delay the diagnostic process. This necessitates enhancing healthcare workers' awareness of traditional treatment practices and improving their traditional sensitivity. Additionally, community education and public health initiatives regarding the risks of these practices are vitally important for the prevention of infections and timely intervention (17). An approach in emergency care that is traditionally sensitive and aware of traditional practices will expedite diagnosis and improve the quality of patient care.

## Conclusion

Traditional practices lacking a scientific basis can lead to severe infections and irreversible complications, especially in older individuals with underlying chronic diseases. As seen in the presented case, a necrotizing soft tissue infection that developed after contact with raw animal tissue was accompanied by serious vascular complications such as arterial occlusion and resulted in a major amputation. This underscores the need to increase clinicians' awareness of traditional treatment practices and the importance of early diagnosis and rapid intervention in similar cases. Moreover, the public should be informed about the risks of such practices and preventive strategies should be developed from a public health perspective.

**Acknowledgment**
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Case Report

Journal of Emergency Medicine Case Reports

# A Case of Methyl Alcohol Intoxication Developing Through Skin Contact

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## Abstract

Methanol is a toxic alcohol that can lead to high-anion gap metabolic acidosis, visual impairment, and CNS depression. While ingestion is the most common route, toxicity may also occur via dermal absorption, especially with prolonged exposure.

A 71-year-old male with diabetes and coronary artery disease presented with sudden bilateral vision loss after repeated dermal application of a methanol-containing solution for joint pain. On arrival, he was disoriented and completely blind. Arterial blood gas showed severe metabolic acidosis (pH 6.9) with elevated anion gap and lactate. Fundoscopic examination revealed bilateral optic disc edema. Due to the unavailability of fomepizole and serum methanol measurement, intravenous ethanol was administered, and hemodialysis was initiated. Despite correction of the metabolic acidosis, the patient developed irreversible cortical blindness. This case highlights dermal methanol absorption as a rare but serious cause of systemic toxicity. Emergency physicians should consider methanol poisoning in patients with visual symptoms and unexplained metabolic acidosis, even in the absence of ingestion history. Early diagnosis and timely treatment are critical to prevent permanent complications.

**Keywords:** Intensive care unit, methanol poisoning, skin exposure, toxicology

## Introduction

Most methanol poisonings in the world occur after the ingestion of methanol-contaminated products, such as ethanol-based beverages, which may be contaminated with methanol. This situation is thought to occur due to alcohol-dependent individuals gaining access to methanol more cheaply, or due to intentional ingestion for suicidal purposes. Cases of toxicity from inhalation or skin exposure are rarely reported (1).

Methanol'soral absorption of reachesit speak within 5-10 minutes on an empty stomach, and 30-60 minutes after consumption on a full stomach. When 200 ppm of methanol is inhaled for 4 hours, it is well absorbed through inhalation, with an average half-life of 0.80 hours (2). Accordingto Association Advancing Occupational and Environmental Health, USA (ACGIH) data, the maximum concentration allowed for skin contact is 200 ppm (270 mg/m$^3$) (3).

Methanol's metabolism primarily occurs in the liver via alcohol dehydrogenase, where it is converted to formaldehyde, then to formic acid, and ultimately to carbon dioxide and water. This pathway is folic acid-dependent. Ethanol and fomepizole inhibit the effect of alcohol dehydrogenase, reducing the formation of toxic metabolites (4).

The accumulation of unmetabolized formic acid results in lactic acidosis with an increased anion gap, specifically targeting the optic nerve's optic disc and retro-laminar section, leading to optic disc oedema, myelin sheath disruption, and optic nerve lesions (5). Imaging studies using magnetic resonance imaging (MRI) and computed tomography (CT), as well as autopsy findings, have revealed oedema and necrotic damage in the basal ganglia, particularly in the putamen, as well as hemorrhages in the subcortical white matter (6).

Methanol typically causes nausea, vomiting, and abdominal pain. In mild to moderate methanol poisoning, common symptoms include headache, vertigo, drowsiness, and confusion. Methanol has much less of a euphoric effect than ethanol. Ophthalmological symptoms range from blurred vision and altered visual fields to complete blindness.

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Blurred vision, decreased visual sharpness, photophobia, and the sensation of being in a "foggy area" are common complaints in more than half of methanol toxicity cases (5).

Elevated serum methanol levels and severe metabolic acidosis with an increased anion gap strongly suggest methanol or ethylene glycol poisoning. The most common radiographic finding after severe methanol poisoning is bilateral necrosis of the putamen. However, brain CT scans performed within the first 24 hours after methanol ingestion are typically normal (7). In clinical examination, despite blindness, the first MRI scans may not show optic abnormalities, but case reports have indicated that repeated MRI scans after one month of methanol poisoning revealed atrophy in the optic chiasm and pre-chiasmatic optic nerves (8).

Methanol and ethylene glycol poisonings cause scores of fatal intoxications annually, and even relatively small ingestions of these alcohols can produce significant toxicity. Rapid recognition and early treatment, including alcohol dehydrogenase (ADH) inhibition and often hemodialysis, are crucial.

