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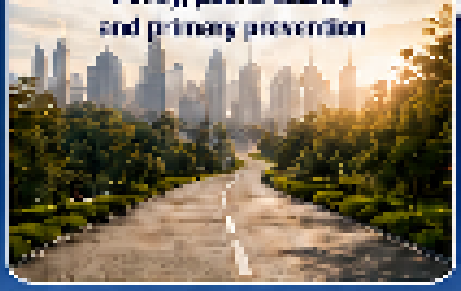
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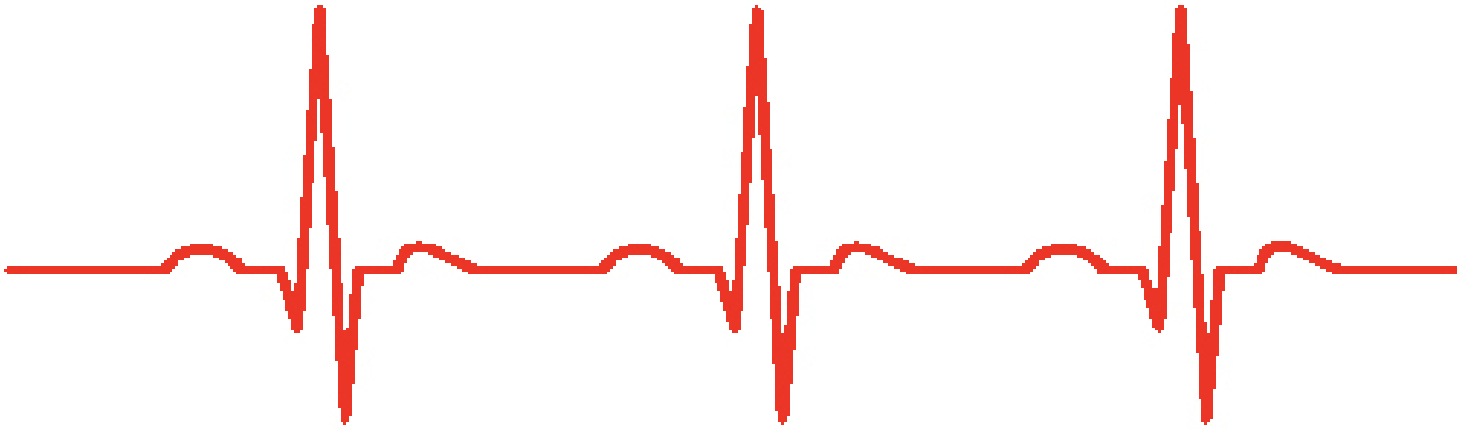
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CPCEM Clinical Practice & Cases

59-year-old Woman with Acute Blindness

Nicolas Contillo, MD*
Michael Sibel, DO†
Laura J Bontempo, MD, MEd†
John David Gatz, MD†

*University of Maryland Medical Center, Department of Emergency Medicine, Baltimore, Maryland
†University of Maryland School of Medicine, Department of Emergency Medicine, Baltimore, Maryland

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When a patient with acute blindness presents to the emergency department (ED), the evaluation typically suggests one of the "can't miss" diagnoses ingrained in emergency physicians during training. How does the differential change when the patient's presentation does not neatly reveal an expected pathology? A 59-year-old woman presented to the ED with acute binocular blindness in the setting of three days of a flu-like illness. This case offers a practical approach to opening the lens on the presentation of acute blindness. [Clin Pract Cases Emerg Med. 2026;10(4):434–439.]

CASE PRESENTATION (DR. CONTILLO)

A 59-year-old woman with a history of asthma, hypertension, type 2 diabetes mellitus with neuropathy, and seasonal allergies presented to the emergency department (ED) after she awoke with complete bilateral vision loss accompanied by nausea, emesis, and diarrhea. She reported three days of flu-like symptoms, including subjective fever, myalgias, nausea, diarrhea, and "dark" emesis. Similar symptoms were reported among other members of her household. Her vitals on arrival were as follows: blood pressure, 83/48 mm Hg; heart rate, 80 beats per minute; respiratory rate, 21 breaths per minute; SpO_2, 100% on room air; and temperature, 31.6 °C.

The patient had been unable to tolerate oral medications for several days due to emesis and admitted to taking a "double dose" of her routine medications the previous day to compensate. She denied any illicit substance ingestion. Past surgical history included a cesarean section. Family history included hypertension and breast cancer. Her home medications were albuterol, cetirizine, fluticasone nasal spray, atorvastatin, gabapentin, metformin, tirzepatide, valsartan, metoprolol, vitamin D, and ferrous sulfate. She was allergic to iodinated contrast (anaphylaxis), lisinopril (angioedema), latex, and the COVID-19 vaccine.

Physical examination revealed a toxic-appearing woman in extremis. She had no external signs of trauma such as abrasions, lacerations, or ecchymoses. Her ocular exam was notable for complete binocular vision loss with 3-mm, round, nonreactive pupils bilaterally. There was no proptosis, lid edema, exudate, conjunctival injection, hyphema, or hypopyon. Her extraocular movements were intact. Neurologic exam was notable for full strength in all extremities, normal light touch sensation, and preserved cranial nerve function with the exception of cranial nerve II. Her mucous membranes appeared dry. Her neck was supple without signs of meningismus. Her heart sounds were normal without murmurs. Her lung fields were clear, and respiratory effort was normal. Her abdomen was soft, nondistended, with mild generalized tenderness and no rebound or guarding. Her extremities were cool to touch, with delayed capillary refill >3 seconds and thready radial and dorsalis pedis pulses. There was no edema nor were there abnormal dermatologic findings such as rashes or mottling.

Initial laboratory studies (see Table) demonstrated a severe elevated anion gap metabolic acidosis, low bicarbonate, and markedly elevated serum lactate. An electrocardiogram (ECG) was obtained (see Figure). Computed tomography (CT) imaging of the brain, chest, abdomen and pelvis was obtained without use of intravenous contrast due to her previously documented history of anaphylaxis to iodinated contrast. No acute abnormalities were observed.

CASE DISCUSSION (DR. SIBEL)

Presented is a 59-year-old woman with a chief complaint of acute, complete blindness preceded by a flu-like illness with

Table 1. Laboratory results of a 59-year-old woman with acute blindness.

Test Name	Patient Value	Reference Range
Complete blood count		
White blood cell	23.8 K/mcL	4.5-11 K/mcL
Hemoglobin	11.0 g/dL	11.9-15.7 g/dL
Hematocrit	36.6%	35.0-45.0%
Platelets	304 K/mcL	153-367 K/mcL
Neutrophils	61.2%	40.0-70.0%
Lymphocytes	21.8%	20.0-45.0%
Eosinophils	0.4%	0.0-6%
Coagulation studies		
Prothrombin time	19.2 seconds	11-13.5 seconds
International normalized ratio	1.5	0.8-1.2
Serum chemistries		
Sodium	137 mmol/L	136-145 mmol/L
Potassium	6.6 mmol/L	3.5-5.1 mmol/L
Chloride	93 mmol/L	98-107 mmol/L
Bicarbonate	<5 mmol/L	21-30 mmol/L
Blood urea nitrogen	60 mg/dL	7-17 mg/dL
Creatinine	11.54 mg/dL	0.52-1.04 mg/dL
Glucose	283 mg/dL	70-100 mg/dL
Albumin	5.0 g/dL	3.2-4.6 g/dL
Anion gap	Indeterminate*	4-12 mEq/L
Magnesium	2.5 mg/dL	1.6-2.6 mg/dL
Phosphorus	19.0 mg/dL	2.5-4.5 mg/dL
Lactate	>17 mmol/L	0.5-2.0 mmol/L
Hepatic studies		
Total bilirubin	1.2 mg/dL	0.3-1.2 mg/dL
Aspartate aminotransferase	80 units/L	14-36 units/L
Alanine aminotransferase	45 units/L	0-34 units/L
Alkaline phosphatase	68 units/L	38-126 units/L

positive sick contacts in the home. Her symptoms were associated with vomiting of dark emesis along with diarrhea. She had been unable to tolerate her medications but reported that she took a "double dose" of her medications the day prior to her ED presentation.

When presented with a patient with acute blindness my first inclination is to consider an ocular problem. Diagnoses that come to mind are retinal detachment, central retinal artery occlusion, central retinal vein occlusion, and vitreous hemorrhage. While possible, these pathologies would not be expected to present with bilateral blindness. This opens the differential substantially, bringing intracranial and systemic processes into consideration. More posterior in the optic chain is the optic nerve and chiasm. Bilateral optic neuritis is possible but again typically presents unilaterally. Compression on the optic chiasm could cause a bilateral visual disturbance. Diagnoses here include pituitary adenoma, pituitary apoplexy, hypothalamic masses, and craniopharyngioma. Most of these diagnoses are indolent and would be expected to first present with visual field deficits prior to complete bilateral blindness. The final consideration involving the optic chain would be the vision center in the brain. Ischemic stroke and intracranial hemorrhage should be considered. Systemic processes that could cause bilateral blindness include toxic ingestions leading to toxic retinopathy, metabolic retinopathy, and severe retinal ischemia secondary to a shock state.

The patient's past medical history was unremarkable and offered no clear indicators regarding the underlying etiology. Her history of diabetes and hypertension, however, raise thoughts to a potential thromboembolic condition such as an ischemic stroke or amaurosis fugax. Additionally, her diagnosis of diabetes could explain blindness in the setting of diabetic retinopathy (DR); however, I would have expected this to be a more indolent course. She denied substance use,

Table. Laboratory results of a 59-year-old woman with acute blindness (continued).

Test Name	Patient Value	Reference Range
Venous Blood Gas		
pH	<7.00	7.31-7.41
pCO_2	19 mmHg	41-51 mmHg
pO_2	64 mmHg	30-50 mmHg
Additional Labs		
Lipase	2,948 U/L	10-60 U/L
Ethanol level	<10 mg/dL	<10.0 mg/dL
Methanol level	<5 mg/dL	<5 mg/dL
Acetone	Small	Negative
Thyroid stimulating hormone	0.4 mIU/L	0.4-4.5 mIU/L
Free thyroxin	1.1 ng/dL	0.8-1.8 ng/dL
Serum osmolality	349 mOsm/kg	275-295 mOsm/kg
Urinalysis studies		
White blood cell count	0 - 2 per hpf	0-5 per hpf
Red blood cell count	3 - 5 per hpf	0-2 per hpf
Leukocyte esterase	negative	negative
Nitrite	negative	negative
Bacteria	negative	negative
Urine pregnancy	negative	negative
Glucose	2+	negative
Ketones	1+	negative

*Unable to calculate due to unmeasurable bicarbonate level.
dL, deciliter; *g*, grams; *hpf*, high-power field; *K*, thousands; *kg*, kilogram; *L*, liter; *mcL*, microliter; *mEq*, milliequivalent; *mg*, milligram; *mIU*, milli-international units; *mmol*, millimole; *mmHg*, millimeters of mercury; *mOsm*, milliosmole; *ng*, nanogram; *pCO_2*, partial pressure of carbon dioxide; *pH*, potential of hydrogen; *pO_2*, partial pressure of oxygen; *U*, units.

making the likelihood of an unprescribed intoxicant such as methanol less likely. Her medications do not seem to be out of the ordinary for her medical history. However, it is notable that she is on tirzepatide, which is a glucagon-like peptide-1 receptor agonist (GLP-1RA). Recent literature has linked the use of medications like this to increased risk of new-onset non-arteritic anterior ischemic optic neuropathy (NAION) in addition to DR.[1,2] While a more insidious course would be expected for DR, a sudden onset of vision loss could be explained by NAION. That, therefore, would make it easy to anchor on tirzepatide being the causative agent of the patient's vision changes. However, this does not explain the whole clinical picture including the hypotension, hypothermia, acute kidney injury, and lactic acidosis, necessitating exploration of alternative etiologies for the patient's blindness.

Her vital signs are most remarkable for hypotension and hypothermia. These two findings help to narrow the differential when considering them as being caused by the same disease process. They raise diagnoses such as sepsis or endocrine pathology (ie, hypothyroidism, adrenal crisis, hypoglycemia, and toxicological exposure) to the top of the differential. Hemorrhage and hypovolemic shock also remain on the differential given her history of diarrhea and dark emesis. Her respiratory status and normal oxygenation make obstructive shock pathologies, such as massive pulmonary embolism and tension pneumothorax, less likely. New-onset cardiogenic causes are also a consideration; however, hypothermia would not typically be expected with these diagnoses. A point-of-care ultrasound to assess her ejection fraction and inferior vena cava diameter would be useful in taking these off the differential.

The patient's exam is most notable for complete binocular blindness with 3-mm nonreactive pupils. An intact pupillary light reflex requires a functional retina, optic nerve, brain stem, and cranial nerve III. Her normal level of consciousness eliminates brain stem dysfunction, and bilateral cranial nerve III deficits would be highly unlikely. This, therefore, leads me to believe that the source of blindness is noncortical and that the origin is somewhere in the retinas or optic nerves. Considering the patient's presentation, high on the differential would be toxicologic/metabolic retinopathy, severe bilateral retinal ischemia, and pituitary apoplexy. Reassuringly, the remainder of her neurologic exam is normal removing a central nervous system cause of the patient's presentation from

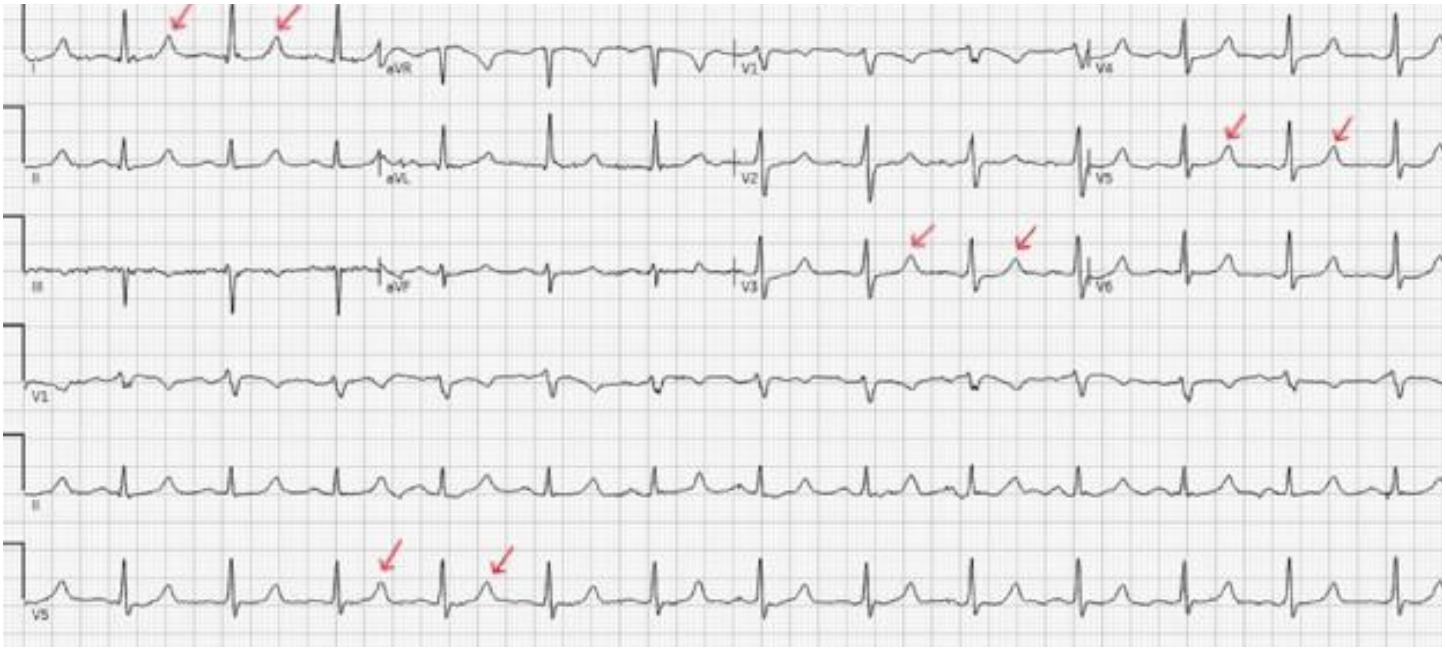


Figure. Electrocardiogram of a 59-year-old woman with acute blindness. Red arrows highlight peaked T waves.

the differential because bilateral blindness from central nervous system pathology would be expected to have other neurologic findings as well. Most diagnoses that I would be looking for in acute blindness such as retinal detachment, optic neuritis, posterior reversible encephalopathy syndrome, and idiopathic intracranial hypertension do not fit with the patient's hemodynamic picture because these pathologies are more localized and are not known to present in a shock state.

Her labs were most notable for an indeterminant anion gap, lactic acidosis, and severe kidney injury. On further investigation, the anion gap is indeterminant due to a critically low bicarbonate that surpassed the tolerances of the lab equipment, giving a value of less than 5 millimoles per liter. Through manual calculation I can estimate her anion gap to be between 39 and 44, adding a profound elevated anion gap to her notable lab values. Her complete blood count (CBC) showed a significant leukocytosis but without a shift in cell lines, which could be explained by an acute phase reaction. This, in addition to relatively unremarkable imaging and essentially negative urinalysis (UA), moves sepsis lower on the differential. Her CBC showed a normal hemoglobin and hematocrit which, along with no source of bleeding, removes hemorrhage from the differential.

Other nonspecific lab abnormalities including elevated lipase and mildly elevated aspartate aminotransferase are less concerning for pancreatic and liver pathology in the setting of negative imaging and nonfocal abdominal tenderness. Of note, acetone and glucose were present in the UA, and she also had an elevated blood sugar, which raised the possibility of diabetic ketoacidosis (DKA). However, small acetone does not necessarily explain the significant anion gap. Early DKA is a consideration but does not explain the patient's entire clinical presentation. Toxic alcohol exposure is excluded because her methanol and ethanol levels are negative. The patient's thyroid stimulating hormone and thyroxine are normal, removing myxedema from the differential. Additionally, her complete metabolic panel does not show electrolyte abnormalities that would suggest pituitary apoplexy or adrenal pathology. Her potassium and phosphorus are elevated, which can be explained by renal insufficiency. Her ECG demonstrates a normal sinus rhythm with normal axis, intervals, and mildly peaked T waves, which would be consistent with her elevated potassium.

Her head CT imaging is negative. While this cannot completely rule out an acute ischemic process, it does, in addition to a normal neurological exam outside her vision loss, help to exclude a central neurologic process from the differential. Additionally, there is no large sellar mass to suggest pituitary apoplexy as a cause for her blindness.

With the differential substantially narrowed, what is left that could explain the patient's bilateral blindness with hypotension, hypothermia, and severe lactic acidosis? For acute bilateral, noncortical blindness, severe bilateral retinal ischemia could be caused by systemic hypotension or toxic/metabolic retinopathy. We have ruled out most pathologies that can cause hypotension; however, severe lactic acidosis could be the culprit, further exacerbated by hypovolemia in the setting of vomiting and diarrhea. With hypothermia, drugs and toxicologic causes are the highest on the differential. With these differentials in mind, the most significant diagnostic findings of profound lactic acidosis and kidney injury can be used to narrow the possibilities. There are only a few things that can cause such profound lactic acidosis in an awake and

alert patient. Potential etiologies include an ischemic process such as limb or bowel ischemia. However, the patient's exam and CT imaging did not reveal any such process. The other is toxicologic, specifically toxins that disrupt metabolism. There is, however, no history to suggest that the patient encountered a significant toxic exposure, except, possibly through her prescribed medications.

In reviewing her medications, the one that stands out is metformin. Metformin inhibits oxidative phosphorylation and shifts cells to anaerobic metabolism. When toxic levels are reached, severe lactic acidosis can occur. This syndrome is known as metformin-associated lactic acidosis (MALA). While there is no history to suggest that she overdosed, she had taken a double dose of her medications the day prior. Typically, this would not be enough to reach toxic blood levels of metformin. Remembering the patient's history, however, she reported that her blindness was preceded by three days of illness which was associated with nausea, vomiting, and diarrhea. This likely caused acute kidney injury (AKI) which would have affected metformin clearance, as metformin is mostly cleared renally. The "double dose" that she took in the setting of profound AKI likely led to toxic metformin levels. This is well documented as a cause of MALA. Her blindness could be explained by toxic metabolic pathology due to severe lactic acidosis. Considering all the above factors in a patient presenting with acute bilateral blindness, a serum metformin assay would be the diagnostic test of choice.

CASE OUTCOME (DR. CONTILLO)

The patient's metformin level ultimately resulted at 17 mcg/mL, well above the therapeutic range of 1-2 mcg/mL, confirming the diagnosis MALA. The patient was admitted to the medical intensive care unit and initiated on high-intensity continuous renal replacement therapy (CRRT) within hours of arrival. She required transient vasopressor support, which was discontinued within 24 hours as the acidosis resolved. Neurologic function and vision fully recovered by hospital day six. She was ultimately discharged home in stable condition with transition to an insulin-based regimen for diabetes management.

RESIDENT DISCUSSION (DR. CONTILLO)

Metformin, a biguanide derived from *Galega officinalis* (goat's rue), was first described in 1922 and approved by the US Food and Drug Administration for use in 1994. It acts primarily via stimulation of the adenosine monophosphate-activated protein kinase pathway, improving insulin sensitivity and decreasing hepatic gluconeogenesis.[3] Toxicity occurs through inhibition of mitochondrial cytochrome complexes, leading to failure of oxidative phosphorylation. As a result, pyruvate metabolism shifts toward lactate production and reactive oxygen species accumulation, resulting in profound metabolic acidosis. MALA typically arises in the setting of renal impairment, overdose, or other hypoperfusive states such as septic or cardiogenic shock.[4]

Clinical features of MALA may include nausea, vomiting, abdominal pain, hemodynamic collapse, and altered mental status. Presentation with complete bilateral blindness, as in this case, is exceedingly rare, although several case reports exist in the literature. While the exact mechanism is unclear, acidosis-induced dysfunction of retinal photoreceptor ion transmission is suspected to play a role in this phenomenon.[5]

The differential diagnosis for MALA includes septic shock, cardiogenic shock, bowel ischemia, DKA, combined MALA-DKA, and acute hepatic failure. Given its nonspecific findings, diagnosis relies on a combination of historical context, lactic acidosis, and elevated serum metformin concentrations, though this laboratory analysis is rarely immediately available to the emergency physician.

The Extracorporeal Treatments in Poisoning (EXTRIP) workgroup has set forth guidelines for the management of MALA.[6] Mild cases may respond to volume resuscitation, correction of underlying causes, vasopressor support, and sodium bicarbonate therapy. Severe MALA—defined by lactate greater than 20 mmol/L, pH less than 7.0, hemodynamic instability, or end-organ dysfunction—requires urgent extracorporeal removal via CRRT or intermittent hemodialysis. Extracorporeal therapy should continue until lactate decreases to less than 3 mmol/L and pH increases to greater than 7.35.

This case highlights the importance of considering MALA in the differential diagnosis for acute visual loss associated with shock and metabolic acidosis in diabetic patients. Early recognition and prompt initiation of renal replacement therapy can be lifesaving, and in this case lead to complete recovery. For emergency physicians, awareness of this rare but critical diagnosis remains essential in identifying at-risk patients and initiating guideline-based management.

FINAL DIAGNOSIS

The final diagnosis is transient blindness from metformin-associated lactic acidosis.

KEY TEACHING POINTS

1. Metformin-associated lactic acidosis can present with chief complaints of neurologic and visual manifestations.
2. In a diabetic patient with volume depletion, AKI, and elevated lactate; MALA should be considered even without an obvious overdose history.
3. A normal noncontrast CT and a nonfocal neurologic exam do not exclude a metabolic diagnosis. Toxic-metabolic etiologies should be considered in patients with abnormal neurologic findings.
4. Early renal replacement therapy is lifesaving in severe MALA. The EXTRIP guidelines recommend extracorporeal treatment for severe toxicity, with continuation until lactate is less than 3 mmol/L and pH is greater than 7.35.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this clinicopathologic case. Documentation on file.

Address for Correspondence: Michael Sibel, DO, University of Maryland, Department of Emergency Medicine, 110 S Paca Street, 6th Floor, Suite 200, Baltimore, MD 21201. Email: Msibel@som.umaryland.edu.

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Medicolegal Pitfalls in Management of Shoulder Dislocations: A Case Series

Clara Billings, OMS-II* *Des Moines University, College of Osteopathic Medicine, Des Moines, Iowa
Abby Bardwell, DO† †Mayo Clinic, Department of Emergency Medicine, Rochester, Minnesota
Aaron Thomas, MD†
Rachel Lindor, MD, JD†

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Introduction: Shoulder dislocations comprise approximately half of all major joint dislocations evaluated in the emergency department. Despite often being considered a simple injury, diagnosing a dislocation can be difficult, especially when accompanied by a fracture associated with unusual mechanism, or when the physical examination is limited. Missed and delayed diagnoses can result in poor patient outcomes and legal risks for clinicians, necessitating a high level of vigilance.

Case Series: We review three medical malpractice cases centered on the failure to recognize shoulder dislocations. Key issues included missed diagnosis, limited physical examination documentation, and inadequate discharge instructions.

Conclusion: Shoulder dislocations can be challenging to identify, and missed diagnoses give rise to subsequent clinical and legal risks. Emergency physicians must maintain a high index of suspicion for this injury, recognize the limitations of standard imaging, and thoroughly document clinical decision-making during both initial evaluation and patient discharge. [Clin Pract Cases Emerg Med. 2026;10(4):440–443.]

INTRODUCTION

Shoulder dislocations constitute nearly half of all major joint dislocations evaluated in the emergency department (ED), with anterior displacement representing up to 98% of these shoulder injuries.[1] The glenohumeral joint is predisposed to instability due to its shallow glenoid fossa, where only about 20% of the humeral head has articular contact with the scapula, as well as a relatively loose joint capsule and ligaments.[1] Patients with shoulder dislocations often present with severe pain in the shoulder and limited range of motion. Patients with anterior dislocations usually hold their arm in abduction and external rotation, while those with posterior dislocations may internally rotate and adduct their arm. The empty glenoid fossa may be palpated behind the humeral head on anterior dislocations, with the opposite finding in posterior dislocations.

Although plain radiographs are often relied on for diagnosis, they have a relatively low sensitivity for associated fractures and posterior dislocations, necessitating thoughtful physical examination and imaging plans. The standard anterior-posterior shoulder radiograph is often insufficient for identifying humeral displacement, especially posterior dislocations, which are missed on most initial ED evaluations.[2] The American College of Sports Medicine and the American Medical Society for Sports Medicine recommend, at a minimum, that true anterior-posterior and axillary views be obtained when imaging for shoulder dislocations.[3] When axillary views are difficult to obtain due to pain or positioning, a scapular Y view or Velpeau view can be useful.[4] Computed tomography or magnetic resonance imaging are increasingly used in clinical practice when radiographs are inconclusive or when there are suspected associated fractures.[3]

Axillary nerve injury in the setting of shoulder dislocations has been reported in up to 60% of cases.[5] This is due to the vulnerability of the nerve in its course around the

surgical neck of the humerus just inferior to the glenohumeral joint. Most injuries result in deltoid muscle weakness and sensory loss over the lateral shoulder. While most of these heal with time, deficits can be persistent, particularly with delayed diagnosis and treatment.[5]

Here we discuss three medical malpractice cases regarding shoulder dislocations, emphasizing common diagnostic oversights and outlining preventive strategies clinicians can employ to reduce the risk of malpractice litigation. The information reported is from actual verdicts and settlements and, thus, is limited to what was provided by the court reporters.

CASE SERIES

Poor Sensitivity of Radiographs

Case 1: *Granger*

A male patient presented to the ED complaining of left shoulder pain. He underwent a radiograph and was informed that his shoulder was not fractured. He was placed in a sling and discharged home. The patient continued to experience pain and limited mobility, and approximately three months later he was diagnosed with a shoulder dislocation, ultimately requiring surgical intervention. He filed a malpractice suit against both the emergency physician and the radiologist, alleging negligence for failure to obtain appropriate radiographs, misinterpretation of the radiograph, and inadequate discharge instructions. The case was settled for $543,000.[6]

Posterior Shoulder Dislocations

Case 2: *Magstadt*

A 45-year-old real estate agent presented to the ED endorsing severe right arm and shoulder pain after a fall. The emergency physician suspected a proximal humerus fracture and ordered radiographs, which were read by the radiologist as negative for fracture. The emergency physician diagnosed the patient with a muscle sprain and discharged him. The patient sought a second opinion due to persistent severe pain and was diagnosed with a posterior shoulder dislocation, requiring surgery and resulting in chronic pain and debility. The patient sued both the radiologist and the initial emergency physician, alleging that he should have examined his shoulder to identify the dislocation, performed range of motion tests to recognize the shoulder joint was affected, diagnosed the dislocation based on the radiographs available, and that he should have provided better follow-up instructions for the patient given his severe, persistent pain at discharge. The case ultimately concluded in favor of the physicians, but it required three years and a full jury trial to resolve.[7]

Concomitant Fracture with Anterior Shoulder Dislocation

Case 3: *Stanard*

A 58-year-old woman presented to the ED with right shoulder pain. The radiologist noted a humerus fracture but did not note the associated dislocation that was also present. The patient developed avascular necrosis, chronic pain, and joint instability, and she sued both the emergency physician and the radiologist for their failure to diagnose her dislocation. The emergency physician defended himself by noting that he relied on the report of the radiologist, which noted only the fracture. Six years after the incident and following a jury trial, the case concluded in favor of the physicians.[8]

DISCUSSION

Diagnosing shoulder dislocations in the ED can be surprisingly challenging. The malpractice cases highlight several common scenarios that cause difficulty for clinicians—namely, poorly controlled pain or concomitant fractures—both of which can complicate the imaging options or exam findings, and the presence of a posterior dislocation, which presents more subtly and requires a higher index of suspicion.

Exam Findings Associated with Joint Dislocations

There are multiple exam findings and mechanisms of injury that should increase the suspicion of a glenohumeral joint dislocation, indicating the need for additional imaging or urgent referral even when optimal imaging is not readily available. For anterior dislocations, falls on an outstretched hand or blunt trauma to an abducted, externally rotated arm are common mechanisms of injury. Patients may present with a physical deformity including noticeable fullness of the axillary region and/or a prominent acromion.[1,3] Posterior dislocations are classically associated with seizures or electrocutions, although the vast majority are secondary to trauma, specifically when the shoulder is internally rotated and forced into flexion and adduction. A patient's physical exam may reveal a posterior prominence, flattening of the anterior shoulder, and an inability to externally rotate the arm.[2]

Appropriate Imaging

The need for plain radiographs with multiple views is imperative during the initial evaluation of all traumatic shoulder injuries. The anterior-posterior radiographic view is useful for evaluating superior-inferior displacement of the humeral head; however, it does not adequately assess anterior-posterior translation. An orthogonal view, such as the axillary view, which requires arm abduction, is essential for accurately identifying shoulder dislocations, particularly in cases of posterior dislocation. Obtaining an axillary view is often difficult due to limited patient cooperation because of significant pain from the dislocation itself, limiting abduction. Consequently, this view is frequently omitted even when explicitly ordered, leading to a potential missed diagnosis.[9] A modified axial projection, such as the Velpeau view, which does not require abduction, can be useful in these situations.[4]

While anterior shoulder dislocations are less often missed due to their characteristic symptoms, signs, and functional limitations, an accurate diagnosis requires maintaining

an appropriate index of suspicion, paired with the proper physical examination and radiographic views.[10] In Case 1, the emergency physician did not suspect a dislocation and relied on plain radiographs to evaluate the patient for a fracture, with angles potentially limited by pain, ultimately missing an anterior dislocation and resulting in a settlement of $543,000—a reflection of the serious complications that can result from delayed diagnosis.[6] Missed dislocations can lead to chronic instability, persistent pain, and irreversible loss of function, which in turn may contribute to decreased earning capacity and long-term disability for the patient. As chronic dislocations progress, the likelihood of successful closed reduction diminishes significantly, thereby increasing the need for operative intervention.[10] This not only elevates the cost of care but also increases patient morbidity and the potential for legal action.

Posterior Dislocations

Unlike anterior dislocations, posterior dislocations can be easily missed due to their subtle clinical signs, less overt radiology findings, and overlapping symptoms with other shoulder injuries, including rotator cuff tears, ligament sprains, and shoulder contusions.[2] In Case 2, the physicians did suspect a shoulder dislocation and obtained radiographs to evaluate.[7] It is unclear what radiographic views were obtained, but the diagnosis of a posterior dislocation was missed. The patient ended up with chronic symptoms, and the physician faced a multi-year lawsuit. It is suggested that posterior dislocations are overlooked up to 80% of the time. This low sensitivity underscores the importance of performing a thorough history and physical exam, which could highlight injury patterns, range of motion, or anatomical nuances suggestive of posterior dislocations, in addition to obtaining appropriate imaging. In patients with the appropriate mechanism of injury and exam findings, clinicians should have a low threshold for computed tomography to further evaluate the shoulder.[11]

Concomitant Fractures

Failing to recognize dislocations alongside significant fractures highlights another risk of shoulder dislocation management. This is illustrated in Case 3, in which the emergency physician discharged a patient with a proximal humerus fracture but failed to recognize his concomitant dislocation, resulting in delayed diagnosis and treatment. The radiological term "satisfaction of search" refers to the tendency of clinicians to stop thoroughly examining imaging after an initial abnormality is found, potentially causing them to miss other coexisting injuries.[9] For example, there may be a mistaken belief that if the patient has a proximal humerus fracture, this injury alone appropriately explains the patient's presentation and symptoms. However, concomitant fracture-dislocations are not uncommon, and finding one should raise suspicions for the other. Clinicians should be cautious not to prematurely conclude the diagnostic process once a single shoulder injury is identified.

LEGAL LESSONS

Regardless of the cause, failure to identify and reduce a shoulder dislocation in a timely manner can result in significant patient morbidity. Chronic unreduced dislocations are associated with a range of complications including degenerative joint changes, disruption of soft tissue stabilizers, osteoarthritis, and avascular necrosis of the humeral head.[2] While the best strategy for risk reduction is certainly to never miss a diagnosis, documentation of thoughtful care may be the second-best step. This starts with full documentation of the patient's history and clinical exam, with special attention to ruling out any neurovascular deficits. In addition, the medical record should reflect that the clinician has considered and evaluated, to the extent they think it reasonable, for the commonly missed injuries such as posterior dislocations and concomitant fractures, when the mechanism calls for it.

Finally, especially in patients who have residual pain, poor function, or other exam limitations at discharge, clear patient education and thorough, written discharge instructions are critical components of risk mitigation. In all cases presented here, plaintiffs cited inadequate post-discharge planning and insufficient follow-up instructions as contributing factors in their malpractice claims.[6-8] Clinicians have a responsibility to ensure patients understand their test results, are aware of signs that should prompt reassessment, and receive clear guidance on symptom management between visits. Spending time to document care and to both discuss and record discharge instructions and follow-up plans can significantly mitigate the risk of future legal claims, even in the face of inevitable clinical uncertainty.

CONCLUSION

Shoulder dislocations are the most common major joint dislocation seen in the ED. Conducting a thorough physical examination and obtaining appropriate, comprehensive imaging are critical steps in minimizing the risk of diagnostic oversight. Our examination of three malpractice cases related to shoulder dislocations illustrates the diagnostic complexities and management pitfalls that can lead to adverse outcomes and legal action. Enhancing clinical awareness of these challenges can support more effective patient care, improve patient outcomes, and help minimize legal exposure.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Rachel Lindor, MD, JD, Mayo Clinic, Department of Emergency Medicine, 200 1st St. SW, Rochester, MN 55905. Email: Lindor.rachel@mayo.edu.

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Transient Electrocardiographic Changes in Anaphylaxis Mimicking Acute Coronary Syndrome: A Case Series

Hari Ganesh S, MD
Amrithanand V T, MD
Kowsthubha B G, MD

Jawaharlal Institute of Postgraduate Medical Education and Research, Department of Emergency Medicine and Trauma, Puducherry, India

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Introduction: Anaphylaxis is a life-threatening emergency that can involve significant cardiovascular manifestations. Transient electrocardiographic (ECG) changes during anaphylaxis and following epinephrine administration may closely mimic acute coronary syndrome, creating a diagnostic dilemma in the emergency department (ED). Failure to recognize these reversible patterns can result in delayed anaphylaxis treatment or unnecessary activation of invasive cardiac pathways.

Case Series: We report a case series of three adult patients presenting to the ED with anaphylaxis who developed alarming ECG changes, including ST-segment elevation, ST-segment depression, and hyperacute T waves. One patient demonstrated transient ECG changes temporally related to intramuscular epinephrine administration, while two patients exhibited features suggestive of hypersensitivity-associated coronary vasospasm or thrombosis manifesting as ECG changes. In all cases, serial ECGs, high-sensitivity cardiac troponin testing, and focused echocardiography revealed resolution of ECG abnormalities without dynamic biomarker elevation or regional wall motion abnormalities. All patients improved with prompt anaphylaxis-directed therapy and were managed conservatively without coronary intervention.

Conclusion: Transient ECG changes during anaphylaxis and after epinephrine administration can closely resemble acute coronary syndrome but may resolve completely with appropriate allergy-directed treatment. Serial ECG assessment, cardiac biomarkers, and focused echocardiography are essential tools for emergency physicians to differentiate transient allergic or catecholamine-mediated effects from true myocardial ischemia. Recognition of these patterns allows timely treatment of anaphylaxis while avoiding unnecessary invasive cardiac interventions. [Clin Pract Cases Emerg Med. 2026;10(4):444–449.]

Keywords: *anaphylaxis; electrocardiography; acute coronary syndrome mimic; epinephrine; Kounis syndrome.*

INTRODUCTION

Anaphylaxis is a severe, life-threatening hypersensitivity reaction characterized by rapid involvement of multiple organ systems, most notably the respiratory and cardiovascular systems. Cardiovascular manifestations are a major cause of morbidity and mortality in anaphylaxis and include hypotension, distributive shock, and cardiac dysfunction resulting from the systemic release of inflammatory mediators. Experimental and clinical evidence suggests that the heart, particularly the coronary circulation, functions as a primary target organ during anaphylaxis, making cardiac involvement both common and clinically significant.[1]

Electrocardiographic (ECG) abnormalities during anaphylaxis are increasingly recognized but remain underreported in routine clinical practice. These changes may include ST-segment elevation or depression, hyperacute T

waves, arrhythmias, and conduction disturbances.[2] Such findings can closely resemble acute coronary syndrome (ACS), especially when accompanied by chest pain, diaphoresis, hypotension, or syncope. The overlap between anaphylaxis and ACS presentations creates a diagnostic challenge in the emergency department (ED), where rapid decision-making is essential and misclassification may lead to either harmful delays in anaphylaxis treatment or unnecessary invasive cardiac interventions.[3]

Multiple mechanisms have been proposed to explain ECG changes in anaphylaxis, including coronary hypoperfusion due to systemic vasodilation and plasma extravasation, direct myocardial depression mediated by inflammatory cytokines, coronary vasospasm, and platelet activation.[4] In some patients, these processes manifest as Kounis syndrome, an allergic ACS characterized by coronary vasospasm, plaque rupture, or stent thrombosis triggered by hypersensitivity reactions.[5-7] Epinephrine, while life-saving in anaphylaxis, exerts complex cardiovascular effects through its α- and β-adrenergic receptor activity. Stimulation of β1-adrenergic receptors increases heart rate, myocardial contractility, and conduction velocity, collectively raising cardiac workload and myocardial oxygen demand. Simultaneously, α-adrenergic-mediated coronary vasoconstriction reduces myocardial oxygen supply, creating a supply-demand mismatch that may precipitate transient subendocardial ischemia, particularly in susceptible individuals. Additional prothrombotic effects, including enhanced platelet aggregation and thromboxane B2 production, may further compromise coronary microcirculatory flow. Together, these mechanisms can produce transient ECG changes—ranging from ST-segment depression and T wave abnormalities to arrhythmias—that may mimic ACS but typically resolve with hemodynamic stabilization and do not reflect irreversible myocardial injury.[8-10]

More recently, these overlapping entities have been conceptualized as part of the ATAK complex—adrenaline, takotsubo cardiomyopathy, anaphylaxis, and Kounis syndrome—highlighting the intricate interplay between allergic reactions, catecholamine exposure, and cardiac dysfunction.[11] Distinguishing among these mechanisms in the acute setting remains challenging, particularly because many anaphylaxis-related ECG changes are transient and resolve with appropriate treatment, often in the absence of significant troponin elevation or echocardiographic abnormalities.

Given the potential for diagnostic confusion and management error, recognition of transient ECG changes associated with anaphylaxis and its treatment is critical for emergency physicians. We present a case series of three patients with anaphylaxis who developed reversible ECG changes during their ED course, illustrating key clinical features that aid differentiation from true ACS and emphasizing the importance of continuous cardiac monitoring and temporal ECG assessment during anaphylaxis management.

CPC-EM Capsule

What do we already know about this clinical entity?
Anaphylaxis can rarely cause transient echocardiogram (ECG) abnormalities and mimic acute coronary syndrome (ACS) through allergic cardiac involvement.

What makes this presentation of disease reportable?
This case series demonstrates dynamic ECG changes during anaphylaxis that closely resembled ACS but resolved after treatment.

What is the major learning point?
Transient ECG abnormalities during anaphylaxis should be interpreted with clinical context before initiating ACS-specific interventions.

How might this improve emergency medicine practice?
Early recognition of anaphylaxis-related ECG changes may prevent misdiagnosis and ensure prompt adrenaline-centered treatment.

CASE SERIES

Case 1: Epinephrine-Related Transient ECG Changes

ED Presentation: A 62-year-old man with no known prior comorbidities presented to the ED with sudden onset generalized pruritus, urticarial wheals, profuse diaphoresis, and left periorbital edema shortly after food intake and ingestion of oral analgesics earlier in the day. He had no chest pain. On arrival, he was diaphoretic and dyspneic, unable to complete sentences, with a weak pulse and unrecordable blood pressure, and oxygen saturation consistent with anaphylactic shock involving mucocutaneous, respiratory and cardiovascular systems. Physical examination was notable for left periorbital edema; cardiovascular, respiratory, and abdominal examinations were otherwise unremarkable. The initial 12-lead ECG demonstrated normal sinus rhythm at 72 beats per minute with a normal axis and no acute ST–T wave abnormalities (Image 1A). Presumed anaphylaxis was treated immediately with intramuscular epinephrine 0.5 mg (1:1,000) administered into the anterolateral thigh, along with intravenous (IV) crystalloids, IV chlorpheniramine 10 mg, and IV hydrocortisone 200 mg with continuous cardiac monitoring was initiated.