This case report it is aimed to emphasize the toxicity of methyl alcohol, which can develop with long-term skin exposure and the management and consequences of complications experienced in a 70-year-old male patient who experienced sudden vision loss.

## Case Report

A 71-year-old male patient presents to the emergency room with complaints of sudden onset of blurry vision followed by progressive complete visual impairment.It was learned that the patient had been diagnosed with coronary artery disease, diabetes mellitus, hyperlipidaemia, and hypertension. The patient had bilateral leg pain that increased when standing for a long time and relieved when lying down and elevating, and he stated that he applied a solution with a high methyl alcohol content for this pain, he had been applying rubbing methyl alcohol to his leg skin for a long time.The patient does not use cigarettes or alcohol.He had regularly used statin, acetylsalicylic acid, empagliflozin, and amlodipine.

During the physical examination, the patient's general condition was assessed as moderate. He was conscious, oriented, and fully cooperative. However, he exhibited complete vision loss in both eyes, and neither direct nor indirect light reflexes could be elicited. The patient displayed Kussmaul breathing. Examination of the lungs revealed equal participation in respiration, with no rales or rhonchi detected. The abdomen was soft and comfortable, with no signs of guarding or rebound tenderness. Capillary refill time was measured at 2.5 seconds. Notable redness was observed in both lower legs. No abnormalities were detected in the examination of other systems. In the fundoscopic examination, the opticdisc was bilaterally oedematous and its borders were unclear.

Laboratory findings showed hemoglobin: 15,9 g/dL, leukocyte count: 14.480/mm3, absolute neutrophil count: 10.530/mm3, and platelet count: 272,000/mm3. Biochemical tests revealed sodium 133 meq/L, potassium 5,1mEq/L, urea: 57 mg/dL, creatinine: 1,1 mg/dL, Cl:102 mg/dL C-reactive protein: 2.66 mg/dL, blood sugar: 213mg/dL. Metabolic acidosis was diagnosed with blood gas results showing a pH of 6.9, $pO_2$ of 92.13 mmHg, $pCO_2$ of 29 mmHg, $HCO_3$ of 8.1 mmol/L, lactate level of 60 mg/dL, and an anion gap of 28.8.1mmol/L.Blood methyl alcohol level could not be measured because a kit was unavailable. Coagulation parameters were normal. A complete urine test revealed pH 5, density of 1020, and ketone 1+.

In upper abdominal ultrasonography, the liver and spleen sizes and parenchyma were normal. The gallbladder was contracted. The kidneys were located and sized naturally, and parenchymal echoes were increased. There wasn't any finding of hydronephrosis. The bladder was collapsed.

Brain hemorrhage, intracranial mass, or acute diffusion restriction were not observed in CT and MRI of the brain. Contrast-enhanced cranial MRI results indicated that the choroidal fissures were slightly widened in the medial temporal lobe bilaterally. The depth of the hemispheric sulci was slightly increased, and there was bilateral symmetric gyral volume loss. A few millimetric nonspecific hyperintense signal changes on T2-flair were observed in both cerebral hemispheres (Figure-1).

The patient was suspectedof methyl alcohol toxicity with the history and clinical findings. Fomepizole could not be started because it was not available in Turkey. Parenteral ethyl alcohol was obtained by contacting the Turkish Ministry of Health Poison Control Centre. A loading dose of 1 mL/kg of 10% solution was applied, and maintenance infusion was started at a rate of 1 mL/kg/hour. Vitamin B complex treatment was started.

Due to metabolic acidosis that is resistant to medical treatment, 2-hour intermittent hemodialysis was decided. Post-dialysis control blood gases revealed pH: 7.13, $PaCO_2$: 26 mmHg, $PaO_2$: 89 mmHg, $HCO_3$: 10 mmol/L, base deficit: -18. 9 mmol/L, lactate: 55 mg/dL.

Since the ethyl alcohol level could not be measured during this period, the ethyl alcohol infusion dose was not changed. After 36 hours of infusion of ethyl alcohol solution, the patient's acidosis resolved (Figure-2).

The patient, whose general condition was good and vital status was stable, was transferred to the general internal medicine ward on the 3rd day of his intensive care admission but there was no improvement in vision loss and was evaluated by ophthalmologists as cortical permanent blindness.

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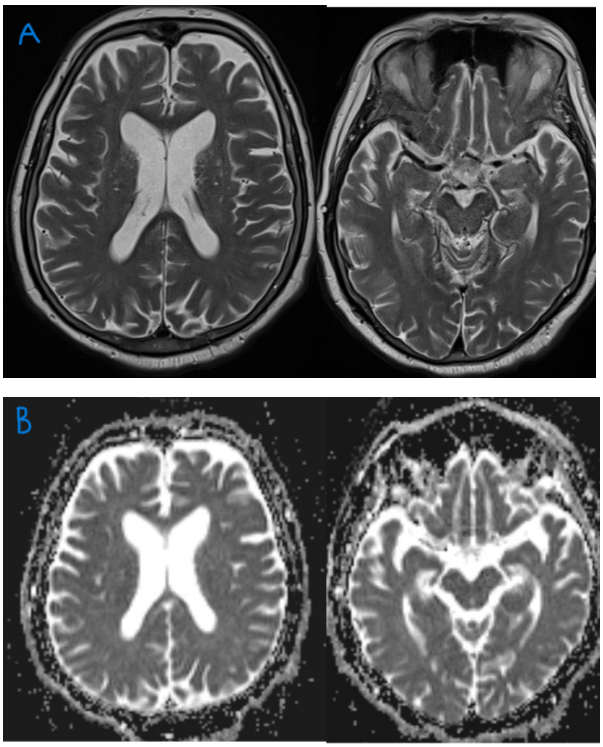