Shortly after epinephrine administration, cardiac

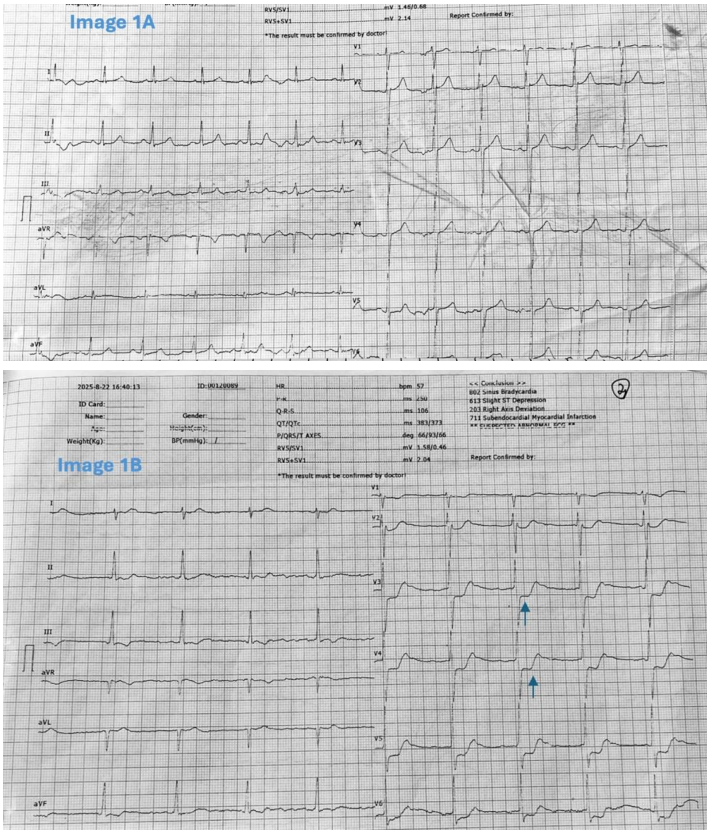


Image 1. A) The initial 12-lead electrocardiogram (ECG) demonstrated normal sinus rhythm at 72 beats per minute, normal axis, and no acute ST-segment or T wave abnormalities. B) A repeat ECG showed sinus bradycardia at 52 beats per minute with right axis deviation and diffuse ST-segment depression in leads V1–V6. Blue arrow pointing ST-segment depression in lead V3 and V4.

monitoring demonstrated new ischemic changes. A repeat ECG showed sinus bradycardia at 52 beats per minute with right axis deviation and diffuse ST-segment depression in leads V1–V6, without posterior or right ventricular lead involvement (Image 1B). As blood pressure improved, a subsequent ECG obtained 30 minutes later demonstrated resolution of ST changes with sinus rhythm at 66 beats per minute and an RSR′ pattern in V1–V2 (Image 1C).

High-sensitivity troponin I was borderline elevated at 19.98 ng/L (reference range, <13 ng/L) without dynamic rise on repeat testing. Transthoracic echocardiography revealed normal left ventricular systolic function with no regional wall motion abnormalities.

After cardiology consultation, the patient was managed conservatively without antiplatelet therapy, anticoagulation, or coronary intervention. Electrocardiogram changes and symptoms resolved completely, and the findings were attributed to transient epinephrine-related myocardial effect in the setting of anaphylaxis.

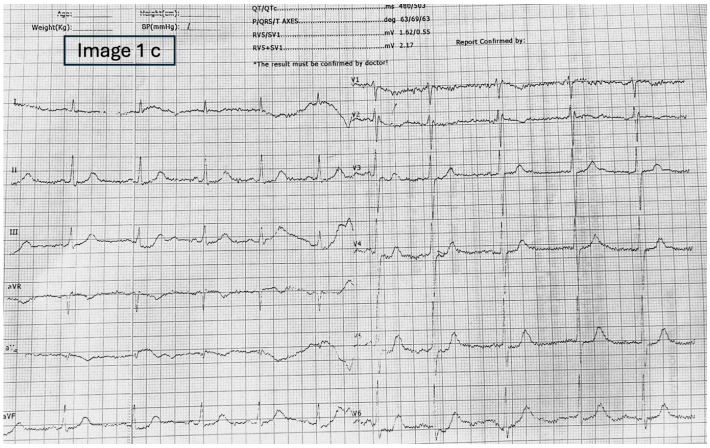


Image 1C. Repeat 12-lead electrocardiogram obtained 30 minutes after epinephrine administration demonstrating resolution of the previously noted ST-segment depression, with restoration of sinus rhythm at 66 beats per minute.

Case 2: Transient ECG Changes Consistent with Kounis Syndrome

A 34-year-old man with no known prior cardiac history presented to the ED with acute-onset left-sided chest pain, generalized pruritus, and diffuse erythematous rash. Symptoms developed within hours of re-exposure to oral amoxicillin–clavulanate prescribed for dental pain. Chest pain was associated with diaphoresis, nausea, and vomiting. On arrival, he was hemodynamically stable with a heart rate of 100 beats per minute, blood pressure of 110/60 mm Hg, and oxygen saturation of 98% on room air. Physical examination revealed a diffuse erythematous rash; chest auscultation was clear with no added sounds, cardiac auscultation was normal, and the abdomen was soft and nontender. The presenting 12-lead ECG demonstrated sinus tachycardia at 100 beats per minute with ST-segment elevation in leads II, III, aVF, and V1–V6, ST depression in leads I and aVL, and hyperacute T waves, including right-sided precordial leads (Image 2A). Given clinical features consistent with anaphylaxis and concurrent ischemic-appearing ECG changes, the patient was treated with intramuscular epinephrine 0.5 mg, IV chlorpheniramine 10 mg, IV hydrocortisone 200 mg, and continuous cardiac monitoring was initiated.

Following clinical stabilization, a repeat ECG demonstrated normalization of ST-segment and T wave morphology (Image 2B). High-sensitivity troponin I measured 7 ng/L, with no subsequent rise on serial testing. Transthoracic echocardiography showed normal biventricular function without regional wall motion abnormalities. In consultation with cardiology, conservative management was continued, as ECG changes were transient and biomarkers remained negative. The presentation was considered consistent with type I Kounis syndrome, with resolution following treatment of the allergic reaction.

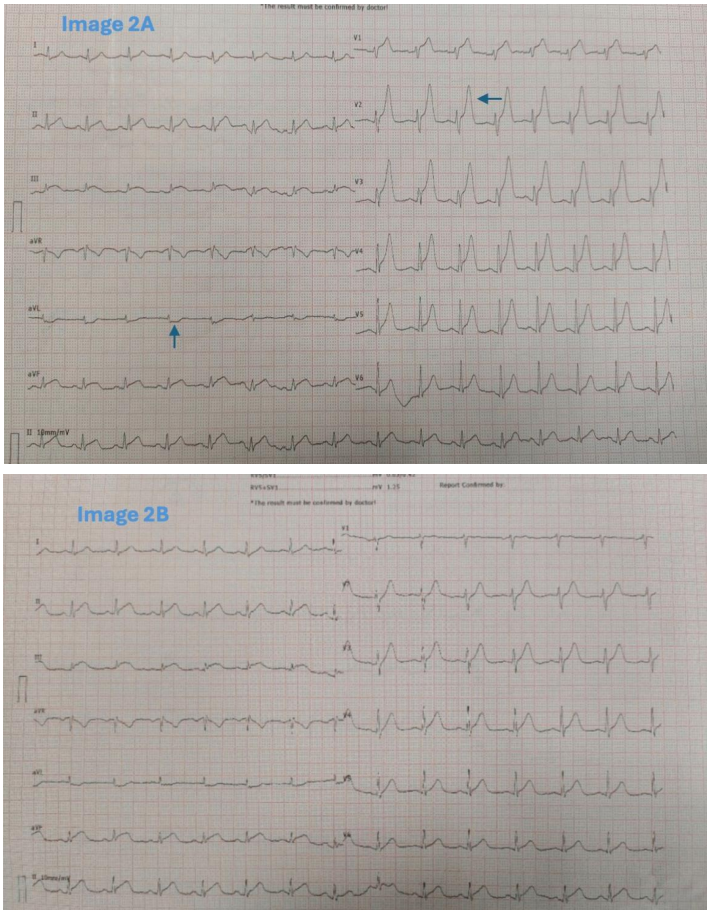


Image 2. A) The presenting 12-lead electrocardiogram demonstrated sinus tachycardia at 100 beats per minute with ST-segment elevation in leads II, III, aVF, and V1–V6, ST-segment depression in leads I and aVL, and hyperacute T waves, including right-sided precordial leads. Blue arrow pointing to hyperacute T wave and ST- segment depression in lead aVL. B) Repeat ECG obtained after stabilization showed normalization of T wave morphology and normalization of ST-segment depression.

Case 3: Anaphylaxis-Associated Transient Ischemic ECG Changes

A 40-year-old man with a recent history of insect bite was brought to the ED with profuse diaphoresis and generalized pruritis without associated chest pain. On arrival, patient was hemodynamically unstable with an unrecordable blood pressure and a heart rate of 109 beats per minute and an unrecordable oxygen saturation in the setting of severe hypotension, consistent with anaphylactic shock involving the cutaneous and cardiovascular systems. Physical examination was notable for generalized urticaria; respiratory and abdominal examinations were unremarkable. The initial 12 lead ECG demonstrated diffuse ST-segment depression with hyperacute T waves in leads V2–V3 (Image 3A).

Immediate resuscitation was initiated with intramuscular epinephrine 0.5 mg (1:1,000) administered to the anterolateral thigh, along with IV fluids bolus of 500 mL followed by maintenance infusion at 150 mL/hour, IV chlorpheniramine 10 mg, and IV hydrocortisone 200 mg, with continuous ECG monitoring maintained throughout. A repeat ECG obtained approximately 30 minutes after clinical stabilization demonstrated complete resolution of hyperacute T waves and ST-segment changes (Image 3B).

Cardiac biomarkers were not elevated, and bedside echocardiography revealed preserved biventricular function without regional wall motion abnormalities. Cardiology was consulted, and in the absence of chest pain, dynamic biomarker rise, or echocardiographic abnormality, conservative management was recommended. The patient's hemodynamics stabilized with resolution of symptoms and ECG abnormalities. He was discharged after observation for six hours with a diagnosis of transient anaphylaxis-related ECG changes, without evidence of ACS.

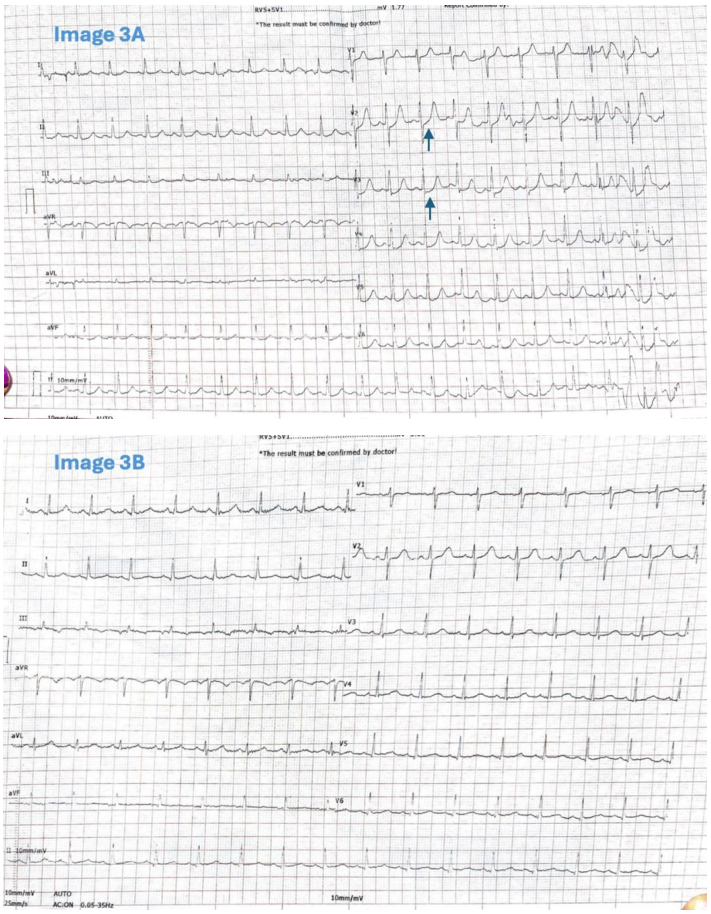


Image 3. A) The initial 12-lead electrocardiogram demonstrated diffuse ST-segment depression with hyperacute T waves in leads V2–V3. Blue arrow pointing to ST depression with hyperacute T waves. B) A repeat electrocardiogram obtained approximately 30 minutes after clinical stabilization demonstrated complete resolution of hyperacute T waves and ST-segment changes.

DISCUSSION

Electrocardiographic abnormalities during anaphylaxis are increasingly recognized yet remain diagnostically challenging in the ED because they can closely mimic ACS. These changes are often transient and may arise from multiple overlapping mechanisms, including systemic vasodilation with coronary hypoperfusion, direct myocardial depression mediated by inflammatory mediators, coronary vasospasm, and the effects of exogenously administered epinephrine.[1,5] In the acute setting, distinguishing among these mechanisms is critical, as misinterpretation may result in delayed treatment of anaphylaxis or unnecessary activation of invasive cardiac pathways.

In anaphylaxis, cardiovascular collapse is primarily driven by systemic vasodilation, increased vascular permeability, and intravascular volume depletion, leading to reduced preload and impaired coronary perfusion. Experimental and clinical data support the concept that the heart—particularly the coronary circulation—is a direct target of anaphylactic mediators, resulting in global myocardial dysfunction rather than focal ischemia.[1,12] This pathophysiology explains why many patients exhibit transient ischemic-appearing ECG changes with normal or minimally elevated cardiac biomarkers and absence of regional wall motion abnormalities on echocardiography, as observed in our cases.

Kounis syndrome represents a distinct but related entity in which allergic mediator release precipitates coronary vasospasm, plaque rupture, or stent thrombosis, leading to true myocardial ischemia or infarction.[5-7] In contrast to transient anaphylaxis-related ECG changes, Kounis syndrome is more likely to demonstrate persistent ECG abnormalities, dynamic troponin elevation, and regional wall motion abnormalities, reflecting myocardial injury. In our series, only one patient presented with chest pain and ECG findings consistent with Type I Kounis syndrome, yet even in this case, ECG changes resolved rapidly and cardiac biomarkers remained within normal limits, supporting conservative management.

Epinephrine, the cornerstone of anaphylaxis treatment, introduces additional diagnostic complexity. Through its α- and β-adrenergic effects, epinephrine can induce coronary vasoconstriction, increase myocardial oxygen demand, and promote platelet aggregation, resulting in transient ischemic ECG changes or, rarely, myocardial infarction.[8-10] These effects are typically short-lived when epinephrine is administered intramuscularly at recommended doses, as demonstrated in Case 1, where ECG changes appeared shortly after administration and resolved as the pharmacologic effect diminished. Recognition of this temporal relationship is essential to avoid inappropriate escalation to conventional ACS pathways.

From an ED perspective, this case series highlights the importance of serial assessment rather than isolated ECG interpretation. In selected patients with anaphylaxis who demonstrate transient ECG changes, a combination of serial ECGs, high-sensitivity troponin measurements, and focused echocardiography may be sufficient to exclude ongoing myocardial ischemia. Resolution of ECG abnormalities, absence of dynamic troponin rise, and normal ventricular function strongly favor transient anaphylaxis- or epinephrine-related effects over ACS. This approach allows emergency physicians to prioritize life-saving anaphylaxis treatment while safely deferring invasive cardiac evaluation.

Importantly, not all patients with ECG changes during anaphylaxis are appropriate for conservative management. Immediate cardiology involvement and consideration of catheterization are warranted when ECG changes persist despite resolution of anaphylaxis, when there is ongoing chest pain, dynamic elevation of cardiac biomarkers, new regional wall motion abnormalities, or hemodynamic instability unexplained by distributive shock. In such cases, Kounis syndrome or concurrent ACS must be strongly considered.[5,7,13]

These overlapping entities—anaphylaxis, epinephrine-induced myocardial effects, Kounis syndrome, and stress-induced cardiomyopathy—have been conceptualized as the ATAK complex, underscoring the interconnected nature of allergic and catecholamine-mediated cardiac pathology.[11] Awareness of this spectrum is essential for emergency physicians, as management decisions must be individualized and guided by clinical trajectory rather than a single diagnostic test. The flowchart on approach is summarized in the figure.

CONCLUSION

Anaphylaxis and its treatment with intramuscular epinephrine can produce transient electrocardiographic changes that closely mimic acute coronary syndrome, creating a high-risk diagnostic scenario in the emergency department. Recognition of the temporal resolution of ECG abnormalities, in conjunction with serial electrocardiography, high-sensitivity troponin testing, and focused echocardiography, allows emergency physicians to safely distinguish transient anaphylaxis- or catecholamine-mediated effects from true myocardial ischemia in selected patients. Importantly, avoidance of reflexive activation of invasive cardiac pathways should not delay definitive anaphylaxis treatment, while early cardiology involvement remains essential when ECG changes persist, biomarkers rise dynamically, or regional wall motion abnormalities are present, raising concern for Kounis syndrome or concurrent acute coronary syndrome. Through vigilant monitoring and trajectory-based decision-making, emergency physicians can prevent both undertreatment of anaphylaxis and unnecessary cardiac interventions.

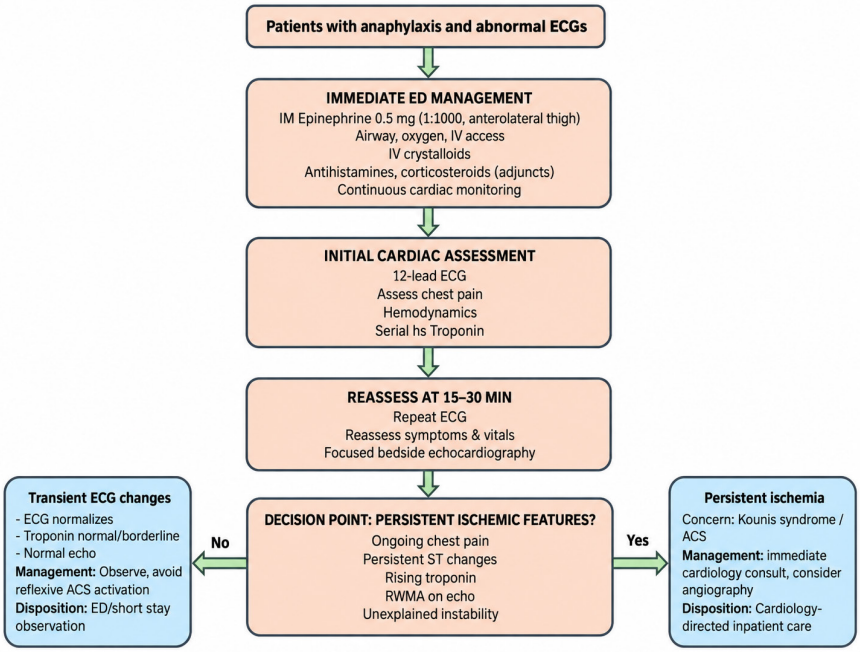


Figure. Emergency department flowchart: approach to anaphylaxis with abnormal ECG.
ACS, acute coronary syndrome; *ECG*, electrocardiogram; *ECHO*, echocardiography; *ED*, emergency department; *hs,* high sensitivity; *IM*, intramuscular; *IV*, intravenous; *min*, minutes; *RWMA*, regional wall motion abnormality.

The authors attest that their institution does not require Institutional Review Board approval for publication of this case report. Patient consent has been obtained and filed for the publication of this case report. Documentation on file.

Address for Correspondence: Amrithanand V T, MD, Assistant Professor, Department of Emergency Medicine and Trauma, Ground Floor, Emergency Medical Services Block, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Dhanvantari Nagar, Puducherry – 605006 India. Email: amrithanandvt@gmail.com.

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Observation Unit Management of Older Adults with Rib Fractures: A Case Series

Phillip D Jenkins, MD*
Elizabeth K Crawford Rowe, DMSc, PA-C†
Colin Gershon, NP, MPH†
Mackenzie R Cook, MD*
John Marshall, MD, MBA†

*Oregon Health & Science University, Department of Surgery, Portland, Oregon
†Oregon Health & Science University, Department of Emergency Medicine, Portland, Oregon

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Introduction: Rib fractures in older adults carry high morbidity and mortality, leading many hospitals to admit patients based on age alone. The Rib Injury Guidelines (RIG) offer a triage system; under RIG 2, patients are typically admitted to inpatient care.

Case Series: This study evaluated whether older adults categorized as RIG 2 solely due to age could be safely managed in an emergency department observation unit. We conducted a 14-month case series at an academic trauma center, reviewing 23 patients 60 years and older with rib fractures. Twenty met RIG 2 criteria, including those later admitted for nontrauma reasons. The mean observation stay was 26.6 hours, and the total hospital stay was 1.8 days. Seventeen patients (85%) were discharged directly from observation, three (15%) were admitted to medicine, and none returned within 72 hours.

Conclusion: Carefully selected older adults with rib fractures may be safely managed in observation units, optimizing resource utilization and patient safety. [Clin Pract Cases Emerg Med. 2026;10(4):450–454.]