**Figure 1.** A few millimetric nonspecific hyperintense signal changes on T2-flair were observed in both cerebral hemispheres (**A:** T2-flair magnetic resonance image **B:** Diffusion-weighted magnetic resonance)

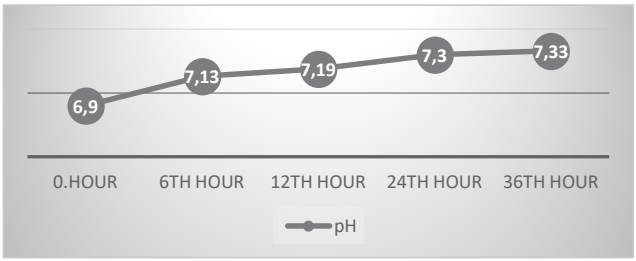


**Figure 2.** The patient's 0th, 6th, 12th, 24th and 36th hour blood gas pH monitoring

## Discussion

Methanol is generally toxic when taken orally, it can also be absorbed transdermally. Methanol absorption through the skin can vary depending on factors such as the permeability of the skin barrier, the amount of methanol, and the duration of application. Studies have shown that methanol is absorbed through the skin very slowly, but can reach toxic levels in large amounts or with prolonged exposure (9). In addition, since transdermal methanol intake can cause systemic effects to appear later, it can lead to delays in diagnosis. This can complicate the treatment process and increase the need for clinical monitoring.

In our case, since the methyl alcohol level could not be measured, the patient was diagnosed with methanol poisoning with only skin contact based on the history, characteristic clinical features, and increased anion gap metabolic acidosis. We did not consider lactic acidosis since metformin was not among the medications he regularly used, and with blood sugar being normal and ketone being 1+, we moved away from diabetic ketoacidosis.

Since methyl alcohol toxicity is a life-threatening condition that requires urgent treatment, rapid diagnosis and early treatment are of great importance. Ocular morbidity from methanol poisoning is well known. Blurred vision, which occurs when perception is normal, is a typical finding for methanol poisoning. In the early period, eye examination of patients reveals significant hyperaemia in the optic disc, and in the late period, dilatation of the retinal veins, retinal oedema, and optic disc atrophy (10). Cases of blindness have been reported after consumption of as little as 4 ml (11). Permanent blindness has been reported in 25-33% of patients with methanol poisoning (12). Although it is not oral intake, it has been observed that toxicity may develop with prolonged skin contact (13), as in our case.

Ethanol, by inhibiting the enzyme alcohol dehydrogenase, prevents the conversion of methanol to formaldehyde (4). The same enzyme pathway as methanol metabolizes ethanol, delaying the production of toxic metabolites. Ethanol therapy can be administered intravenously or orally. This treatment is crucial for patients with high methanol levels. Fomepizole is a selective inhibitor of alcohol dehydrogenase. Compared to ethanol therapy, fomepizole has fewer side effects and can be administered intravenously. This treatment prevents the formation of toxic metabolites from methanol. Metabolic acidosis is a common occurrence in methanol poisoning, and intravenous bicarbonate infusions can help balance the blood's pH levels. Lactic acid levels should also be monitored, and steps should be taken to correct the acidic environment.

Hemodialysis is fundamental in treating severely poisoned patients and is the best method to rapidly remove both toxic acid metabolites and the parent alcohol (14). The positive effects of early dialysis on neurological functions are mentioned in the literature (15). Indications for dialysis in methyl alcohol intoxication are central nervous system involvement, visual disturbances, severe metabolic acidosis (pH <7.25), acute renal failure, electrolyte disturbances, and poor response to treatment (15). The pH level of the patient when they first apply is important in the prognosis. While severe acidosis and coma indicate a poor prognosis, being conscious and able to hyperventilate at the time of application indicates a good prognosis (16). In our case, it was observed that the patient had a poor prognosis due to low pH at hospital admission, development of neurological symptoms, treatment-resistant metabolic acidosis, and chronic methyl alcohol exposure. Although publications claim that there is no difference between ethanol and fomepizole in terms of the need for hemodialysis, there are also publications stating that, on the contrary, fomepizole shortens the duration of hemodialysis (16).