Keywords: *rib fractures; observation units; quality improvement; hospital resource utilization; older adults.*

INTRODUCTION

Rib fractures are common thoracic injuries in older adults and are associated with significant morbidity and mortality.[1,2] Compared with younger patients, older adults suffer disproportionately from rib fracture complications, including pneumonia, respiratory failure, and death.[1] Each additional rib fractured in a geriatric patient increases pneumonia risk by approximately 27% and mortality by nearly 20%.[1,2] After adjusting for comorbidities and injury severity, trauma patients 65 years of age and older have five-fold higher odds of death from chest injuries compared with younger counterparts.[3] These risks make accurate triage and timely management a priority for improving quality and outcomes in this vulnerable population. Traditional risk stratification, using age and the number of rib fractures, was based on the 2001 Bulger study. As imaging has improved over the past quarter-century, it has become clear that risk stratification that accounts for pain/respiratory function could lead to more appropriate resource use.[1]

Although traditional teaching has favored intensive care unit (ICU) admission for elderly rib fracture patients, structured management protocols now guide disposition decisions, helping clinicians identify which patients are safe for discharge and which need only a standard ward bed.[1] The Rib Injury Guidelines (RIG) provide a standardized approach for triage based on age, number of rib fractures, comorbidities, pain control, pulmonary function, and other risk factors.[4] (Table 1). Patients meeting RIG 1 criteria are generally safe for discharge, RIG 2 patients typically require

inpatient admission, and RIG 3 patients are recommended for ICU-level monitoring. In a prospective validation study, RIG implementation reduced unnecessary ICU admissions and shortened length of stay without compromising safety.[5] Other rib fracture protocols have similarly demonstrated improved efficiency and outcomes when applied to older trauma patients.[6]

Despite these advances, practice variation remains substantial. In a study of nearly 24,000 patients 65 years of age and older with isolated rib fractures, ICU admission rates varied from 0% to 92% across trauma centers, with higher utilization centers demonstrating lower pneumonia and complication rates.[7] Such findings highlight the tension between aggressive monitoring and resource stewardship. While guidelines recommend admission for many older adults, some patients, particularly those whose only RIG 2 criterion is age, may not require a full hospital admission if appropriately monitored.

Emergency department observation units (EDOU) represent a significant opportunity for optimizing the management of blunt chest trauma patients, often reducing hospital admissions and length of stay (LOS) without compromising safety.[6] Prior studies affirm that carefully selected patients can be safely managed in observation settings, leading to positive clinical and financial outcomes and RIG protocol provide us a validated structure to carefully select those patients.[7] The application of EDOUs aligns with previous work estimating a national savings of approximately $3.1 billion annually,[8] underlining the financial benefit of using observation units for short-stay patients, such as those with less severe blunt chest trauma who do not require extensive inpatient care.

Moreover, studies specifically addressing trauma patients further validate the efficacy of observation units. One study highlighted that blunt chest trauma constitutes a significant portion of trauma presentations in EDs, and identifying which patients require intensive monitoring can be challenging.[9] This analysis suggests that certain subsets of these patients can be effectively managed on a less intensive basis, which resonates with the operational framework of EDOUs. By implementing a selective observation strategy, hospitals may observe a decrease in LOS and a likely reduction in related complications such as hospital-acquired pneumonia, as noted in research linking inadequate pain control with extended admissions in chest trauma cases.[10]

Our study evaluates a targeted application of RIG 2 triage to older adults whose only qualifying factor was age. We present a case series of patients managed in an EDOU rather than admitted to the hospital, to assess feasibility, safety, and resource use. By integrating guideline-based triage into observation unit practice, this work provides proof of concept for a novel approach to rib fracture management and a foundation for future studies.

CPC-EM Capsule

What do we already know about this clinical entity?
Rib fractures in older adults carry high morbidity. Rib Injury Guidelines (RIG) 2 patients are typically admitted to an inpatient ward, often based on age alone.

What makes this presentation of disease reportable?
This is the first report of managing age-only RIG 2 patients in an emergency department observation unit (EDOU), demonstrating shorter stays without 72-hour returns.

What is the major learning point?
Older adults meeting RIG 2 criteria solely due to age may be safely managed in an EDOU, reducing unnecessary inpatient admissions.

How might this improve emergency medicine practice?
Integrating guideline-based triage with EDOU pathways can reduce inpatient admissions, lower costs, and preserve safety for selected older rib fracture patients.

METHODS

Study Design and Setting

This was a case series conducted at an academic Level I trauma center over a 14-month period from February 2023 to April 2024. The trauma and ED teams jointly decided to incorporate the RIG scoring tool into hospital policy to help guide the disposition of patients with rib fractures. Following this initial implementation, a second plan-do-study-act cycle was conducted in early 2024 to evaluate outcomes across various patient groups with rib fractures. This review identified well-appearing older patients categorized as RIG 2 only due to their age as a potentially safe cohort for EDOU admission, ultimately leading to the 14-month trial on which this paper is based. As a result of this second plan-do-study-act cycle, modified RIG protocol was implemented to guide disposition decisions for older adults with rib fractures. Under this protocol, patients were managed in the EDOU when their clinical condition permitted, rather than being routinely admitted to the hospital. At our institution, "triage" in this context refers to the disposition decision made by the trauma and emergency medicine teams after initial ED evaluation, not

Table 1. Rib Injury Guidelines (RIG) classification system used in a study that evaluated whether older adults categorized as RIG 2 solely due to age could be safely managed in an observation unit.

Category*	Recommended disposition	Representative criteria
RIG 1 (low risk)	Discharge home	Younger age, few rib fractures, adequate pain control, preserved pulmonary function and cough, no significant comorbidities, and no high-risk imaging features
RIG 2 (moderate risk)	Inpatient ward admission	Older age (≥ 60 years), moderate number of rib fractures, mild-to-moderate comorbidities, pain partially controlled, mildly impaired pulmonary function or cough, or other intermediate risk features not meeting RIG 3 criteria
RIG 3 (high risk)	ICU-level monitoring	Multiple rib fractures, bilateral fractures, flail chest, pulmonary contusion, significant comorbidities (eg, COPD), poorly controlled pain, impaired cough, or markedly reduced inspiratory spirometry

*Categories are adapted from Nelson et al.[4]
COPD, chronic obstructive pulmonary disease; *ICU*, intensive care unit; *RIG*, Rib Injury Guidelines.

the conventional acuity-based triage that occurs at ED arrival. After workup, eligible patients were assigned to EDOU-level care under the modified RIG pathway. A subset of these patients subsequently required inpatient admission to the hospital medicine service for nontrauma medical reasons (eg, management of comorbid conditions identified during observation); these patients were retained in the analytic cohort because they met all inclusion criteria at the time of EDOU assignment.

Inclusion and Exclusion Criteria

All patients 60 years of age and older with rib fractures who were triaged to the EDOU during the 14-month study period were eligible for review. Patients were included in the analytic cohort if they were managed initially in the EDOU and had a reviewer-calculated RIG score of 4 points, with points for age 60 and older only. Patients who were admitted to an inpatient service after an initial EDOU stay for nontrauma medical reasons were retained in the analytic cohort, as they met all inclusion criteria at the time of triage.

Patients were excluded if they were not actually managed in the EDOU during the index visit. Specifically, two patients were excluded because they were admitted directly to a hospital-based observation unit rather than the EDOU, and one patient was excluded because their rib fractures occurred during a different encounter outside the study window.

Data Collection and Scoring

Electronic health records were reviewed. One author retrospectively reviewed RIG scores based on available chart data, including demographics, comorbidities, injury details, and hospital course. Emergency department observation unit LOS, total hospital LOS, Injury Severity Score (ISS), comorbidities, and 72-hour return visits were extracted for analysis.

Analysis

We used descriptive statistics to summarize patient demographics, injury characteristics, LOS, comorbidities, and return visits. Continuous variables are reported as mean (standard deviation) and median with interquartile range (IQR). Categorical variables are reported as counts and percentages.

Ethics

This study was reviewed and approved by the institutional review board (STUDY00027037).

Statistical Software

All analyses were performed in Python version 3.12.4 (Python Software Foundation,). Data management and descriptive statistics were conducted using pandas, NumPy, and SciPy. Tables were prepared with python-docx.

RESULTS

A total of 23 patients 60 years of age and older with rib fractures were triaged to the EDOU during the study period. Three patients were excluded from analysis: two were admitted directly to hospital-based observation units rather than the EDOU; and one had rib fractures at a separate encounter outside the study window. The final analytic cohort included 20 patients managed initially in the EDOU.

Analytic Cohort (n = 20)

Patients had a mean age of 73.7 years (SD 9.2, median 71.5, IQR 65.5–83.0) and a mean ISS of 9.2 (SD 3.6, median 10, IQR 7.3–10.3). Observation unit LOS averaged 26.6 hours (SD 15.4, median 21.1, IQR 14.6–41.0). Total hospital LOS averaged 1.8 days (SD 1.2, median 1.3, IQR 0.9–2.2). No patients returned to the ED within 72 hours. Comorbid conditions were common, present in 18 patients (90%). A summary of patient characteristics is provided in Table 2.

DISCUSSION

This case series demonstrates that a structured rib fracture triage protocol may be adapted to safely manage selected

Table 2. Characteristics of patients ≥ 60 years triaged to emergency department observation with rib fractures.

Variable	RIG 2 (0 if not for age) (n = 20)
Age (years)	73.7 (9.2) (Median 71.5, IQR 65.5–83.0)
ISS	9.2 (3.6) (Median 10, IQR 7.3–10.3)
EDOU LOS (hours)	26.6 (15.4) (Median 21.1, IQR 14.6–41.0)
Total LOS (days)	1.8 (1.2) (Median 1.3, IQR 0.9–2.2)
Return ≤ 72 hours	0 (0%)
Comorbidities	18 (90%)
Admitted from EDOU for Trauma	0 (0%)

EDOU, emergency department observation unit; *IQR*, interquartile range; *ISS*, Injury Severity Score; *LOS*, length of stay; *RIG*, Rib Injury Guidelines.

older adults in an EDOU. Specifically, patients whose RIG 2 categorization was based solely on age were observed for short periods, discharged without subsequent admissions, and had no 72-hour return visits. This is a novel approach not described in the original RIG studies and is being implemented through a thoughtful, rigorous plan-do-study-act cycle. By aligning guideline-based triage with observation unit resources, this approach reduced inpatient admissions while preserving patient safety.

Historically, older adults with rib fractures have been admitted reflexively due to age-associated risk, despite heterogeneity in their actual clinical needs. By separating age-only RIG 2 patients from those with additional risk factors, hospitals may reduce unnecessary admissions while still identifying patients who require higher. intensity monitoring. The result is a more precise allocation of hospital resources and a care model that balances safety with stewardship.

From a cost perspective, we know that the elderly population in the United States is growing significantly, such that recent estimates place one in five individuals being 65 years or older by 2030, and contributes significantly to rising inpatient trauma costs.[11] Review of Centers for Medicare & Medicaid Services data review shows that in patients admitted for similar diagnoses, short inpatient stays cost approximately three times that of observation stays (~US $5,142 for a short inpatient stay versus ~US $1,741 for an observation stay).[12] The potential to manage appropriately risk-stratified patients in an EDOU could significantly decrease the cost of appropriate care in this population.

Our results also highlight the importance of differentiating rib fracture–related care needs from nontrauma medical drivers of admission. Patients excluded from the analytic cohort were hospitalized for comorbid or unrelated medical conditions, underscoring that outcomes depend not only on injury severity but also on broader clinical context. This distinction is critical for hospitals seeking to reduce LOS, prevent avoidable admissions, and ensure comprehensive care for medically complex older adults.

LIMITATIONS

This study is limited by its small sample size and single-center design, which may affect generalizability. The absence of a direct inpatient comparator group limits conclusions about relative outcomes. Finally, observation units vary in availability and structure across healthcare systems, which may influence the feasibility of adopting this approach in other institutions.

CONCLUSION

A modification of the Rib Injury Guidelines demonstrated that older adults with rib fractures categorized as RIG 2 based on age alone may be safely managed in an EDOU setting. This strategy reduced inpatient admissions and overall length of stay without compromising safety. Larger multicenter studies with comparator groups are needed to confirm these findings, refine selection criteria for age-only RIG 2 patients, and evaluate downstream outcomes such as readmission, functional recovery, and cost. This case series provides a foundation for those investigations and supports continued protocol development as observation unit pathways for older adults with rib fractures mature.

The Institutional Review Board approval has been documented and filed for publication of this case series.

Address for Correspondence: Phillip D Jenkins, MD, Oregon Health & Science University, Department of Surgery, 3181 Sam Jackson Park Rd, Portland, OR 97239. Email: jenkinph@ohsu.edu.

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Bougie-Assisted Blakemore Tube Insertion: A Case Series

Shane Gardner, DO[*†]
Andrew Tachney, MD[*]
Adele Cobbs, MD[*]
Margaret Putman, DO[‡]
Travis Hase, MD[*§¶]

[*]Advocate Christ Medical Center, Department of Emergency Medicine, Oak Lawn, Illinois
[†]Northwestern University Feinberg School of Medicine, Department of Anesthesiology, Chicago, Illinois
[‡]The University of Chicago Pritzker School of Medicine, Department of Medicine, Section of Geriatrics and Palliative Medicine, Chicago, Illinois
[§]University of Illinois College of Medicine at Chicago, Department of Emergency Medicine, Chicago, Illinois
[¶]Wake Forest University School of Medicine, Department of Emergency Medicine, Winston-Salem, North Carolina

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Introduction: Variceal hemorrhage is a life-threatening complication of portal hypertension that requires rapid hemorrhage control. Gastroesophageal balloon tamponade devices are effective temporizing measures when endoscopic or definitive therapies are delayed or unsuccessful, but placement can be technically challenging during active bleeding. A bougie-assisted technique has been proposed to improve ease and success of placement.

Case Series: We describe three patients with cirrhosis and varices who presented with massive upper gastrointestinal bleeding and hemorrhagic shock. All received standard medical therapy and required escalation due to ongoing bleeding. In each case, a Sengstaken-Blakemore tube was placed using a bougie tracheal tube introducer as a semirigid stylet. The device was advanced under laryngoscopy through the esophagus and into the stomach without procedural complications. Hemostasis was achieved in all cases following balloon inflation, allowing hemodynamic stabilization and transition to definitive therapies. Two patients ultimately died due to complications of hypoxic-ischemic injury, while one survived to discharge. Use of the proximal gastric aspiration port appeared to reduce the risk of tip displacement compared to distal port insertion.

Conclusion: Bougie-assisted gastroesophageal balloon tamponade device placement is a practical and effective modification for managing catastrophic upper gastrointestinal hemorrhage, and this approach may improve placement success and speed when standard technique has been unsuccessful. Wider adoption of this readily available adjunct could enhance procedural reliability, although further study is needed to better define outcomes and optimal technique. [Clin Pract Cases Emerg Med. 2026;10(4):455–460.]

Keywords: *Blakemore; bougie; gastrointestinal hemorrhage; varices; case series.*

INTRODUCTION

Variceal hemorrhage is a life-threatening complication of portal hypertension, most commonly occurring in patients with advanced cirrhosis.[1] Rapid control of bleeding is essential, as mortality remains high despite advances in medical and endoscopic therapies.[2] When definitive intervention is delayed, unavailable, or unsuccessful, temporizing measures such as a gastroesophageal balloon tamponade may be lifesaving.[3–5]

Successful placement of gastroesophageal balloon tamponade devices, such as the Sengstaken–Blakemore tube, is technically challenging, particularly in the setting of

ongoing hemorrhage and poor visualization. Specifically, the length and flexibility of the Blakemore contributes to a loss of directional control of its tip, commonly resulting in coiling and a failure to advance. Using a freezer or ice water bath may help to stiffen the tubing; however, this is time-consuming, and the effect is transient. Magill forceps may be used to grasp the tube and inch it forward, but this risks damage to the balloon and may injure surrounding tissues and does not mitigate the tendency for coiling.[4] Additionally, several modified techniques using various guidewires have been described, but these approaches have not substantially improved ease or reliability of placement and may require unfamiliar or unavailable equipment.[6–8] Bougie tracheal tube introducers are ubiquitous, and their use as a semirigid external stylet for placing a gastroesophageal balloon tamponade device has shown promise in simulation studies, improving both time to placement and first-pass success; however, in vivo experience remains limited.[9, 10]

We report three cases of massive upper gastrointestinal (GI) bleeding in which a bougie-assisted technique facilitated successful placement of a gastroesophageal balloon tamponade device. These cases highlight the safety and effectiveness of this modified approach, which offers a practical and readily available adjunct for managing catastrophic hemorrhage when conventional placement methods prove difficult.

CPC-EM Capsule

What do we already know about this clinical entity?
Blakemore tubes are used to temporize uncontrolled upper gastrointestinal bleeding as a bridge to definitive care, but they are challenging to place.

What makes this presentation of disease reportable?
A simulation study of bougie-assisted Blakemore placement showed improved first-pass success and decreased placement time, but in vivo experience is limited.

What is the major learning point?
A bougie can serve as a semirigid external stylet to aid Blakemore placement. The added rigidity gives an advancing vector of force to the tip and prevents coiling.

How might this improve emergency medicine practice?
Bougie-assisted Blakemore placement is an effective rescue technique using a familiar and ubiquitous tool without an apparent increase in risk.

CASE SERIES

We describe three patients with varices who presented to the emergency department (ED) with massive upper GI bleeding and hemorrhagic shock requiring temporizing bleeding control. In all cases, a bougie tracheal tube introducer was used as a semirigid stylet to facilitate placement of a Blakemore tube.

Case 1

A 62-year-old man with history of alcohol-related cirrhosis, hepatocellular carcinoma, portal vein thrombosis, and esophageal varices presented for altered mental status and hematemesis. Upon arrival, his heart rate was 46 beats per minute (bpm) and blood pressure (BP) was 82/64 mm Hg. On examination, he was unresponsive to noxious stimuli and had large-volume hematemesis with aspiration. The patient was intubated for airway protection, and standard therapies for suspected variceal bleeding were initiated, including ceftriaxone, pantoprazole, and octreotide. Persistent shock prompted initiation of a massive transfusion protocol, and vasopressors were started.

Due to uncontrolled hemorrhage and delay to endoscopy, a Blakemore tube was placed using a bougie-assisted technique. The bougie was inserted into the distal gastric aspiration port and used as a semirigid stylet to guide placement under video laryngoscopy. Once positioned, the gastric balloon was partially inflated, and the position was confirmed radiographically. The gastric balloon was then fully inflated, and the bougie withdrawn (Image 1).

The distal tip of the Blakemore was inadvertently withdrawn into the esophagus, likely related to use of the distal port. The gastric balloon remained appropriately positioned within the stomach. Minimal ongoing bleeding was noted from a preexisting gastric tube and a subsequently placed gastric tube terminating in the esophagus. The patient sufficiently stabilized to undergo endoscopic variceal band ligation and transjugular intrahepatic portosystemic shunt (TIPS) placement. His hospital course was complicated by the development of multiorgan failure and hypoxic-ischemic brain injury. The patient died on hospital day 11.

Case 2

A 63-year-old man with alcohol-related cirrhosis complicated by ascites and esophageal varices presented with large-volume hematemesis. Upon arrival, his heart rate was 82 bpm, and BP was 77/53 mm Hg. Examination demonstrated ongoing hematemesis, scleral icterus, and abdominal ascites.

He was intubated, standard therapies for suspected variceal bleeding were initiated, and he was resuscitated with

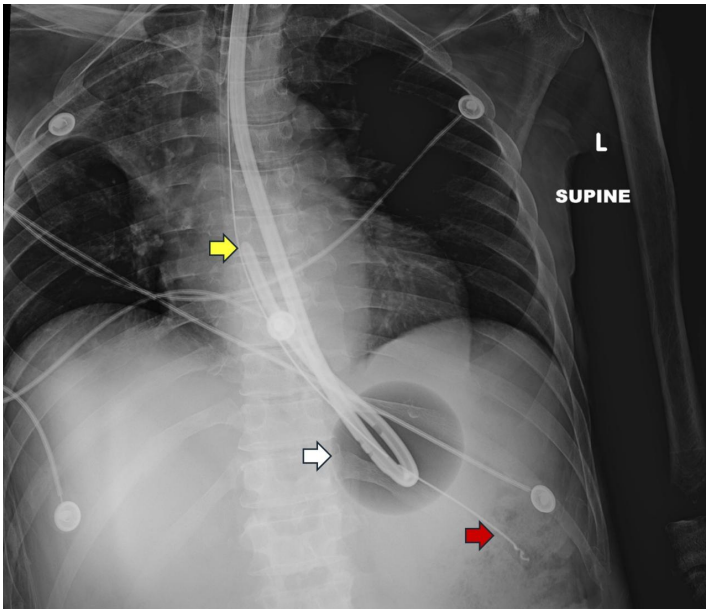


Image 1. Sengstaken-Blakemore tube placement. The gastric balloon (white arrow) is inflated within the stomach, but the tip (yellow arrow) has been inadvertently withdrawn into the esophagus. The companion gastric tube (red arrow) remains correctly positioned within the stomach.