## Conclusion

Transdermal methanol intoxication is a rare but serious condition that can be difficult to manage clinically. These cases demonstrate that methanol can have toxic effects not only orally but also through the skin. Early diagnosis and intervention are critical to improving the chances of recovery. Therefore, health care professionals need to recognize transdermal methanol exposure and develop appropriate strategies for treatment. In addition, raising public awareness about the dermal use of methanol is an important step in preventing such poisonings. Future studies will contribute to a beter understanding of transdermal methanol intoxication and to improving treatment methods.

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Case Report

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# Spontaneous Hepatic Artery Dissection Presenting with Common Symptoms: A Diagnostic Challenge

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## Abstract

Spontaneous hepatic artery dissection is an extremely rare subtype of isolated visceral artery dissection (IVAD) and often presents with nonspecific abdominal pain, complicating early diagnosis. Prompt recognition is essential, as delayed intervention may result in life-threatening complications including ischemia or rupture. A 44-year-old man with no significant medical history presented with postprandial right upper quadrant pain radiating to the back. Initial examination and laboratory tests, including ultrasound, were inconclusive. Given persistent clinical suspicion, computed tomography angiography revealed a long-segment hepatic artery dissection with near-complete luminal narrowing and intraluminal thrombus. Liver perfusion was preserved, with no biochemical or radiological signs of ischemia. After multidisciplinary consultation, conservative management with anticoagulation and serial Doppler ultrasonography was initiated. The patient remained clinically stable without complications. This case highlights the diagnostic challenges of hepatic artery dissection and underscores the importance of maintaining clinical suspicion in unexplained abdominal pain. In stable patients without organ ischemia, conservative treatment with close imaging follow-up can be a safe and effective management strategy. Due to its rarity, standardized treatment guidelines for hepatic artery dissection remain undefined.

**Keywords:** Anticoagulants, computed tomography, hepatic artery, dissection, visceral artery dissection

## Introduction

Approximately 5% to 10% of emergency department visits are attributed to abdominal pain. While this symptom may result from benign and self-limiting conditions, it can also be a manifestation of life-threatening pathologies that require urgent intervention. One of the rarer causes of abdominal pain is isolated visceral artery dissection (IVAD). These dissections are most commonly observed in the celiac artery, superior mesenteric artery, inferior mesenteric artery, and their branches (1). In patients presenting with abdominal pain, IVAD is often diagnosed incidentally through advanced radiological imaging, typically performed based on the clinician's level of suspicion. Indeed, several studies have shown that with the increased use of abdominal computed tomography, spontaneous IVADs are now diagnosed more frequently, including cases that may have previously gone undetected (2).

Current knowledge regarding the pathogenesis of isolated visceral artery dissection (IVAD) remains limited, and there is no established consensus on optimal treatment strategies. Given that most cases present with mild or nonspecific symptoms, conservative management is often favored as the initial approach (3). However, some researchers argue that conservative treatment may carry the risk of delayed complications, such as aneurysm formation or progression of the dissection, potentially resulting in missed opportunities for timely intervention (4). Early complications of IVAD may include dissection rupture, organ ischemia, and tissue necrosis, while the most serious late complication is the rupture of a dissecting aneurysm, which can lead to life-threatening outcomes (5).

The aim of presenting this case is to highlight that, although more common visceral artery pathologies such as celiac or mesenteric artery dissection and thrombosis are typically considered first in patients presenting to the emergency department with acute abdominal pain, dissection and thrombosis of the hepatic artery—an exceptionally rare localization—should not be overlooked in the differential diagnosis. As demonstrated in this case, with appropriate clinical suspicion and timely imaging, this uncommon yet potentially serious condition can be accurately identified and managed before critical complications arise.

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## Case Report

A 44-year-old male patient with no known systemic illness presented to the emergency department with a two-day history of intermittent and progressively worsening abdominal pain. He described the pain as a continuous, squeezing sensation localized to the right side of the abdomen, radiating to the back. The patient also noted that the intensity of the pain increased following meals. His medical history included a 20-year history of smoking approximately one pack of cigarettes per day. On initial evaluation in the emergency department, his vital signs were stable (blood pressure: 120/70 mmHg, heart rate: 86 bpm, respiratory rate: 18 breaths/min, body temperature: 36.5°C). Abdominal examination revealed marked tenderness in the right upper quadrant, but no guarding or rebound tenderness was observed. No pathological findings were detected on systemic examination. These physical findings suggested a visceral origin of the abdominal pain.

At the time of presentation to the emergency department, the patient's complete blood count revealed borderline leukocytosis (WBC: 13.23 × $10^3$/μL). Aside from this finding, no abnormalities were noted in other hematological parameters or in the extended biochemical panel. The electrocardiogram (ECG) showed normal sinus rhythm, with no evidence of ischemia. Following the clinical evaluation, a bedside abdominal ultrasound was performed. No acute pathology was observed in the intra-abdominal solid organs or adjacent structures, and the appendix appeared normal in thickness and surrounding tissue. Given the elevated leukocyte count, particular attention was paid to evaluating the appendix for possible retrocecal appendicitis; however, no significant findings were noted to support this diagnosis. As the visceral nature of the abdominal pain persisted and its postprandial exacerbation raised suspicion for mesenteric angina, a contrast-enhanced computed tomography angiography (CTA) of the abdomen was planned to assess the abdominal aorta and its branches.

Abdominal computed tomography angiography demonstrated a dissection flap extending along the hepatic artery (Figure-1). The dissection originated from the proximal segment and was characterized by an intraluminal thrombus spanning a long segment, leading to near-complete luminal narrowing. Additionally, small thrombi were observed in the proximal splenic artery, although these did not significantly compromise the vessel diameter. Perfusion of the liver with the true lumen was preserved, and no radiological signs of ischemic damage were detected in the hepatic parenchyma. These findings were consistent with the patient's liver function tests, which remained within normal limits.

Following imaging, anticoagulant therapy was initiated with 60 mg of low-molecular-weight heparin administered intravenously, and intravenous isotonic fluid was given for hydration. A multidisciplinary bedside evaluation involving cardiovascular surgery, gastroenterology, general surgery, and interventional radiology concluded that the patient should initially be managed medically, with serial follow-up of the hepatic artery via Doppler ultrasonography. The interventional radiology team considered the proposed procedure to be high-risk due to the vascular anatomy and the length of the dissection segment. Accordingly, Doppler ultrasound follow-ups were scheduled weekly at first, with

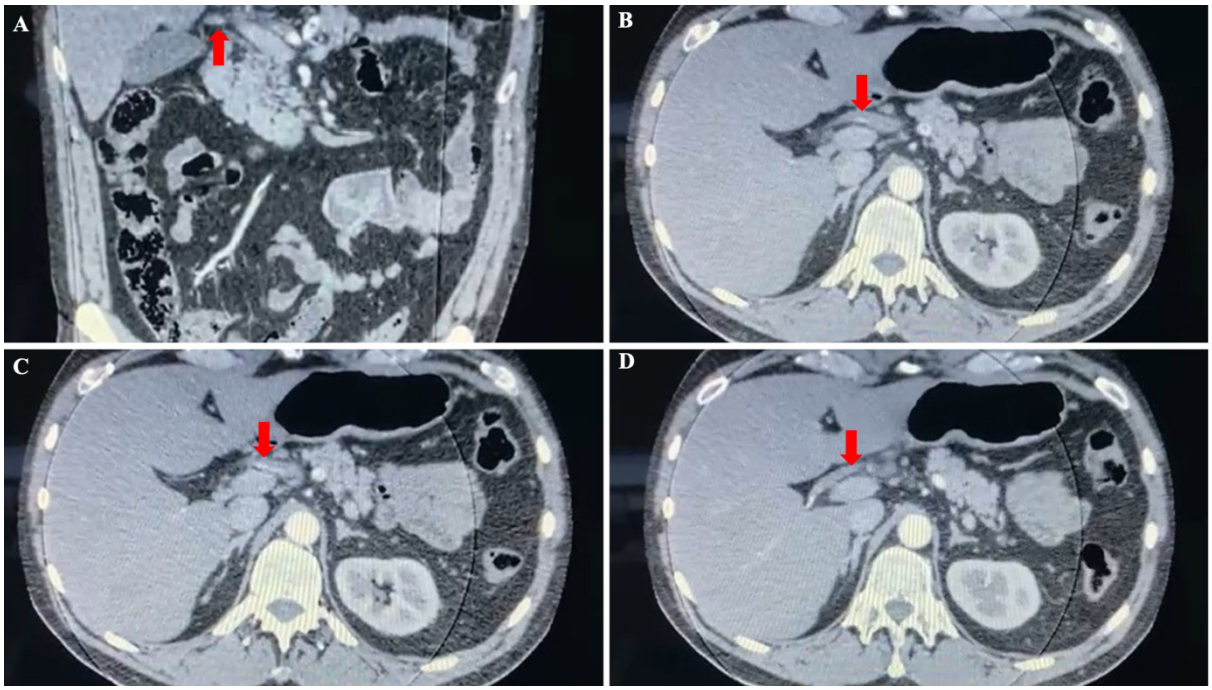


**Figure 1.** Imaging findings of hepatic artery dissection on abdominal CT angiography. The dissected segments are indicated by red arrows (A: Coronal view; B, C, D: Axial views).

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gradually extended intervals based on clinical stability.

Pain control was successfully achieved. A treatment plan was established by general surgery, cardiovascular surgery, and gastroenterology teams involving subcutaneous administration of low-molecular-weight heparin at a dose of 60 mg twice daily. Additionally, the patient was provided with guidance on an appropriate diet and nutritional regimen.

Written informed consent was obtained from the patient for the anonymized use of clinical data and radiological images in this case report. This case report was prepared in accordance with the principles of the Declaration of Helsinki.

## Discussion

Visceral artery dissections (IVADs) are rare vascular pathologies that can occur in isolation, without any association with aortic dissection. They most commonly involve the superior mesenteric artery, the celiac artery, and their major branches. Hepatic artery dissection represents one of the rarest forms of IVAD and has been described in the literature through only a limited number of case reports (6). Etiologic factors implicated in IVAD include hypertension, smoking, vasculitis, trauma, and structural abnormalities of the arterial wall (1, 6, 7). In the present case, the only identifiable risk factor was a long-standing history of cigarette smoking.