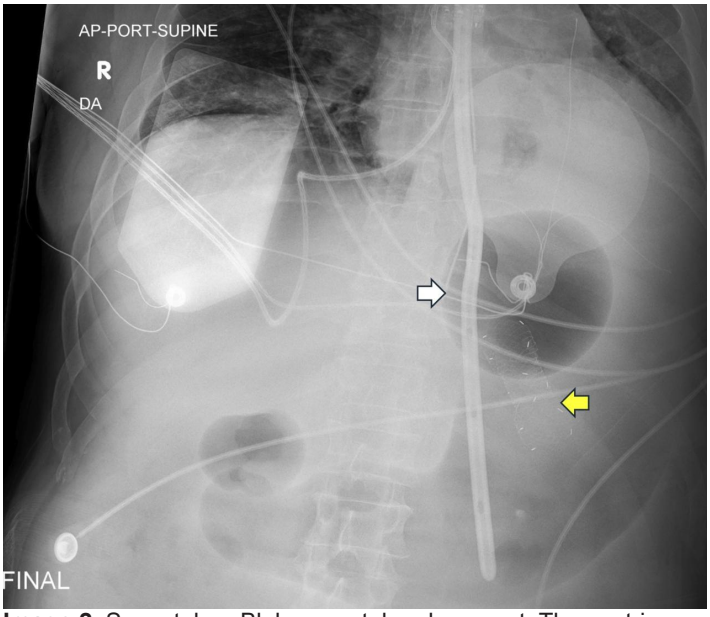


Image 2. Sengstaken-Blakemore tube placement. The gastric balloon (white arrow) is inflated within the stomach. A previously placed esophageal stent is displaced into the stomach (yellow arrow).

a massive transfusion protocol and vasopressors. While in the ED, emergent esophagogastroduodenoscopy (EGD) identified an actively bleeding distal esophageal varix that could not be successfully banded, and an esophageal stent was placed by the gastroenterology team. Unfortunately, bleeding recurred, prompting placement of a Blakemore tube for temporizing hemorrhage control. A bougie-assisted technique was again used under video laryngoscopy. In this case, the bougie was inserted into the proximal gastric aspiration port. Placement was straightforward and without complication (Image 2).

Despite inflation of the gastric balloon, bleeding from the esophagus persisted, prompting inflation of the esophageal balloon to a pressure of 34 mm Hg, which achieved hemostasis. After improvement in hemodynamics, the patient underwent successful embolization. Unfortunately, he developed severe hypoxic-ischemic neurologic injury and cerebral edema, and care was transitioned to comfort-focused measures. The patient died on hospital day 2.

Case 3

A 48-year-old man with alcohol-related cirrhosis and varices was transferred with ongoing hemorrhage after endoscopic control of only one of two bleeding varices. Upon arrival, his heart rate was 110 bpm, and BP was 92/53 mm Hg. He had been intubated by the transferring facility, and standard therapies for variceal hemorrhage were ongoing, including resuscitation with blood products. In response to uncontrolled bleeding despite endoscopic therapies, a Blakemore tube was placed. A bougie-assisted technique was again employed as described in Case 2, using the proximal gastric aspiration port. Under direct laryngoscopy, the device was appropriately positioned without difficulty and successfully achieved hemostasis with inflation of the gastric balloon alone (Image 3).

The patient underwent a TIPS procedure, which was unsuccessful. He subsequently had coil embolization of two gastroesophageal varices. Repeat EGD showed three large varices, which were banded. After treatment of aspiration pneumonia and hepatic encephalopathy, he was discharged on hospital day 9.

DISCUSSION

These cases highlight a practical, resource-conscious modification to a high-risk and technically challenging procedure used in the emergent management of life-threatening upper GI bleeding. Advancement of a Blakemore tube during active hemorrhage is frequently complicated by coiling, malposition, and placement delay related to multiple attempts, with risks including ongoing bleeding, procedural failure, airway compromise, and esophageal perforation.

Use of a bougie as a semirigid external stylet improves directional control and mitigates the tendency of the tube to coil or kink in the oropharynx and esophagus. Once inflated, the gastric balloon provides sufficient resistance to allow the bougie tip to dislodge from the side port, releasing the bougie while leaving the gastric tip appropriately positioned within

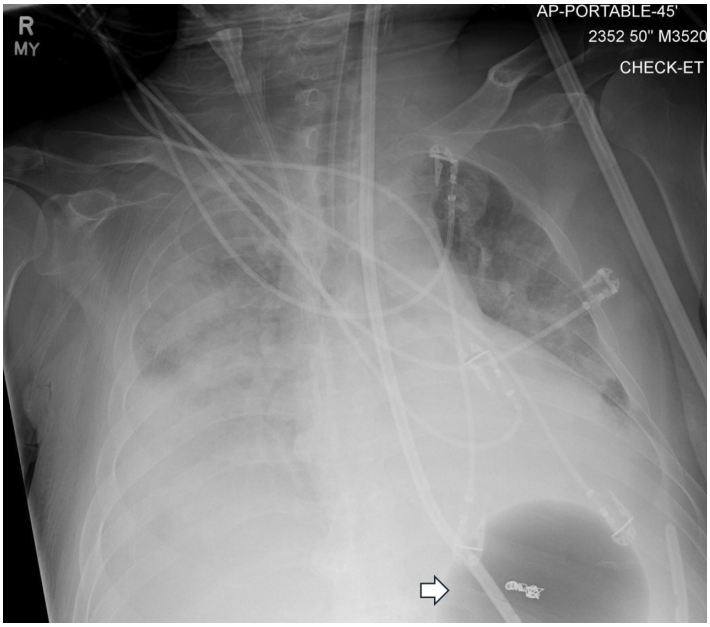


Image 3. Sengstaken-Blakemore tube placement. White arrow indicates the gastric balloon inflated within the stomach.

the stomach. This observation aligns with emerging data, including simulation-based research demonstrating improved first-pass success and reduced placement time compared to standard technique under conditions mimicking massive upper GI hemorrhage and airway contamination.[9]

To place a Blakemore tube using this modified technique (Video):[4]

1. Prepare the gastroesophageal balloon tamponade device and check balloon inflation to ensure proper function. Fully deflate the balloons before insertion.
2. Assemble the unit by wedging the coudé tip of the bougie into the most proximal gastric aspiration side port of the Blakemore tube (Image 4).
3. Holding some tension, wrap the Blakemore a few times around the bougie and then lubricate the unit. This tension and added rigidity allow the operator to transmit an advancing vector of force to the tip of the Blakemore.
4. Use either direct or video laryngoscopy to lift the glottic structures anteriorly, ensure stability of the endotracheal tube, and allow exposure of the proximal esophagus.
5. Place the Blakemore-bougie unit into the esophagus and gently advance to a depth of approximately 50 cm or until there is resistance.
6. Inflate the gastric balloon with 50 mL of air and confirm appropriate positioning with a radiograph.
7. Inflate the gastric balloon fully with an additional 200 mL of air (total 250 mL).
8. Retract the unit gently until there is resistance, indicating the gastric balloon is abutting the gastroesophageal junction.
9. Apply gentle traction to only the bougie, dislodging the tip from the gastric aspiration port. There will be some slight resistance as the tip detaches from the side port.
10. Withdraw the bougie fully from the patient.
11. Place the device to 1 kg of traction.
12. Confirm final Blakemore and gastric balloon positioning with a radiograph.
13. Assess for and manage ongoing bleeding, inflating the esophageal balloon only if clinically indicated.

In all three cases, the bougie-assisted approach allowed rapid, controlled placement without procedural complications, and hemostasis was achieved in each instance. These findings are consistent with a prior series describing this modification, although that report was limited to a single Blakemore tube type.[10] Notably, technique variation may be important. Use of the distal gastric aspiration port, as in Case 1, was associated with unintended withdrawal of the tube tip into the esophagus upon removal of the bougie. This is suboptimal because the gastric drainage ports are intended to decompress the stomach and monitor for ongoing gastric bleeding. In our case, a prepositioned orogastric tube allowed continued decompression and monitoring of gastric output, and the Blakemore's gastric balloon positioning was unaffected; therefore, the displaced tip of the Blakemore was left in place. In contrast, use of the proximal gastric aspiration port in Cases 2 and 3 allowed reliable placement without displacement of the device tip during detachment of the bougie. This distinction may represent a simple technical optimization that improves procedural reliability. We hypothesize that using the proximal gastric aspiration port leaves a length of unattached, pliable tubing that cannot readily be drawn past the inflated gastric balloon into the esophagus.

Our experience underscores the importance of procedural adaptability when conventional methods prove inadequate. The ability to quickly modify a lifesaving intervention with readily available equipment, such as a bougie, may substantially influence patient outcomes when definitive therapy is delayed. Importantly, procedural success did not necessarily translate into survival, reflecting the severity of underlying disease and the high mortality associated with this condition. However, in each case, the gastroesophageal balloon tamponade achieved its intended role as a bridge to definitive therapy.

Although no complications occurred in these three cases, the risk profile of this modification is not fully characterized. Theoretical risks include injury to the glottis, esophagus, or stomach. Disruption of varices or displacement of previously placed hemorrhage-control measures is a risk with any instrumentation of the esophagus. Several factors mitigate these concerns, however. The bougie is specifically designed for insertion into the airway and is pliable. Theoretically, it should pose little added risk to esophageal tissue, particularly as the tip is shrouded within the soft, flexible tip of the Blakemore tube. In our experience, minimal force is required

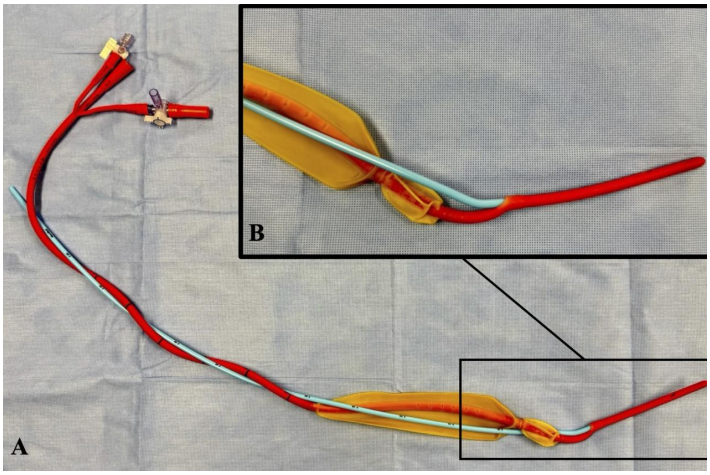


Image 4. Unit assembly for bougie-assisted Sengstaken-Blakemore tube placement. A, With gastric and esophageal balloons fully deflated, the coudé tip of the bougie (tracheal tube introducer) is wedged into the most proximal gastric aspiration port. The tube is wrapped around the bougie to provide rigidity and directional control. B, Magnified view showing the attachment point where the bougie tip is wedged approximately 0.5 centimeters into the gastric aspiration port.

to advance the unit, and the improved directional control avoids forceful or repeat blind placement attempts as well as the introduction of more rigid tools such as Magill forceps. Balanced against the immediate threat of exsanguination, we believe the incremental risk of this technique is low and the risk-benefit balance favorable.

Limitations include a small sample size, retrospective observation, and inability to compare outcomes directly with standard placement technique. The bougie-assisted approach was used as the initial placement method in all three cases rather than as a rescue after failed conventional placement, so no comparative conclusions regarding standard technique can be drawn. Assessment of placement ease is operator-reported and neither standardized nor objective and may reflect operator experience rather than the technique itself.

CONCLUSION

This case series adds to a limited but growing body of evidence supporting bougie-assisted gastroesophageal balloon tamponade device placement as an effective, accessible, and potentially more reliable modification to traditional technique. It expands prior published experience by demonstrating reproducible use across multiple cases and identifies a potential technical advantage of the proximal gastric aspiration port. Although further study is needed to define success rates, complications, and patient-centered outcomes compared with conventional technique, the infrequent use of gastroesophageal balloon tamponade devices will likely impose methodologic challenges to future investigation. By bridging the gap between simulation-based findings and clinical application, this series emphasizes the value of readily available adjuncts in the management of catastrophic upper GI hemorrhage. Although promising, there are insufficient data and experience to suggest a bougie-assisted technique be first-line when gastroesophageal balloon tamponade placement is indicated. While an attempt using standard technique is advisable, familiarity with bougie-assisted Blakemore placement as a rescue tool may prove beneficial.

Video. Instructional video demonstrating bougie-assisted Blakemore tube placement on a task training model.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case series. Documentation on file.

Address for Correspondence: Travis Hase, MD, Advocate Christ Medical Center, Department of Emergency Medicine, 4440 W. 95th St., Oak Lawn, IL, 60453. Email: travis.hase@advocatehealth.org and travishase@gmail.com.

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Prehospital Cold-Water Immersion for Undifferentiated Heat Stroke: A Case Series

David Lindow, DO*
Bryan Everitt, MD†
Nick Smith, LP‡
Michael Wells, LP‡
Robert Dickson, MD‡
Casey Patrick, MD*‡§

*University of Houston, Department of Emergency Medicine, Kingwood, Texas
†Allegiance Mobile Health Emergency Medical Services, Corpus Christi, Texas
‡Montgomery County Hospital District Emergency Medical Services, Conroe, Texas
§Baylor College of Medicine, Department of Emergency Medicine, Houston, Texas

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Introduction: Heat stroke is a time-sensitive condition that requires early intervention to prevent morbidity and mortality. Heat-related deaths are also increasing. Multiple studies have evaluated various cooling methods and assessed their effectiveness, safety, and practicality across diverse settings and populations. Earlier and faster cooling leads to improved patient outcomes. The National Association of Emergency Medical Services Physicians recommends initiating prehospital cold-water immersion before emergency medical services (EMS) transport.

Case Series: This case series evaluated the feasibility and effectiveness of prehospital cold-water immersion initiated before and continued during EMS transportation from the scene to emergency department arrival in this critically ill patient population.

Conclusion: Rapid initiation and maintenance of prehospital cold-water immersion in undifferentiated heat stroke was both feasible and effective. Over the course of the case series, no deaths were reported, and all patients were eventually discharged home. [Clin Pract Cases Emerg Med. 2026;10(4):461–468.]

Keywords: *heat stroke; emergency medical services; cold water immersion; resuscitation; critical care.*

INTRODUCTION

Heat stroke is a severe condition that requires early intervention to prevent long-term sequelae, morbidity, and mortality.[1-4] Heat stroke is classified into two distinct types: classical and exertional. Classical heat stroke typically affects individuals at the extremes of age, owing to a combination of environmental and underlying medical factors. In contrast, exertional heat stroke typically occurs in younger, healthier individuals. Heat stroke develops when the body loses its ability to regulate its core temperature. The elevated body temperatures during heat stroke have been shown to cause denaturing of proteins on a molecular level, leading to cellular death and organ failure, including the central nervous system, heart, kidney, lungs, liver, spleen, muscle, and intestines.[1,2]

Death due to heat-related illnesses, such as heat stroke, has been a prevalent problem in the United States. Between 2018 and 2020, the Centers for Disease Control and Prevention recorded 3,066 heat-related deaths, with the highest percentage occurring among individuals 55 to 74 years of age.[1] One study reported that exertional heat-related deaths are the top noncardiac cause of death in laborers.[2] Although it is difficult to get an accurate account of heat-related deaths due to multiple variables, heat-related illnesses and mortality increase significantly during heat waves and periods of elevated temperatures.[3] The earlier a patient can be treated and cooled, the lower the risk of long-term sequelae and death.[3–6] The most effective current treatment for heat-related illness is to rapidly cool the body's core temperature early.[2,4,6]

Numerous studies have been conducted to evaluate various cooling methods, assessing their effectiveness, safety, and practicality in diverse settings and populations. These methods include passive cooling with rest, cold showers,[7] warm cooling,[8] cold water cooling,[9] ice water cooling, ice sheets,[10-12] tarp cooling,[13–16] body bag use,[17] cold fluid intravenous (IV) infusion,[11] and ice packs. Most of the studies conclude that more rapid cooling improves outcomes and is associated with fewer long-term sequelae and negative neurological outcomes.[4–6,18,19] Cold water immersion (CWI) is the most effective cooling method, decreasing core body temperature at a rate of 0.20 °C/min to 0.36 °C/min..[5–7,20] Some studies challenge the general consensus, arguing that cooling too rapidly is dangerous, with potential risks including shock, dysrhythmias, hypotension, and seizures; suggesting that warmer temperatures should be used instead.[8,21] Although the risks mentioned above can and often do occur, they are manageable if proper precautions are taken with medications and countermeasures in place.[6] In an 18-year retrospective study conducted at the Falmouth Road Race, Falmouth, MA,. patients with exertional heat stroke treated with cold water immersion (CWI) in water less than 10 °C cooled at at an average rate of 0.22 °C/min.[22] Although that study did not track incidence rates for those specific complications, the overall survival rate was 100%, and 93% of the 270 patients were discharged directly from the medical tent.[22] Cold-water immersion has been shown to be safe and effective, such that it can be performed without serial rectal temperature measurements and can be based on initial temperature and immersion time.[22,23]

CPC-EM Capsule

What do we already know about this clinical entity?
Heatstroke is a time-sensitive emergency. Rapid cooling and emergency department transport are the foundations of prehospital care.

What makes this presentation of disease reportable?
Limited evidence exists to support initiating cold-water immersion for undifferentiated heatstroke in the civilian prehospital setting.

What is the major learning point?
Prehospital cold-water immersion for critically ill heatstroke patients is feasible and effective in a ground-based emergency medical services system.

How might this improve emergency medicine practice?
These preliminary data should support larger prospective studies of cold-water immersion for the treatment of prehospital heatstroke.

Cold-water immersion for heat stroke treatment has become the standard of care, with proposed algorithms to guide its successful implementation in emergency department (ED) and athletic event settings.[6,7,24] Tishukaj noted that exertional heat stroke patients are 3.0 to 3.7 times less likely to die when they are treated with CWI prior to transport.[2] The National Association of Emergency Medical Services Physicians examined these cases and others and considered the risks, benefits, and outcomes. Their recommendation is for CWI with complete immersion to the level of the clavicles in water at 10 °C or below.[25] Along with this recommendation, they also suggest initiating cooling of the patient prior to transportation, which aligns with the study conducted by Tishukaj.[26] Most studies have been conducted in young, healthy individuals, focusing on exertional heat stroke rather than classic heat stroke. However, there have been a few case reports in the literature regarding CWI treatment for classic heat stroke in elderly patients, which have proven safe and effective.[16,26,27]

Cold-water immersion initiation prior to transportation is the standard of care for treating heat stroke.[4–6] One case series featuring five elderly patients with classical heat stroke reported treatment on scene with CWI, using water with continuous flow from a fire hydrant prior to transportation.[26] While effective cooling prior to emergency medical services (EMS) transport was shown, in one instance it took 69 minutes to decrease the core temperature to the point they could safely remove the patient from the water and transport them to the hospital.[27] This highlights the need to balance time to patient cooling and the necessity of rapid hospital transport for additional diagnostic and treatment efforts. Emergency medical services must have the equipment and capabilities to quickly initiate CWI on scene, package the patient, and continue cooling during transport. However, due to limited space, limited access to reliable ice/water, and motion during transport, prehospital CWI presents unique challenges, raising questions about its prehospital feasibility and effectiveness.

This case series summarizes nine patients treated under a previously approved and implemented EMS protocol for initiation of CWI in patients with undifferentiated prehospital heat stroke. The participating agency is a ground-based EMS service in the greater Houston area that employs nearly 350 EMS clinicians, including paramedics, advanced emergency medical technicians, and emergency medical technicians and services all emergency dispatch response and transport for an area of approximately 1,100 square miles. This area has a population of roughly 750,000, aimed at which the service receives approximately 90,000 service calls per year. The nine

patients in this case series were treated between July 18, 2023, and September 14, 2024.

This case series was part of the continuous quality improvement of the CWI protocol, which was implemented on May 25, 2022 (Figure). To ensure patient safety, all CWI cases were thoroughly reviewed by EMS medical direction for protocol compliance and clinical decision-making. Direct paramedic feedback was provided when necessary and appropriate. Hospital data was obtained as part of the continuous quality improvement project, as well.

Before protocol rollout, all EMS clinicians attended a mandatory one-hour lecture-based protocol and heat stroke education session, followed by a training video. The CWI protocol inclusion criteria include suspected heat stroke-related illness, defined in the protocol as altered mental status accompanied by a core temperature of greater than 40.6 °C (105 °F). Advanced age defined as older than 60 years, was considered a relative contraindication due to the increased risk of infectious causes of fever, and protocol teaching emphasized the necessity of obtaining a thorough history prior to initiation of CWI. Online consultation with permission from either a district chief/supervisor paramedic, or medical direction was required prior to initiation of CWI. Equipment required for the procedure included an immersion bag, ice, and water. Patients were undressed and placed in the bag on the stretcher with the head elevated to a minimum of 15°. The bag was then filled with ice and water to the patient's chest (Image). Emergency medical services (Table 1) and ED/hospital data (Table 2) were collected.

MCHD	PROCEDURES PROC- 28	IMMERSION COOLING	Page 1 of 1 Last Revision: 05/23/2022

Indications:
- Suspected heatstroke related illness with altered mental status and a core temperature >105 F

Contraindications:
- Age > 60 years (relative)
- Core temperature < 105F
- Suspected infection or fever

Procedure:
- Plan
  - District Chief or MD consult is required prior to immersion
  - Lay cooling bag on stretcher
  - Undress the patient
  - Place patient in cooling bag on stretcher with head elevated at minimum 15 degrees
  - Fill the cooling bag with ice and/or cold water to patient's mid-axillary line up to nipple-line
  - Zip bag up only enough to keep water/ice in the bag
  - Continue to monitor temperature

Equipment:
- Cooling Bag
- Ice
- Water

Medication:
- Midazolam to control seizures as needed

Considerations:
- If ice and/or water is not available do not delay transport. Use other cooling methods.
- Verification of cooling by obtaining additional core temperatures should occur prior to transport.
- Ensure early pre-notification to receiving facility that the patient has received cold water/ice immersion therapy.
- If indicated, do not delay any procedures prior to immersion.
- Do not interrupt cooling for diarrhea, emesis, combativeness or seizures

Figure. Cold-water immersion protocol.
MD, medical director; *F*, Fahrenheit.