In reported cases of hepatic artery dissection, 71% have been documented to present with visceral abdominal pain localized to the right upper quadrant (7). The underlying mechanism of pain is thought to involve luminal narrowing and vascular tension caused by the dissection process itself. Although the clinical presentation often resembles mesenteric ischemia, the diagnosis is most frequently made incidentally through advanced imaging modalities such as computed tomography (2). In the present case, no pathology was identified on initial investigations; however, due to persistent clinical suspicion, CT angiography was performed, which confirmed the diagnosis of hepatic artery dissection. The clinical importance of this rare vascular condition lies in its potential to progress to rupture or organ ischemia, with fatal outcomes reported in up to 44% of cases (7). This case illustrates a rare presentation of hepatic artery dissection that was not identified on initial evaluation but was diagnosed through advanced imaging. It highlights the fact that the diagnosis of visceral artery dissections often depends on high clinical suspicion guiding the use of appropriate imaging techniques.

Several case reports in the literature have demonstrated that hepatic artery dissection can be successfully managed with medical therapy and radiological follow-up alone. Dong et al. (8) emphasized that, in patients without signs of peritoneal irritation, a short course of anticoagulant therapy may be sufficient. Treatment strategies in such cases should be tailored to the patient's clinical presentation and accompanied by continued radiological monitoring using ultrasonography. In addition, endovascular embolization has been proposed as a potential alternative in patients at high surgical risk. In such instances, careful assessment of the liver's collateral perfusion capacity is considered essential (9).

Thrombus formation within the true lumen may lead to compromised hepatic perfusion and subsequent ischemic injury, which can necessitate urgent intervention. When such complications are present or carry a high risk of developing, surgical management should be prioritized. Nevertheless, due to the limited number of hepatic artery dissection cases reported in the literature, there is still a lack of standardized guidelines regarding optimal patient management (10).

In the present case, the dissection involved the hepatic artery along a long segment, which was deemed technically high-risk for intervention by the interventional radiology team. Moreover, no radiological or biochemical evidence suggestive of hepatic ischemia was identified. Based on a multidisciplinary bedside evaluation, conservative management combined with regular follow-up using Doppler ultrasonography was considered a safe and appropriate approach.

## Conclusion

Hepatic artery dissection is a rare form of isolated visceral artery dissection, typically presenting with nonspecific abdominal pain and often requiring advanced imaging for diagnosis. Maintaining clinical suspicion and conducting appropriate radiological evaluations are critical for timely diagnosis and the prevention of potentially severe complications. In cases where the patient is hemodynamically stable and organ perfusion is preserved, conservative management combined with close radiological monitoring under a multidisciplinary approach represents a safe and effective treatment strategy.

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# A Rare Case Report of Acute Vitamin D Toxicity due to Suicide Attempt

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## Abstract

Acute vitamin D toxicity is an uncommon event that might happen especially owing to attempted of suicide. Vitamin D toxicity could induce hypercalcemia, Acute kidney injury, and cardiac problems in patients. A 49-year-old Iranian man who has a known case of cerebellar palsy and major depressive disorder refers to us with an issue of calcium – vitamin D tablets overdose due to suicide attempt. He admitted with impression of acute vitamin D toxicity. After treatment with hydration, dialysis, Lasix, calcitonin, Denosumab, and Zoledronic Acid patient clinics improved significantly. Physicians must treat vitamin D overdose patients immediately to prevent secondary adverse effects of hypercalcemia.

**Keywords:** Case report, hypercalcemia, suicide attempt, vitamin D toxicity

## Introduction

Vitamin D is categorized as a fat-soluble vitamin that remains long time in humans' adipose tissue and is released in the body slowly (1, 2). Vitamin D improves humans' immune system and also prevents cell mutations, thus it could prevent many types of malignancies (3). In addition, vitamin D is mandatory for bone mineralization, skeletal firmness, and prevention of osteoporosis (1, 3). Not only in Iran but also in many countries a huge number of individuals suffer from insufficient levels of vitamin D as a global issue (4, 5). Therefore, it is common to purchase vitamin D supplements as on-the-counter medication or online (3, 4). By using the unmonitored and unprescribed amount of vitamin D, drug toxicities might be occurred (2, 6). Many studies mentioned that vitamin D levels for adults should be between 30 pg/ml and 60 pg/ml and the above level of 150 pg/ml is categorized as a toxic level that is harmful to the patient hemostasis, thus controlling the amount of vitamin D level is literally mandatory and helpful to prevent further medical conditions (1, 7, 8).

Individuals who suffered from toxic levels of vitamin D mainly presented with nausea, vomiting, headache, sensorium, loss of conciseness, drowsiness, polyuria, and in rare cases seizure (5, 6). Furthermore, toxic levels of vitamin D could affect different aspects of the human body. Hypercalcemia, short QT interval on electrocardiogram (ECG), pancreatitis, and acute kidney injury (AKI) are the main significant harmful consequences of hypervitaminosis D that must be treated immediately (3, 5, 7-9).