Image. Cold water immersion example.

CASE SERIES

Case #1

A 67-year-old man was initially found unconscious by his family on his bed. The family reported that the air conditioner in the house had not been functioning properly, and the interior of the house had become extremely hot. Emergency medical services found the patient unresponsive with rapid, shallow breathing, tachycardia, and a reported Glasgow Coma Scale (GCS) score of 3. The patient's airway was addressed with bag-valve mask ventilations. The rectal temperature was 41.8 °C (107.3 °F). The CWI protocol was initiated, bilateral IV access sites were established, and a cold 0.9% normal saline bolus was administered. The patient was intubated with ketamine and rocuronium due to concerns for a lack of adequate airway protection.

Approximately 20 minutes after immersion, the patient's blood pressure decreased to 89/57 mm Hg. He received a 20 mcg push dose of IV epinephrine, which resulted in an appropriate increase to 119/82 mm Hg. Eight minutes later, he received a second dose, which increased the blood pressure to 149/75 mm Hg. Just before ED arrival, a repeat rectal temperature was recorded as 39.6 °C (103.3 °F). Hospital data demonstrated an elevated initial lactic acid of 3 mmol/L, (reference range, < 2 mmol/L), with normal aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (AST 5-40 U/L and ALT 7-56 U/L) values. The patient was admitted to the intensive care unit (ICU) with a length of stay (LOS) of seven days and 16 hours before being discharged home.

Case #2

A 72-year-old male was found unresponsive in direct sunlight by his family. Emergency medical services described the patient to be unresponsive with agonal, ineffective breathing and hot to the touch, with GCS 4. Emergency

Table 1. Patient data—prehospital—used in a case series evaluating the feasibility and effectiveness of prehospital cold-water immersion initiated before/during emergency medical services transport.

Case	Age	Sex	Initial EMS GCS	Initial EMS Pulse (BPM)	Initial EMS BP (mmHg)	Initial EMS Temp (°C)	Final EMS GCS	Final EMS Pulse (BPM)	Final EMS BP (mmHg)	Final EMS Temp (°C)	EMS Scene Time (min)	EMS Transport Time (min)	Time from Immersion to last rectal temp (min)	Time from Immersion to ED arrival time (min)	EMS Intubation (Y/N)
1	67	M	3	160	109/73	41.8	3T	139	154/89	39.6	47	18	48	48	Y
2	72	M	4	124	130/68	41.3	3T	146	193/79	NA	48	24	NA	41	Y
3	78	M	3	132	136/61	40.9	13	113	126/53	39.3	28	10	22	22	N
4	60	F	3	175	85/52	40.9	9	160	92/50	39.7	21	15	17	20	N
5	49	M	7	179	179/71	42.7	7	155	143/83	40.0	16	29	20	20	N
6	51	M	10	111	108/69	40.6	10	98	126/71	39.4	27	13	11	14	N
7	60	M	9	86	150/90	40.7	14	119	109/67	NA	29	31	NA	36	N
8	70	M	12	127	73/48	41.3	15	104	134/73	NA	20	25	NA	40	N
9	50	F	6	122	66/47	42.1	6	110	119/57	39.0	30	20	23	23	N

BP,blood pressure; *BPM*, beats per minute; *°C,* degrees celcius; *EMS*, emergency medical services; *F*, female; *GCS*, Glasgow Coma Scale; *M*, male; *mm Hg*, millimeters of mercury; *min,* minute; *N*, No; *NA*, not available; *Y*, yes; *3T*, GCS of 3 and Intubated.

medical services provided airway suctioning and immediately administered oxygen via bag-valve mask. The patient's rectal temperature was recorded as 41.3 °C (106.4 °F). External cooling attempts were initiated by placing ice packs in the armpits, groin, and behind the neck. An IV was established, and the patient received a 0.9% normal saline bolus and the initiation of the CWI. Ten minutes after cooling initiation and six minutes after CWI initiation, the patient was noted to be hypotensive at 82/52 mm Hg. The patient was administered a 20-mcg push dose of epinephrine, which increased blood pressure to 127/64 mm Hg. The patient's GCS score at this time also increased to 7. The respiratory status was still inadequate, requiring bag-valve mask ventilations; so, the patient was intubated using ketamine and rocuronium. Approximately 12 minutes after the initial dose of epinephrine, the blood pressure decreased to 81/44 mm Hg, and a second 20 mcg push-dose of epinephrine was administered. The patient again responded appropriately, with blood pressure increasing to 109/69 mm Hg. Upon ED arrival, the patient's initial lactic acid was elevated at 5.9 mmol/L, and AST/ALT were elevated to 56 U/L and 258 U/L, respectively. The patient also had acute kidney injury (AKI) with an initial creatinine of 8.8 mg/dL (reference range, 0.7-1.3 mg/dL) with associated hypokalemia of 2.2 mEq/L (3.5-5.0 mEq/L). While at the hospital, the patient was admitted to the ICU and stayed for nine days, 18 hours before being discharged home.

Case #3

A 78-year-old man was initially found unconscious in his vehicle by bystanders who removed him and called emergency dispatch. Upon EMS arrival, the patient had a GCS score of 3. The prehospital electrocardiogram was noted to be sinus tachycardia, and the patient was tachypneic and hypoxic. Emergency medical services provided supplemental oxygen via bag-valve mask at 15 liters/min. He was found to have a rectal temperature of 40.9 °C (105.6 °F). Cooling measures were started by placing ice packs in the patient's armpits, groin, and neck. Intravenous placement was initiated, a cold 0.9% normal saline bolus was administered, and CWI was initiated. Upon ED arrival, rectal temperature was reassessed and noted to have decreased to 39.3 °C (102.7 °F). The patient was reported to be more alert and answered some questions appropriately with a documented GCS score of 13. The time from CWI initiation to the repeat rectal temperature was 22 minutes. Lactic acid and AST/ALT were normal. The patient did have AKI with an initial creatinine of 4.3 mg/dL. He was admitted to the hospital floor and remained for three days, 20 hours before discharge home.

Case #4

A 60-year-old woman was found unresponsive in her home. Upon EMS arrival, the patient had an initial GCS score of 3 but then began seizing with oxygen saturations of 60%. Paramedics immediately began addressing the hypoxia by using a bag-valve mask at 15 L/min and placing a nasopharyngeal airway. She was given 10 mg intramuscular midazolam for the seizure, which subsequently ceased. The patient's rectal temperature was found to be 40.9 °C (105.6 °F). Cold-water immersion was initiated, an IV was established, and the patient received a cold 0.9% normal saline fluid bolus. Two minutes after CWI initiation, the blood pressure dropped to 81/39 mm Hg. A 20-mcg push-dose of epinephrine was administered, resulting in an appropriate response, with blood pressure increasing to 133/69 mm Hg.

Table 2. Patient data—hospital—used in a case series evaluating the feasibility and effectiveness of prehospital cold-water immersion initiated before/during emergency medical services transport.

Case	Admission	ICU	Death	LOS	ED HR (BPM)	ED BP (mmHg)	ED GCS	ED Temp (°C)	ED Lactic Acid (mmol/L)	Initial Potassium (mEq/dL)	Initial AST/ALT (U/L)	Initial Creatinine (mg/dL)	CK (IU/L)	ED Intubation (Y/N)
1	Y	Y	N	7D16H	160	154/89	3T	41.8	3	3.7	50/40	1.6	163	N/A
2	Y	Y	N	9D18H	101	134/62	3T	41.3	5.9	2.2	256/258	8.8	88	N/A
3	Y	N	N	3D20H	113	126/53	14	39.3	1.2	3.8	21/51	4.3	197	N
4	Y	Y	N	5D	170	85/52	14	40.9	2.5	4.5	15/less than 7	1.9	77	N
5	Y	Y	N	7D5H	170	179/71	10	40.0	1.4	3.0	339/245	1.8	10750	Y
6	Y	Y	N	17D19H	98	126/71	14	40.7	1.7	3.1	30/27	1.1	72	N
7	Y	N	N	19H	119	109/67	14	37.3	1.7	3	23/9	0.9	121	N
8	Y	Y	N	14D20H	104	122/76	15	36.4	5.9	4.7	52/92	3.9	159	N
9	Y	Y	N	36D6H	109	72/43	3	39.3	4.2	2.7	90/53	1.1	154	Y

ALT, alanine aminotransferase; *AST*, aspartate aminotransferase; *BP*, blood pressure; *BPM*, beats per minute; *CK,* creatine kinase; *°C,* degrees celcius; *ED*, emergency department; *D*, day; *GCS*, Glasgow Coma Scale; *H*, hour; *HR*, heart Rater *ICU*, intensive care unit; *LOS*, length of stay; *min,* minute; *N/A*, not applicable or not available; *N*, no; *Y*, yes; *3T*, GCS of 3 and Intubated.

During transport, the patient was observed opening her eyes and withdrawing from painful stimuli. Upon ED arrival, 17 minutes after CWI initiation, the patient's rectal temperature was 39.7 °C (103.5 °F). The patient's mentation also notably improved, as evidenced by a GCS score of 9. The patient's initial lactic acid was elevated at 2.5 mmol/L, and AST/ALT were normal. The patient had mild AKI with an initial creatinine of 1.9 mg/dL. The patient was admitted to the ICU and stayed for five days before being discharged home.

Case #5

A 49-year-old man was found altered by coworkers while working outside. Upon EMS arrival, the patient was reported to be altered, listless, pale, and hot to the touch. The patient had a GCS score of 7, with a rectal temperature of 42.7 °C (108.9 °F). He was placed on oxygen at 15 L/min. via a nonrebreather mask. Cold packs were applied to his groin and armpits. An IV was started, and a cold 0.9% normal saline bolus was infused. The patient became combative, requiring sedation using 100 mg IV ketamine. Cold-water immersion was partially initiated; however, the crew had no ice available. During transport, EMS stopped at a gas station and purchased ice to cool the patient further. The patient again became more combative and was given a second dose of 100 mg IV ketamine. The patient required suctioning to control secretions but maintained intact airway reflexes. Just prior to arrival at the hospital, seizure activity was observed, and 5 mg IV midazolam was administered. Upon ED arrival, 20 minutes after initiating CWI, the patient's rectal temperature had decreased to 40 °C (104.0 °F). Lactic acid was normal, but AST/ALT were elevated to 339 and 245 U/L, respectively. The patient had mild AKI with an initial creatinine of 1.8 mg/dL and rhabdomyolysis with creatine kinase of 10,750 IU/L (reference range, 25-320 U/L). The patient was intubated in the ED, admitted to the ICU, and stayed for seven days and five hours before being discharged home.

Case #6

A 51-year-old man was found outside with altered mentation. Upon EMS arrival, the patient was altered, tachycardic, tachypneic, hot to the touch, with a GCS score of 10. The patient's initial oral temperature was recorded as 40.6 °C (105.0 °F). He was placed on oxygen at 15 L/min due to oxygen saturations of 91%, and cold packs were applied to his neck, axillae, and groin. Cold-water immersion was initiated, intraosseous (IO) access was established, and a cold 0.9% normal saline fluid bolus was infused. Two mg IO midazolam was administered for suspected focal seizure activity. An IV was then established, and continued fluid administration was maintained. Upon ED arrival, 11 minutes after initiation of CWI, the patient's temperature was 39.4 °C (102.9 °F). The patient's initial lactic acid, AST/ALT, and creatinine were normal. The patient was admitted to the ICU and had a hospital LOS of 17 days and 19 hours before being discharged home.

Case #7

A 60-year-old man was found outside with altered mental status. Upon EMS arrival, the patient was found with a GCS score of 10. The patient had minor abrasions but otherwise had no obvious signs of significant injury. The initial rectal temperature was 40.7 °C (105.3 °F), prompting initiation of CWI. An IV was established, and the patient received a cold

0.9% normal saline bolus. On ED arrival, the patient was noted to be speaking, answering questions, and following commands with a GCS score of 14. Time from EMS arrival to CWI initiation was 29 minutes, and 36 minutes from CWI initiation to hospital arrival. The patient's initial temperature was 37.3 °C (99.1 °F). His initial lactic acid, AST/ALT, and creatinine were normal. He was admitted to the hospital floor and LOS was 19 hours before being discharged home.

Case #8

A 70-year-old man was found with unexplained confusion, and the family called emergency dispatch. Upon EMS arrival, the patient was found altered with a GCS score of 12. Vital signs revealed hypotension (73/48 mm Hg), tachycardia (127 beats/min[bpm]), and a rectal temperature of 41.3°C (106.4 °F). Oxygen was administered with a nasal cannula at six L/min. Additionally, an IV was started, a 20-mcg push dose of epinephrine was administered, a cold 0.9% normal saline bolus was initiated, and CWI was performed. During transport, the patient's mental status improved, and he answered questions appropriately. The mentation improved to a GCS score of 15. There were five minutes from EMS arriving on scene to CWI initiation and 40 minutes from CWI initiation to hospital arrival; the initial ED temperature was 36.4 °C (97.6 °F). The patient's initial lactic acid was elevated at 5.9 mmol/L, and AST/ALT were normal. The patient had an AKI with an initial creatinine of 3.9 mg/dL. He was admitted to the ICU and LOS was 14 days and 20 hours before being discharged home.

Case #9

A 50-year-old woman was found with severe altered mentation. EMS noted initial hypotension (66/47 mm Hg) and tachycardia (122 bpm), with a GCS score of 6. Initial rectal temperature was 42.1 °C (107.8 °F). A nonrebreather oxygen mask was applied, and cold packs were placed in the groin, armpits, and the back of the neck. An IV was established, a cold 0.9% normal saline bolus was administered, and CWI was initiated. A total of 22 minutes elapsed from EMS arrival to CWI initiation. En route to the hospital, the ambulance stopped to obtain additional ice for further cooling. Before ED arrival, a seizure was noted, and 10 mg IV midazolam was administered. The patient's initial ED rectal temperature 23 minutes after CWI initiation was 39.3 °C (102.2 °F), with a GCS score of 6. Lactic acid was elevated at 4.2 mm/dL, and AST/ALT were mildly elevated to 90 U/L and 53 U/L, respectively. Creatinine level was normal. The patient was intubated in the ED, admitted to the ICU, and had a LOS of 36 days and six hours before being discharged home.

DISCUSSION

In this case series, prehospital CWI was feasible and effective. Cold-water immersion has been shown to be the prehospital treatment of choice for exertional heat stroke in both military and athletic environments.[5-7] However, few data exist regarding prehospital treatment of undifferentiated heat stroke in a less controlled, more variable emergency dispatch ground-based response setting.

These patients were critically ill with markedly deranged hemodynamics, respiratory status, and altered mental status with associated seizure activity. All patients received 0.9% normal saline fluid boluses, and four required vasopressor support with push-dose epinephrine. However, no patient required long-term vasopressor support via a continuous infusion (which was available under the EMS protocols in effect during the study period). Although hypotension occurred in several of these patients after CWI, its significance to the potential cold shock response remains unclear.[28,29] Since hypotension is an expected effect of rapid cooling, vasopressor preparation and readiness are crucial when initiating CWI.

Three patients were also noted to have reported seizure activity. One patient (case #4) was noted to be seizing prior to CWI initiation, while two other patients (case #5 and #6) developed seizure activity post-CWI. All received appropriate prehospital administration of parenteral midazolam with EMS documentation of seizure activity cessation. Persistent nonconvulsive seizure activity is a possibility, as electroencephalography was not available in the field or ED. Because seizure activity is a known complication of heat stroke,[3,4,19] benzodiazepine treatment should be available and prepared when encountering heat stroke and initiating CWI. Two patients required intubation prior to ED arrival, and two additional patients were intubated in the ED upon hospital arrival. The critical nature of heat stroke was further demonstrated by seven of nine patients also requiring ICU admission.

No cases of significant hyperkalemia were noted; however, unsurprisingly, over half (5/9) had lactic acidosis and AKI on ED laboratory investigation. Rhabdomyolysis and significant AST/ALT elevation were both uncommon but were noted in a patient experiencing exertional heat stroke (Case #5). While our small sample size limits detailed analysis, heat stroke should be recognized as a time-sensitive emergency with a high likelihood of hemodynamic instability, seizure, respiratory compromise, and ICU admission. Emergency medical services and ED protocols should be prepared and proactive in anticipating these events.

Logistical challenges arose in initiating prehospital CWI. We did not stipulate where the paramedics should obtain ice and left the decision to their discretion. In some cases, our fire department first-responder partners had water and ice available, whereas in others, paramedics obtained supplies from the patients' homes and local businesses. Ambulance space is limited, making it difficult for EMS crews to carry ice. Water is readily available in most places; however, ice is more difficult to obtain. In two cases, because no ice was available initially, the patient was immersed in water until the ambulance stopped at a business where bags of ice were obtained and added to the water to improve patient cooling.

Future studies should explore methods for carrying and storing ice more efficiently on ambulances, with guidance on the minimum amounts of ice and water required to initiate CWI.

Some aspects of prehospital CWI, however, went smoothly. Monitoring the patient's vital signs during immersion was successfully accomplished, because the monitoring cables are waterproof. Patients were attached to monitoring before being submerged. While the patient was submerged in water, the wires were left in place for continuous monitoring, and cable connections were kept dry. In prior work, this has been shown to be possible using a continuous rectal monitoring probe.[26] As in earlier studies, a waterproof immersion bag was used and proved effective.[2,5,7,15,17,24,25] These bags are relatively inexpensive, easily compactible, and easily stocked on each EMS apparatus.

Ultimately, we feel that CWI is feasible for undifferentiated heat stroke patients presenting in the ground-based, emergency dispatch prehospital setting and should be considered for all EMS protocols. The introduction is uncomplicated and inexpensive, involving only the purchase of waterproof immersion bags and initial training. Despite the apparent benefits and relative ease of implementing prehospital CWI, evidence indicates that EMS protocols remain significantly lacking.[30] Continuing educational and awareness efforts for EMS medical directors and clinicians are likely warranted.

LIMITATIONS

Generalizability is limited by the small number of patients treated, the retrospective nature of the data, and the absence of a comparator group in this case series. Due to the pragmatic nature of the immersion protocol, which is intended for use in nonmilitary or athletic event situations, the amount of ice and the timing of ice placement in the water baths were variable. While we did have some laboratory, admission, and mortality data from our hospital partners, additional details, including CWI immersion time, cooling reduction rate, and functional status at discharge, would be desirable in future studies.

CONCLUSION

In this case series, rapid initiation and maintenance of prehospital CWI for undifferentiated heat stroke were both feasible and effective when initiated by paramedics. These patients were critically ill, with seven of the nine requiring ICU admission; however, there were no reported deaths, and all patients were discharged home. Larger prospective studies are needed to further investigate this treatment.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case series. Documentation on file.

Address for Correspondence: Casey Patrick, MD, Montgomery County Hospital District Emergency Medical Services, 1400 South Loop 336 West, Conroe, TX 77304. Email: cpatrick@mchd-tx.org.

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A Case Report of Spontaneous Pneumomediastinum Following Chest Massage: A Rare Clinical Presentation

Naveed Ul Hassan H, MBBS*
Kowsthubha B G, MBBS, MD†
Amaravathi Udhayakumar, MBBS, MD†

*Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India
†Jawaharlal Institute of Postgraduate Medical Education and Research, Department of Emergency Medicine, Puducherry, India

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Introduction: Spontaneous pneumomediastinum is a rare condition characterized by the presence of air in the mediastinum without preceding trauma. It is often triggered by minor events such as coughing, physical exertion, or traditional therapies.

Case Report: A 52-year-old male with a history of pulmonary tuberculosis developed sudden left-sided chest pain and swelling following traditional chest massage therapy. Examination revealed subcutaneous emphysema with palpable crepitus. Imaging showed characteristic signs of pneumomediastinum with bilateral lung opacities. As a complication of pneumomediastinum the patient developed mediastinitis and a mediastinal abscess, requiring open thoracotomy and drainage. He recovered with multidisciplinary care.

Conclusion: Early recognition of spontaneous pneumomediastinum in emergency settings is crucial, especially when chest pain is accompanied by subcutaneous emphysema, to prevent serious complications. [Clin Pract Cases Emerg Med. 2026;10(4):469–472.]

Keywords: *chest massage; spontaneous pneumomediastinum; Macklin effect; mediastinitis; mediastinal abscess.*

INTRODUCTION

Spontaneous pneumomediastinum, also known as Macklin or Hamman syndrome, is an uncommon clinical entity with an incidence of approximately one in 30,000 emergency visits.[1] It involves the presence of free air in the mediastinum without antecedent trauma, predominantly affecting young males. The pathogenesis involves alveolar rupture caused by a sudden increase in intra-alveolar pressure, with air dissecting along bronchovascular sheaths—the Macklin effect.[2,3] Common triggers include vigorous coughing, strenuous physical activity, labor, vomiting, and the Valsalva maneuver.[1] Underlying pulmonary diseases, such as asthma and tuberculosis, may predispose individuals to spontaneous pneumomediastinum.