In many cases process of vitamin D toxicity happens chronically as a result of using uncontrolled amounts and self-prescribing amount of vitamin D supplements without any medical indication for a long period of time (4). Herein, in the current novel case report our aim is to present a patient who experienced vitamin D toxicity acutely as a consequence of suicide attempt.

## Case Report

A 49 years old Iranian married man known case of cerebral palsy (CP) and major depressive disorder (MDD) was referred to Loghman Hakim Hospital, Tehran, Iran with refer reason of a high level of calcium and vitamin D owing to poisoning with 60 tablets of vitamin D - calcium, an unknown amount of ibuprofen 400mg and adult cold tablets. Moreover, he used Bupropion as a past drug history. As well, in his social history he did not use alcohol or cigarette and his family history was negative. At the time of admission, his blood pressure was 100/54, temperature 36.8 degrees

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Celsius, respiratory rate 17, heart rate 80, and O2 saturation 96%. In addition, although the condition of the patient was ill, he had a normal clinical examination, his neurological examinations such as deep tendon reflux, responses to light, and so on were literally normal. He is oriented to time, place, and person, however, he answered to our question with delay. Also, his Glasgow Coma State was 15 out of 15, and his muscle power was 4 out of 5. The patient did not have neck rigidity, headache, nausea, vomiting, and sensorium. The patient's laboratory investigation (table 1) depicted the following abnormal results: Calcium 17.7 mEq/L, Vitamin D level (25-OH vitamin D3) 2407 ng/ml, Blood Urea Nitrogen (BUN) 77 mg/dl, Creatinine (Cr.) 2.4 mg/dl, and LDH 781. His Vessels Blood Gas (VBG) showed ph 7.46, co2 51.1, hco3 36.9, and base excess 11. Also, his Urine Analysis showed a specific gravity of 1.020, urine protein +, and his stool exam was negative. He had a normal parathyroid hormone (PTH) level (6.4 pg/ml), normal electrocardiogram (ECG) without QT prolongation and ST changes, and normal abdomen-pelvic sonography. Finally, he was admitted to the intensive care unit (ICU) with the impression of hypercalcemia on the base of acute vitamin D overdose (toxicity) due to attempt suicide. During the course of hospitalization, as a consult with a nephrologist and an endocrinologist his hypercalcemia was treated with intravenous normal saline 4lit/ day, intravenous Lasix 20 mg, calcitonin, intravenous Denosumab 60 mg, and Zoledronic Acid 4 mg/ 100 ml solution for infusion. Furthermore, during patient hospitalization, he was faced with hypokalemia which was treated with a suitable amount of potassium in his intravenous serum therapy. Also, hemodialysis was performed for his AKI and his electrolyte imbalance. His laboratory data such as complete blood count (CBC), 25-OH vitamin D3, calcium, phosphor, sodium, potassium, BUN, Cr, VBG, and urine analysis monitored regularly. During 14 days, he illustrated clinical improvement and his laboratory data diminished gradually. Also, during the course of admission, several psychologists visited him owing to his attempted suicide. Finally, the patient was discharged after 3 weeks from the internal ward with a prescription for a calcium and vitamin D restricted diet. Moreover, the patient was referred to a psychiatrist for his suicidal ideas. We recommend him for 1 month follow up, however, due to living in other city he lost his follow up.

**Table 1:** Patient's laboratory test findings at the time of admission

| Laboratory test | Result | Unit | Reference Range |
|---|---|---|---|
| CBC | | | |
| WBC | 7.9 | $10^3/\mu L$ | 4.5 - 11 |
| RBC | 5.07 | $10^6/\mu L$ | 4.6 – 6.2 |
| Hemoglobin | 14.5 | g/dL | 13.5 – 17.5 |
| Hematocrit | 43 | % | 45 - 52 |
| M.C.V. | 84.81 | fL | 80 - 100 |
| M.C.H. | 28.6 | pg | 27 - 32 |
| M.C.H.C. | 33.72 | g/dL | 32-36 |
| Platelets | 213 | $10^3/\mu L$ | 150 - 450 |
| RDW-CV | 14 | % | 11 – 14.5 |
| Coagulation test | | | |
| PT (Patient) | 13.4 | Sec | 10 - 14 |
| INR | 1.12 | | |
| PT (Control) | 12.0 | Sec | 12.0 |
| PTT | 31 | Sec | 27 - 45 |
| Biochemistry | | | |
| BUN | 77 | mg/dL | 19 - 44 |
| Creatinine | 2.4 | mg/dL | 0.9 – 1.4 |
| SGOT | 17 | IU/L | Up to 43 |
| SGPT | 24 | IU/L | Up to 40 |
| Alkaline Phosphatase | 318 | U/L | 64 - 319 |
| LDH | 781 | | Up to 480 |
| Calcium | 17.7 | mEq/L | 8.5 – 10.2 |
| Phosphor | 3.4 | mEq/L | 2.6 – 4.5 |
| Sodium | 144 | mEq/L | 135 - 145 |
| Potassium | 3.5 | mEq/L | 3.5 – 5.1 |
| Magnesium | 2.0 | mEq/L | 1.9 – 2.5 |
| 25-Hydroxy Vitamin D3 | 2407 | ng/ml | Toxicity: >150 |
| Amylase | 42 | U/L | Up to 110 |
| Lipase | 21 | U/L | Up to 60 |
| Hormonal Test | | | |
| PTH | 6.4 | pg/ml | Up to 65 |
| TSH | 1.08 | µlu/ml | 0.3 – 4.04 |
| T3 | 0.94 | ng/ml | 0.52 – 1.85 |
| T4 | 64.1 | Ng/ml | 51 - 125 |