Chest pain is the most frequent symptom, often accompanied by dyspnea, cough, or neck swelling. Although usually benign, the condition can lead to serious complications such as mediastinitis and myocardial injury if not promptly diagnosed, which can be life-threatening. This case is distinctive due to the occurrence of spontaneous pneumomediastinum in a middle-aged man precipitated by traditional massage therapy—an unusual trigger. The patient experienced mediastinitis, mediastinal abscess, and non-ischemic myocardial injury, highlighting diagnostic and management challenges.

CASE REPORT

A 52-year-old male presented to the emergency department (ED) with continuous dull aching left-sided chest pain and chest wall swelling for four days. Symptoms began after receiving traditional chest massage therapy. He denied trauma, respiratory symptoms, fever, or systemic complaints.

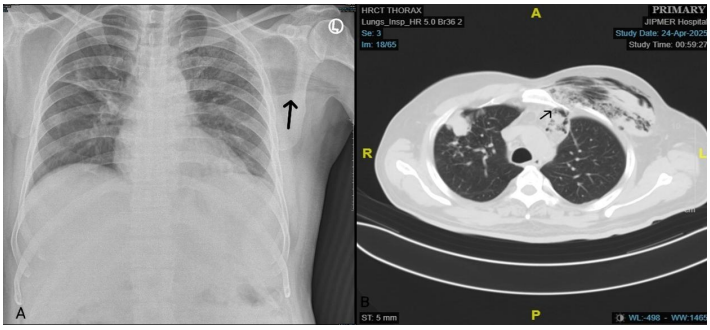


Image 1. A) chest radiograph in posteroanterior view showing the left pectoralis muscle air collection (arrow), ginko leaf sign, and right middle zone fibrotic bands. B) High-resolution computed tomography of the chest in axial section showing pneumomediastinum predominantly over the left side (arrow), with consolidation in the right lung and air collection in the sternoclavicular joint and left pectoralis muscle.

His past history included pulmonary tuberculosis treated with a six-month course of antitubercular therapy three years prior and type two diabetes mellitus managed with oral hypoglycaemic agents. He was a former smoker with a history of alcoholism who had abstained for the prior five years. Family and psychosocial histories were unremarkable.

On physical examination, vital signs were normal except for elevated blood glucose. Oxygen saturation was 98% on room air and respiratory rate 20 breaths per minute. Local inspection revealed an 8 x 6 cm area of edema on the left anterior chest wall extending from the clavicle to the nipple, with palpable crepitus and tenderness. Cardiovascular and respiratory examinations were unremarkable. Electrocardiogram (ECG) showed normal sinus rhythm, complete right bundle branch block, and right axis deviation without dynamic changes on serial ECG.

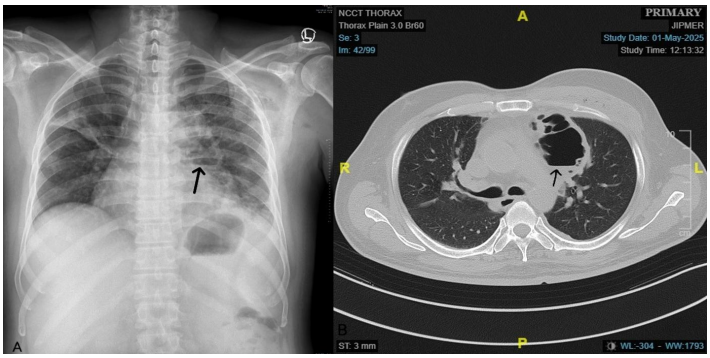


Image 2. A) The chest radiograph in posteroanterior view now demonstrates air-fluid level at the left side of the pneumomediastinum (arrow), suggesting mediastinal abscess and right middle zone fibrotic bands. B) Repeat high-resolution computed tomography in axial view now shows left-sided mediastinal collection with air-fluid level within it.

CPC-EM Capsule

What do we already know about this clinical entity?
Spontaneous pneumomediastinum is a rare, usually benign condition caused by alveolar rupture after raised intrathoracic pressure.

What makes this presentation of disease reportable?
Traditional chest massage triggered pneumomediastinum complicated by mediastinitis, mediastinal abscess, and myocardial injury.

What is the major learning point?
Chest pain with subcutaneous emphysema should prompt evaluation for pneumomediastinum and monitoring for cardiac complications.

How might this improve emergency medicine practice?
Early chest radiograph and electrocardiogram-troponin assessment in suspected pneumomediastinum may prevent delayed, life-threatening complications.

Chest radiograph revealed subcutaneous emphysema over the left pectoralis muscle with the characteristic ginkgo leaf sign and fibrotic bands in the right middle lung zone (Image 1). High-resolution computed tomography (CT) of the thorax confirmed left-sided pneumomediastinum extending into the sternoclavicular joint, manubrium sterni, and pectoralis muscle (Image 2). Patchy consolidations and centrilobular nodules were present bilaterally.

In the ED, the patient was started on analgesics and empirical intravenous (IV) antibiotics. He was admitted under pulmonary medicine and treated with IV clindamycin (600 mg thrice daily), analgesics, and insulin for glycemic control. Multiple skin incisions were done over the subcutaneous emphysema in the left chest wall by the cardiovascular and thoracic surgery team to relieve the subcutaneous emphysema.

On hospital day 3, the patient developed sinus tachycardia with persistent axis deviation. Troponin I was elevated with a value of 93 ng/L (reference range, < 15 ng/L) without significant serial change, suggestive of nonischemic myocardial injury. On hospital day 7, he developed mediastinitis with a mediastinal abscess requiring intubation and open thoracotomy for drainage.

Intraoperative cultures were sterile. The patient was extubated the following day, transitioned to oral antibiotics, and improved clinically. He was discharged in stable condition.

DISCUSSION

Spontaneous pneumomediastinum occurs due to alveolar rupture caused by a sudden rise in intrathoracic pressure, allowing air to track along the bronchovascular sheaths into the mediastinum—a phenomenon known as the Macklin effect.[2,3] Chest pain and subcutaneous emphysema are hallmark signs, although symptoms may vary. The ginkgo leaf sign on chest radiograph and high-resolution CT confirmation are key diagnostic modalities.[5,6] The ginkgo leaf sign is a distinctive radiographic finding in spontaneous pneumomediastinum, seen as a fan-shaped lucency created by air dissecting between the pectoralis major muscle and the chest wall. This appearance resembles the shape of a ginkgo biloba leaf and serves as a useful clue on chest radiograph to identify subcutaneous emphysema and underlying pneumomediastinum.[5] The Hamman sign (a crunching sound synchronous with the heartbeat) may aid in diagnosis but was absent in this case. The patient was thought to have pneumothorax or esophageal rupture, later diagnosed to have spontaneous pneumomediastinum.

While most cases resolve with conservative treatment—oxygen, analgesia, and observation—complications such as mediastinitis, mediastinal abscess, and myocardial injury may occur, particularly in those with pre-existing pulmonary disease.[5] Empirical antibiotics are typically reserved for suspected infections or complications.[8] Asymptomatic patients with stable vitals and no high-risk features can be observed and safely discharged with a follow-up date.

Our patient's spontaneous pneumomediastinum was precipitated by traditional chest massage, likely increasing intrathoracic pressure and causing alveolar rupture. Prior pulmonary tuberculosis may have weakened the lung parenchyma, enhancing susceptibility. The patient received multiple skin incisions for subcutaneous emphysema for decompression, although the literature supports a single incision over the emphysema.[9] Despite early antibiotics, he developed mediastinitis and abscess requiring surgical drainage, a rare but severe complication. The raised troponin I level in our patient, without significant serial rise or fall, likely reflected nonischemic myocardial injury secondary to inflammatory mediastinal involvement rather than acute coronary syndrome. The mediastinitis and local inflammation can cause myocardial irritation or myocarditis-like injury, leading to myocardial cell damage and troponin release.[7]

Spontaneous pneumomediastinum associated with elevated troponin levels should prompt consideration of myocarditis or inflammatory cardiac injury secondary to mediastinal inflammation. Echocardiography or cardiac magnetic resonance imaging can aid in further characterization, although such investigations were deferred in this case due to clinical improvement.

The development of mediastinitis in spontaneous pneumomediastinum is extremely rare but can be fatal if missed. Possible mechanisms include secondary infection of mediastinal air collections or translocation of skin flora during subcutaneous dissection. Persistence of chest pain despite adequate analgesia raised concerns for alternative causes and underlying complications, our case underscores the need for close clinical monitoring and early imaging follow-up even in seemingly benign spontaneous pneumomediastinum.

Atypical triggers such as chest massage should be recognized by clinicians, especially in patients with compromised lung architecture. A multidisciplinary approach involving emergency medicine, pulmonology, and thoracic surgery is vital in complex cases. This case illustrates the importance of prompt diagnosis and multidisciplinary management.

CONCLUSION

Spontaneous pneumomediastinum is a rare yet important cause of chest pain that may follow uncommon triggers such as traditional chest massage. Although often benign, it can lead to serious complications such as mediastinitis and myocardial injury. The presence of spontaneous, symptomatic subcutaneous emphysema warrants emergency physicians to maintain a high index of suspicion and to proceed for advanced imaging. Timely imaging and multidisciplinary intervention are essential for optimal outcomes.

IRB approval was obtained for publication of this case report. Documentation on file.

Address for Correspondence: Naveed Ul Hassan H, MBBS, Department of Emergency Medicine, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry, India. Email: jr8693@jipmer.ac.in.

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Primary Group A Streptococcus Peritonitis in a Healthy Child: A Case Report

Alex Drinnan, MD
James Naprawa, MD
Sunghoon Kim, MD
Karim Mansour, MD

University of California, San Francisco, Benioff Children's Hospital Oakland, Department of Emergency Medicine, Oakland, California

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Introduction: Group A streptococcus (GAS, *Streptococcus pyogenes*) is commonly associated with pharyngitis and skin and soft tissue infections, but invasive infection can occur. Primary bacterial peritonitis due to GAS is rare, especially in healthy children without nephrotic syndrome or immunocompromise.

Case Report: We present a 21-month-old previously healthy male with four days of fever, irritability, and refusal to walk. Examination revealed abdominal distention and peritonitis. Computed tomography showed ascites and a normal appendix. Diagnostic laparoscopy revealed copious purulent peritoneal fluid; both peritoneal fluid and blood cultures were positive for GAS. He was treated with ampicillin-sulbactam followed by oral amoxicillin-clavulanate with complete recovery.

Conclusion: This case highlights a rare presentation of invasive GAS as primary peritonitis in a healthy child. Clinicians should consider this diagnosis when peritonitis is present but imaging does not confirm appendicitis. Early surgical evaluation and targeted antimicrobials are essential. [Clin Pract Cases Emerg Med. 2026;10(4):473–476.]

Keywords: *group A streptococcus; spontaneous bacterial peritonitis; pediatric peritonitis; appendicitis mimic; case report.*

INTRODUCTION

Group A streptococcus (GAS; *Streptococcus pyogenes*) is an aerobic gram-positive coccus responsible for a wide range of clinical diseases.[1–3] Although commonly associated with pharyngitis and superficial skin and soft tissue infections, GAS can also cause serious invasive infections including bacteremia, pneumonia, osteomyelitis, septic arthritis, necrotizing fasciitis, and streptococcal toxic shock syndrome.[1–3] Invasive GAS infection occurs most frequently in young children, particularly those < 2 years of age.[2,3] Primary bacterial peritonitis is uncommon in children, accounting for only 1–3% of cases of acute abdomen.[5] Primary bacterial peritonitis is typically diagnosed in children with underlying comorbidities such as nephrotic syndrome or chronic liver disease.[5–7] In these populations, *S pneumoniae* and *Staphylococcus aureus* are the leading causes.[5–7] In contrast, primary bacterial peritonitis due to GAS in healthy children without predisposing conditions is exceedingly rare, with few pediatric cases described.[9,10] We report a case of primary GAS peritonitis in a previously healthy toddler to highlight diagnostic challenges and emphasize the importance of early surgical and antimicrobial management.

CASE REPORT

A 21-month-old previously healthy male presented with four days of fever, irritability, decreased oral intake, and refusal to walk. On day 1 of illness, he presented to the emergency department (ED) with a peak temperature of 104

°F and generalized shaking suggestive of a febrile seizure. He improved with antipyretics. On physical exam at the initial ED visit, he was alert and playful, and parents reported he was back to baseline. The abdominal examination was benign with no focal tenderness; parents reported the child was moving normally at that time. Rapid test results for severe acute respiratory syndrome coronavirus 2, respiratory syncytial virus, and influenza were negative. The clinical impression was a febrile illness with transient rigors, and the patient was discharged home with return precautions.

Two days later, he was seen by his primary care pediatrician for persistent fever. Parents reported that he also did not want to walk. On exam, he was able to ambulate but had a wide-based gait and possible ataxia. Pediatric neurology was consulted by phone and cerebellitis was considered; clinicians elected observation with referral to neurology if symptoms persisted or worsened. At that visit, lab testing revealed the following: leukocytosis, white blood cell count, 16.4 × 10^9 per liter (L) (reference range, 5–15 × 10^9/L); elevated C-reactive protein, 103 mg/L (< 10 mg/L); hemoglobin, 9.8 g/dL (10.5–13.5 g/dL); sodium, 128 mEq/L (135–145 mEq/L), and albumin 2.7 g/dL (3.5–5.0 g/dL). Given persistent fever and abnormal labs, the patient was referred to the ED for further evaluation.

On return to the ED, the patient appeared ill and uncomfortable. He had multiple episodes of watery, non-bloody diarrhea, and his parents noted that he stated, "everything hurts" when they lifted him. Vital signs were as follows: tachycardia, heart rate, 167 beats per minute; respiratory rate, 23 breaths per minute; temperature, 37.3 °C, and peripheral oxygen saturation, 95% on room air. Abdominal exam was notable for distention, diffuse tenderness, and involuntary guarding; parents had not noticed distention until it was pointed out.

Repeat laboratory evaluation demonstrated persistent leukocytosis with a white blood cell count of 16.1 × 10^9/L) and C-reactive protein level was 376 mg/L, representing a marked elevation. Serum sodium was 134 mEq/L; albumin, 3.4 g/dL; and lactate 1.0 mmol/L (0.5–2.0 mmol/L). Abdominal ultrasound showed a small to moderate volume of complex free fluid predominantly in the lower abdomen; the appendix was not visualized. Computed tomography of the abdomen and pelvis with contrast demonstrated moderate ascites with a normal-appearing appendix and no periappendiceal inflammation.

Given concern for peritonitis, empiric ceftriaxone and metronidazole were administered, and pediatric surgery was consulted. The pediatric surgery attending examined the patient and recommended urgent diagnostic laparoscopy. Approximately 100 mL of purulent fluid was evacuated upon entering the peritoneal cavity. The appendix was grossly unremarkable aside from subtle tip inflammation without perforation. An appendectomy was performed and purulent fluid was aspirated for culture.

CPC-EM Capsule

What do we already know about this clinical entity?
Primary bacterial peritonitis is rare in healthy children and usually occurs in those with nephrotic syndrome or liver disease.

What makes this presentation of disease reportable?
Group A streptococcus caused primary peritonitis in a healthy toddler, mimicking appendicitis without perforation.

What is the major learning point?
Ascites with a normal appendix in a febrile child should prompt consideration of primary group A streptococcus peritonitis.

How might this improve emergency medicine practice?
Early recognition and surgical consultation allow targeted antibiotics and prevent delayed treatment.

Pathology showed mild acute inflammation of the appendiceal tip without perforation. Gram stain demonstrated gram-positive cocci on the external surface of the appendix. Peritoneal fluid culture and blood culture both grew beta-hemolytic group A streptococcus. Empiric ceftriaxone and metronidazole were transitioned to ampicillin-sulbactam, and the patient improved rapidly. He completed a 10-day course of antibiotics (intravenous, followed by oral amoxicillin-clavulanate) and was discharged on hospital day 5. At follow-up two days after discharge the patient was afebrile, playful, tolerating oral intake, and without abdominal symptoms.

DISCUSSION

This case represents an unusual instance of primary GAS peritonitis in a healthy child with no predisposing risk factors. Peritonitis in the pediatric population is most often secondary to appendicitis, intestinal perforation, or prior intra-abdominal surgery. When imaging demonstrates free fluid but no evidence of appendiceal rupture or other intra-abdominal source, the diagnosis becomes less straightforward. Primary peritonitis, defined as infection of the peritoneal cavity without an evident intra-abdominal source, is rare in children without nephrotic syndrome or liver disease.[5-7]

Appendicitis is the most frequent cause of pediatric

peritonitis and is particularly challenging to diagnose in children < 5 years of age, in whom classic symptoms may be absent; up to 57% of cases in this group may present with perforation.[8] Our patient's imaging demonstrated ascites with a normal appendix, and surgical findings and pathology confirmed absence of perforation, making primary peritonitis the most likely diagnosis.

The pathogenesis of primary GAS peritonitis is not fully understood. Hematogenous spread from an unrecognized primary site, such as pharyngeal or cutaneous colonization, is the most accepted mechanism, with transient bacteremia after mucosal disruption allowing seeding of the peritoneum. Group A streptococcus virulence factors, including superantigens and exotoxins, likely contribute to invasive disease.[4] In many reported cases, no clear antecedent focus is identified, as in our patient.

The existing literature includes only a small number of pediatric cases of primary GAS peritonitis.[9,10] Most describe a sudden, severe presentation with abdominal pain, diffuse peritonitis, and, in some instances, hemodynamic instability requiring intensive care management.[9] Dann et al reported two children presenting with abdominal pain and ascites in whom GAS was isolated from peritoneal fluid despite near-normal appendices.[9] Sharp et al identified only 28 pediatric cases reported since 1975, underscoring the rarity of this diagnosis.[10] Compared with prior reports, our patient had a more indolent course, suggesting that GAS peritonitis may present across a broader clinical spectrum than previously recognized.

Recognition of this entity is critical because GAS peritonitis may closely mimic perforated appendicitis. Clinicians should consider GAS when imaging demonstrates ascites without clear appendiceal inflammation, in the setting of systemic illness (eg, high fever and markedly elevated inflammatory markers). While empiric therapy for suspected perforated appendicitis often provides broad coverage, definitive therapy for GAS relies on penicillin-class agents once the organism is identified. Early differentiation between perforated appendicitis and alternative causes of pediatric peritonitis is critical in emergency practice. Key distinguishing clinical and imaging features are summarized in the table.

The patient's initial ED visit was notable for a benign abdominal examination and transient clinical improvement after antipyretics; at that time, discharge with clear return precautions was a reasonable course of action. Pediatric illness is dynamic, particularly in preverbal children, and early examinations may not reveal evolving intra-abdominal pathology. This case highlights the importance of reassessment and low threshold to re-evaluate and obtain further testing when parents report new or worsening signs such as reluctance to ambulate, persistent high fever, or poor oral intake.

Difficulty ambulating has a broad differential in young children; abdominal pain may be an overlooked cause. Abdominal exams in preverbal children are challenging because these patients often cannot cooperate; paradoxically, peritoneal irritation may sometimes make the exam easier because the child resists movement. In our case, reluctance to ambulate likely represented early peritoneal irritation that evolved over days.

Multidisciplinary care involving emergency medicine, pediatric surgery, pathology, and infectious diseases facilitated timely diagnosis and effective management. Early attending-to-attending discussion and prompt intraoperative sampling were key to obtaining the microbiologic diagnosis and narrowing antimicrobial therapy.

LIMITATIONS

This case report has several limitations. First, while primary GAS peritonitis is rare, it has been previously reported; therefore, this case does not represent a novel disease entity or a new diagnostic technique. Second, this was a single retrospective case with limited generalizability.

Table. Distinguishing features of pediatric peritonitis.

Feature	Acute appendicitis (± perforation)	Primary GAS peritonitis	Other etiologies (SBP, pneumonia, UTI, viral illness)
Typical age	Any age (↑ perforation risk <5 yrs)	Young children (rare)	Variable
Fever	Low early; higher with perforated	High early	Variable
Pain pattern	RLQ → diffuse if ruptured	Diffuse	Variable
Refusal to walk	Possible, especially if perforated	May occur early	Uncommon
Imaging findings	Dilated appendix, periappendiceal fat stranding ± small fluid	Moderate/free ascites with normal appendix	Variable
Blood cultures	Rare	May be positive	Rare
Mechanism	Luminal obstruction → perforation	Hematogenous spread	Varies

GAS, group A streptococcal; *SBP,* spontaneous bacterial peritonitis; *UTI,* urinary tract infection; *RLQ,* right lower quadrant; *yrs,* years.

Third, although hematogenous spread is the most plausible mechanism, the exact source of bacteremia was not identified. Finally, reflection on the initial decision to discharge is retrospective and may not capture all clinical factors considered at the time.

CONCLUSION

Primary group A streptococcal peritonitis is a rare diagnosis in otherwise healthy children, but it carries important implications for emergency and surgical management. When children present with fever and peritonitis and imaging demonstrates ascites without evidence of appendiceal perforation, clinicians should consider this entity. Prompt recognition, early surgical consultation, and targeted antimicrobial therapy result in favorable outcomes.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Address for Correspondence: Karim Mansour, MD, UCSF Benioff Children's Hospital Oakland, Department of Emergency Medicine, 747 52nd Street, Oakland, CA 94609. Email: Karim.Mansour@ucsf.edu.