CBC: Complete Blood Count, WBC: Wight Blood Cell, RBC: Red Blood Cell, M.C.V.: Mea Corpuscular Volume, M.C.H.: Mean Corpuscular Hemoglobin, M.C.H.C: Mean Corpuscular Hemoglobin Concentration, RDW-CV: Red Cell Distribution Width–Coefficient of Variation, PT: Prothrombin Time, INR: International Normalized Ratio, PTT: Partial Thromboplastin Time, BUN: Blood Urea Nitrogen, SGOT: Serum Glutamic Oxaloacetic Transaminase, SGPT: Serum Glutamic Pyruvic Transaminase, PTH: Para-Thyroid Hormone, TSH: Thyroid Stimulating Hormone

## Discussion

As we know, many previous studies depicted that vitamin D toxicity occurred chronically due to unmonitored use of vitamin D supplement both orally as tablet or pearl and/or intravenous as vitamin D ampoule (4, 6, 7). However, the current case report illustrated the patient had been poisoned with vitamin D acutely as a result of overdose of 60 tablets of calcium - vitamin D, so this case report is novel. In addition, as the same as our patient, John P. Lee et. al in their study mentioned that the serum level of vitamin D did not correlate with the patient's symptoms and in acute vitamin D toxicity many patients did not present any specific symptoms, for instance; nausea, vomiting, headache, and sensorium (5).

Vitamin D is a prohormone component that is mandatory for a healthy lifestyle and it affect calcium hemostasis (7, 10). Vitamin D prevents many types of mutation which

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occasionally happen in the pathophysiology of several malignancies (11). As well, it plays a significant role in cell growth. Also, it is quite mandatory for bone mineralization and stability of skeletal structure (12). In addition, vitamin D could improve the human immune system, especially during the COVID – 19 pandemic many studies illustrated the role of vitamin D immunization against the virus (13-15). Therefore, Vitamin D supplements are available as over-the-counter medication in drugstores and it could easily be accessible (4). As a result of that, many clients use vitamin D routinely without any vitamin D level monitoring or prescription (4, 7, 16). Thus vitamin D toxicitymight happen in a number of patients (6). As a consequence of hyper-vitaminosis D, life-threatening hypercalcemia might be happen and it could impact patients' kidneys and AKI as a main secondary disorder might presented (9, 17-19). Also, it could cause patients cardiac ventricular repolarization abnormalities thus cardiac arrhythmias might present in patients (20, 21), therefore immediate treatment of hypercalcemia and vitamin D overdoseis crucial (22).

In the current case report, the patient overdosed calcium - vitamin D tablets for suicidal issues. His hypercalcemia might be affected secondary to an overdose of vitamin D as a secondary metabolic pathway, and also as a main pathway induced by calcium tablets overdose. Although his ECG was normal, as a result of his laboratory data(BUN 77 mg/dl and Cr. 2.4 mg/dl), the patient initially suffered from AKI secondary to his drug overused.Thus, we performed hydration and hemodialysis for his kidney injury and electrolyte imbalance to prevent the worsening of his condition. As well, K. Feghali et. al illustrated that in vitamin D toxicity which induced AKI, a dialysis is a suitable option for patient treatment and it would reduce the side effects of hypervitaminosis D (4). In addition, as a consult with nephrologist and endocrinologist the patient (with a calcium serum level of 17.7 mEq/l)was treated by Calcitonin, Denosumab, and Zoledronic Acid were started to diminish his calcium serum level to prevent other adverse effects of hypercalcemia (23) as the same as M.M. Basso S. et al study (24). Furthermore, the patient was evaluated for his suicide attempt by a psychiatrist and his MDD treatments were prescribed. Also, at the time of discharge he did not suffer from any suicidal ideas.

Finally, we suggest that in acute toxicity with vitamin D, immediate patient management is mandatory and physicians must immediately proceed patient's treatment to prevent life-threatening adverse effect of acute hypercalcemia as a secondary pathway of vitamin D toxicity. Furthermore, when a patient admitted to the emergency ward owing to an attempted suicide, over-the-counter medication such as calcium–vitamin D tablet overdose must be considered.

## Conclusion

This case report underscores the critical importance of recognizing that vitamin D toxicity can arise not only from chronic exposure but also following acute ingestion. Emergency physicians should remain alert to the potential for severe, and even fatal, complications associated with single high-dose administration. Early recognition and prompt management of acute vitamin D overdose are essential in mitigating the secondary effects of hypercalcemia and improving patient outcomes.

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