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From Pain to Clarity: A Case of Pneumoperitoneum Diagnosed by Point-of-care Ultrasound

Archana Nair, MD
Soubhik Chakraborty, MBBS
Tej Prakash Sinha, MS
Sanjeev Bhoi, MD

All India Institute of Medical Sciences, Department of Emergency Medicine, New Delhi, India

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Introduction: Acute abdomen is a common emergency presentation, and gastrointestinal perforation can be rapidly fatal if not promptly identified. Point-of-care ultrasound (POCUS) offers a rapid bedside alternative when advanced imaging is delayed or unavailable.

Case Report: We report a 40-year-old man presenting in cardiac arrest, resuscitated after ten minutes, with subsequent findings of abdominal distension and prior epigastric pain. Point-of-care ultrasound demonstrated classic signs of pneumoperitoneum, and intestinal perforation was confirmed by the drainage of feculent fluid. Despite intervention, the patient expired.

Conclusion: This case underscores the value of POCUS in diagnosing perforation in unstable patients, guiding emergent decisions when transfer or definitive imaging is not feasible. [Clin Pract Cases Emerg Med. 2026;10(4):477–480.]

Keywords: *point-of-care ultrasound; intestinal perforation; pneumoperitoneum; case report*.

INTRODUCTION

Undifferentiated abdominal pain is a commonly encountered emergency. It requires prompt differentiation between medical and surgical causes. While initial evaluation is guided by history and physical examination, establishing a definitive diagnosis in patients with abdominal pain generally requires adjunctive laboratory and imaging studies. Increasingly, point-of-care ultrasound (POCUS) is being recognized as an effective, rapid diagnostic modality capable of identifying life-threatening intra-abdominal conditions, reducing reliance on radiologic imaging, and expediting clinical decision-making. The sonographic evaluation for pneumoperitoneum using POCUS is relatively straightforward but highly dependent on the operator's proficiency. This case review illustrates the clinical utility of abdominal POCUS in diagnosing pneumoperitoneum and supports its broader integration into the evaluation of acute abdominal presentations. The integration of multiple classic sonographic signs- including enhanced peritoneal stripe sign, abdominal A-lines, absent sliding, and the gut point, enabled rapid bedside diagnosis and directly guided emergent intervention. This case reinforces the value of POCUS not only as a diagnostic modality but also as a critical decision-making tool in time-sensitive, resource-limited scenarios. Furthermore, it highlights the importance of considering intra-abdominal causes in non-traumatic cardiac arrest, an area that may be under-recognized in emergency practice.

CASE REPORT

A 40-year-old male with no prior comorbidities presented to the emergency department in cardiac arrest. Following advanced cardiac life support, return of spontaneous circulation was achieved after approximately 10 minutes. Post-resuscitation care was initiated. Detailed clinical history revealed epigastric pain two days prior. On examination, his abdomen was distended and rigid.

Point-of-care ultrasound was performed and demonstrated the following key findings: An enhanced peritoneal stripe

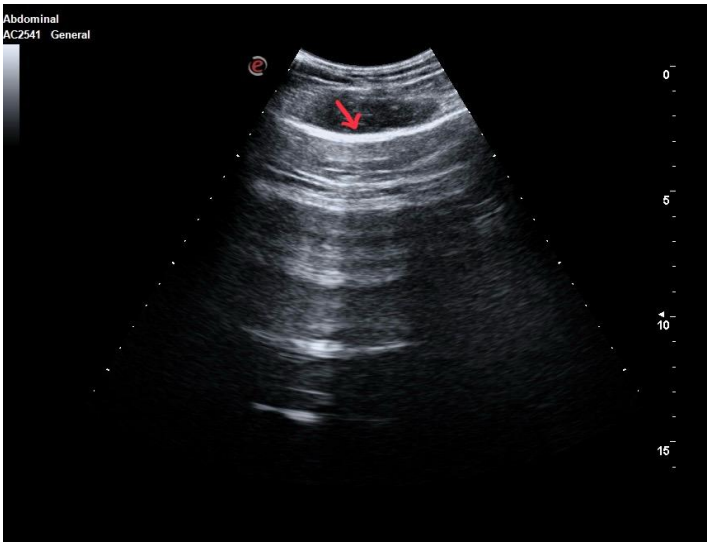


Image 1. Enhanced peritoneal stripe sign (arrow) in a patient with acute abdominal pain indicative of free peritoneal air.

sign (Image 1), absent gut sliding (gut barcode sign) (Image 2), abdominal Aprofile (Image 3) and free fluid containing echogenic particulate matter (Image 4), all consistent with intestinal perforation with pneumoperitoneum.

Subsequent formal abdominal ultrasound confirmed pneumoperitoneum, most notably via the enhanced peritoneal stripe sign—a hyper-echoic thickening of the peritoneal stripe indicative of free intraperitoneal air.[1]

Due to hemodynamic instability requiring dual vasopressor support, the patient was deemed unfit for transfer to the operating theatre for exploratory laparotomy. Ultrasound findings prompted immediate bedside intervention with a 22 French drain, yielding approximately 2500 mL feculent fluid—a temporizing measure given the inability to reach operation theatre. Abdominal radiography corroborated the ultrasound findings, demonstrating subdiaphragmatic free air and dilated bowel loops (Image 5).

The patient was started on broad spectrum antibiotic coverage and other supportive measures. Despite aggressive resuscitation patient expired. This case emphasizes the importance of point-of-care ultrasound in diagnosing a perforation peritonitis and pneumoperitoneum.

CPC-EM Capsule

What do we already know about this clinical entity?
Pneumoperitoneum from gastrointestinal perforation is life-threatening.

What makes this presentation of disease reportable?
Point-of-care ultrasound (POCUS) enabled a rapid diagnosis in an unstable patient, enabling bedside drainage when operating room transfer was impossible.

What is the major learning point?
Integrate multiple POCUS signs—E-point septal separation, A-lines, gut point, and absent sliding— to diagnose pneumoperitoneum in unstable patients before imaging.

How might this improve emergency medicine practice?
Routine POCUS for acute abdomen can expedite diagnosis of perforation, guide bedside intervention, and reduce delays when transfer is not feasible.

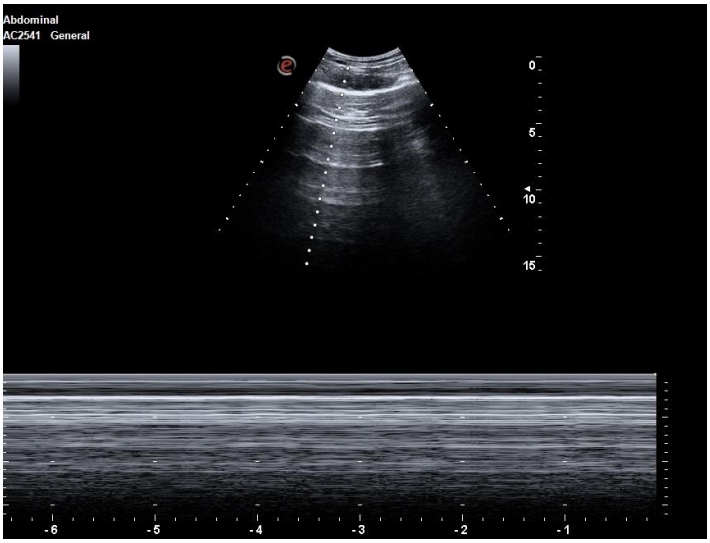


Image 2. Absent gut sliding (gut barcode sign) in M mode in a patient with acute abdominal pain signifying the presence of free air.

DISCUSSION

Abdominal ultrasound is performed with a low frequency abdominal probe. It is positioned horizontally with pointer towards right side of the patient. Numerous parallel, equidistant, horizontal hyperechoic artifacts can be seen extending down the image, with an absence of identifiable abdominal structures. These are similar to the reverberation artefacts of pleura in lung ultrasound, which are called as A lines. Therefore, these reverberation artefacts due to presence

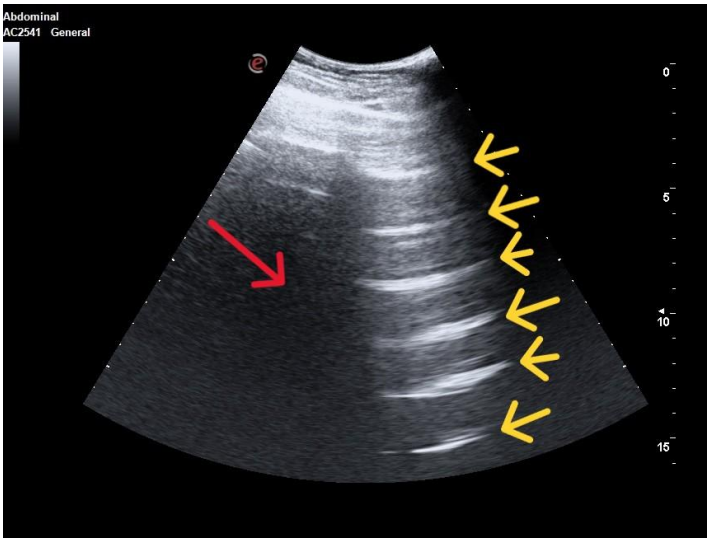


Image 3. Abdominal A profile with right half not showing A lines because of the liver. Red arrow shows liver and yellow arrows shows A lines, in a patient with acute abdominal pain suggesting pneumoperitoneum.

of pneumoperitoneum are called abdominal A lines.[2]

A highly echogenic peritoneal stripe is evident at the junction between the parietal peritoneum and intraperitoneal free air. This appearance is due to the marked acoustic impedance mismatch between soft tissue and free intraperitoneal air, which causes near-total reflection of ultrasound waves at the peritoneal interface, resulting in a brightly echogenic stripe and associated reverberation

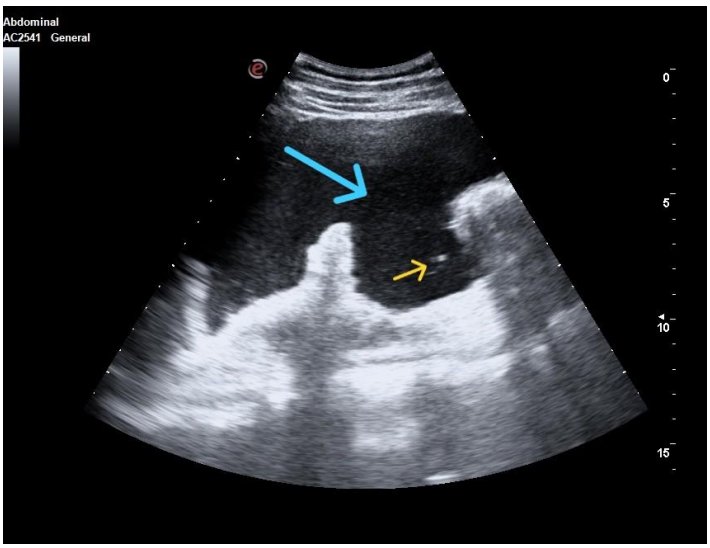


Image 4. Free fluid containing echogenic particulate matter. Blue arrow shows the free fluid and yellow arrow shows particulate matter, in a patient with acute abdominal pain, indicative of intestinal perforation.

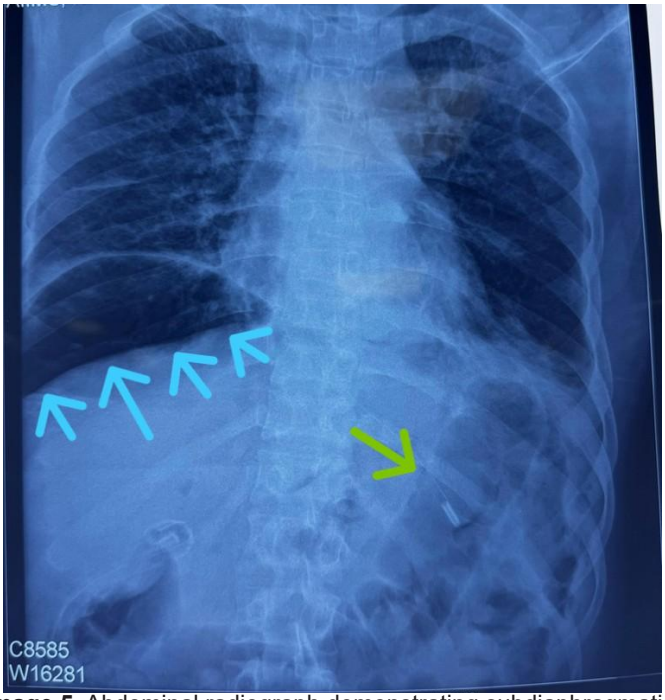


Image 5. Abdominal radiograph demonstrating subdiaphragmatic free air and dilated bowel loops in a patient with acute abdominal pain. Blue arrows show air under diaphragm and green arrow shows dilated bowel.

artifacts. Multiple repeating horizontal artefacts, corresponding to reverberation of the peritoneal line due to free intra-abdominal air, are present. The 'gut point' refers to the transition zone where the sliding motion of the parietal peritoneum ceases and these reverberation artefacts begin to appear. These ultrasound findings closely resemble those seen in thoracic air leaks, such as the presence of A-lines and the absence of pleural sliding on lung ultrasound.[3] Abdominal free air produces a strong linear echo with a pathognomonic peritoneal stripe sign.

Gut point is analoguous to lung point of pneumothorax.[4] A diagnostic hallmark is the identification of the "gut point," which represents the interface between dynamic abdominal wall sliding and a static, non-sliding area, signifying the presence of free air. Normally, a subtle shimmering or sliding movement along the peritoneal line signifies the normal apposition of the visceral and parietal peritoneum. The abnormal accumulation of free intraperitoneal air disrupts this interface, resulting in the separation of these peritoneal layers.

Despite its utility, POCUS for pneumoperitoneum is subject to potential pitfalls. Intraluminal bowel gas can mimic free intraperitoneal air by producing echogenic interfaces and reverberation artifacts, potentially leading to false-positive interpretations. However, in such cases, the echogenic line typically corresponds to the bowel wall and is associated with an overlying hypoechoic bowel wall layer and visible

peristalsis. In contrast, free intraperitoneal air accumulates immediately beneath the anterior abdominal wall, producing an enhanced peritoneal stripe without an intervening hypoechoic layer and often demonstrating positional changes with patient movement. Careful attention to probe positioning, dynamic assessment, and identification of the "gut point" can further improve diagnostic accuracy and reduce misinterpretation.

CONCLUSION

This case illustrates the power of pointofcare ultrasound in early detection of perforation peritonitis and pneumoperitoneum. Computed tomography remains gold standard, but POCUS offers rapid, nontransfer-dependent bedside diagnosis—critical in unstable patients. Given its excellent sensitivity and specificity, affordability, and favorable safety profile, abdominal ultrasound ought to be regarded as the initial imaging choice for detecting pneumoperitoneum and should be broadly adopted by emergency medicine physicians.

Patient consent has been obtained for publication of this case report. Documentation on file.

Address for Correspondence: Archana Nair, MD, All India Institute of Medical Sciences, Department of Emergency Medicine, AIIMS, New Delhi, India 110029. Email: dr.archananair10392@gmail.com.

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Case Report of Orthopedic Screw Migration Presenting as Abdominal Pain in the Emergency Department

Eric Jones, DO*
Marium Khan, BS†
Emmelyn J. Samones, CCRP*
Paul Brian Savino, MD*

*Loma Linda University Medical Center, Department of Emergency Medicine, Loma Linda, California
†Loma Linda University Health School of Medicine, Loma Linda, California

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Introduction: Hip fractures are a concern in older adults, with incidence expected to rise as the population ages. Despite advances in surgical technique and hardware design, complications remain, influenced by patient factors and surgical choices. Rarely, hardware migration into the pelvis occurs, which poses serious risks. In the emergency setting, abdominal pain is a common complaint, and when evaluating patients with complex histories or prior orthopedic surgeries, clinicians must maintain a broad differential. This case details the discovery of a medially displaced hip screw into the pelvis as the cause of abdominal pain in a patient in the emergency department (ED).

Case Report: A 44-year-old female with end stage renal disease on hemodialysis, anemia, and osteoporosis presented to the ED with sudden-onset, bilateral lower quadrant abdominal pain, vomiting, and diarrhea. The patient reported undergoing left hip open reduction and internal fixation seven months prior. Imaging of the abdomen noted medial displacement of the cephalomedullary nail through the left acetabulum into the pelvis with associated extraperitoneal hematoma. She underwent hardware removal and exploratory laparotomy without bowel injury.

Conclusion: Orthopedic screw migration is a rare but serious complication, with limited documentation in the literature. Risk increases in patients with poor bone quality and healing, often due to chronic conditions. In patients with multiple comorbidities, careful history and a thorough exam when presenting to the ED are critical to identifying complications. Strategic use of imaging and clinical judgement can help emergency physicians refine their differential diagnosis. [Clin Pract Cases Emerg Med. 2026;10(4):481–484.]

Keywords: *abdominal pain; hip fracture; open reduction and internal fixation (ORIF); orthopedic screw migration; case report.*

INTRODUCTION

Hip fractures remain a major public health concern and frequent presenting chief complaint in the emergency department (ED). In 2019, there were approximately 30,000 hip fracture-related ED visits in the United States among adults ≥ 65 years, a number that is anticipated to rise significantly with the continued aging of the population.[1] Despite advances in surgical techniques and hardware design, complications remain common and often multifactorial in origin, with patient age, comorbidities, and procedural complications leading patients to return to the ED.[2]

Hardware migration following open reduction and internal fixation of the hip is a recognized complication; however, intrapelvic migration causing vascular injury or hematoma formation is rare and poses a significant diagnostic challenge. These patients often present with nonspecific abdominal or pelvic pain that mimics common conditions such as gastroenteritis, appendicitis, or urinary tract infection, and

may not associate their remote surgical history with their current symptoms. Delayed recognition can result in life-threatening hemorrhage.

In this case report, we present a rare instance of abdominal pain initially attributed to food poisoning but found to be secondary to hardware failure involving medial displacement of a hip screw from an open reduction and internal fixation causing an intrapelvic hematoma. This case highlights the importance of maintaining a high index of suspicion and strong diagnostic vigilance when evaluating potential postoperative complications in the ED.

CPC-EM Capsule

What do we already know about this clinical entity?
Patients presenting to the emergency department with abdominal pain have a broad differential diagnosis.

What makes this presentation of disease reportable?
This patient presented with a rare case of an orthopedic hip screw that had migrated into the pelvis as the source of abdominal pain.

What is the major learning point?
A thorough history and physical exam, combined with a broad differential, can uncover rare diagnoses.

How might this improve emergency medicine practice?
It is important for emergency physicians to consider extra-abdominal sources as a potential cause of abdominal pain, especially in patients with comorbidities.

CASE REPORT

A 44-year-old female with a past medical history significant for end-stage renal disease on hemodialysis with anemia and osteoporosis presented to the ED with sudden onset of bilateral lower quadrant abdominal pain for two hours associated with nonbloody vomiting and diarrhea. The patient stated that the pain began suddenly while she was driving and initially attributed the pain to a burrito she had eaten the night before. Her past surgical history included a surgical repair of a left hip fracture seven months prior, complicated by continued pain and need for a wheelchair since the procedure.

Initial vital signs were as follows: temperature, 99.1 °F; pulse, 82 beats per minute; blood pressure, 169/94 mm Hg; respiratory rate, 16 breaths per minute; and oxygen saturation, 94% on room air. On physical examination, the patient was found to be in distress. The abdominal exam revealed severe tenderness to palpation of the bilateral lower quadrants with noted distension, but no rebound. The upper abdomen was noted to be soft and nontender to palpation. She was also found to have significant tenderness to palpation of the left hip with severely limited range of motion secondary to pain. The patient was unable to bear weight due to severe bilateral hip pain, which was worse on the left compared to the right. Lab results were notable for a hemoglobin of 8.7 g/dL (reference range, 10.5-15 g/dL) and alkaline phosphatase of 1,196 U/L (37-132 U/L). Elevated alkaline phosphatase has been shown to be associated with both subacute fracture and bone remodeling after injury, which we suspected was the case here.[3]

Radiograph of the left hip was obtained due to left hip exam findings, which revealed postsurgical changes of open reduction and internal fixation with displacement of a cephalomedullary nail through the left acetabulum found to terminate within the pelvis (Image 1). Computed tomography (CT) of the abdomen and pelvis with contrast revealed similar findings with pelvis lesion consistent with an extraperitoneal hematoma. Orthopedic surgery and general surgery were consulted and recommended CT angiography (CTA) of the abdomen and pelvis to rule out arterial injury with ongoing bleeding. The CTA confirmed displacement of the cephalomedullary nail and associated pelvis lesion consistent with an extraperitoneal hematoma and possible venous bleed without arterial extravasation (Image 2).

The patient was administered hydromorphone and morphine for acute pain relief, as well as trimethobenzamide for nausea. She was admitted for surgical repair to both general and orthopedic surgery, where removal of hardware and exploratory laparotomy with abdominal washout was performed. She was noted to have poor bone quality with associated lack of fixation of prior fracture and no presence of bowel injury. The patient's hospital course was complicated by a right femoral neck stress fracture discovered on day 10 and periprosthetic right femur fracture identified on day 19, requiring additional revisions. She was discharged home after a 23-day hospitalization with orthopedic surgery follow-up.

DISCUSSION

Orthopedic screw migration following hip fracture fixation is a rare but life-threatening complication that poses diagnostic challenges in the ED. While medial screw migration is not uncommon and has been described in the literature, intrapelvic migration of the hardware resulting in vascular injury is rare. Its clinical presentation is often

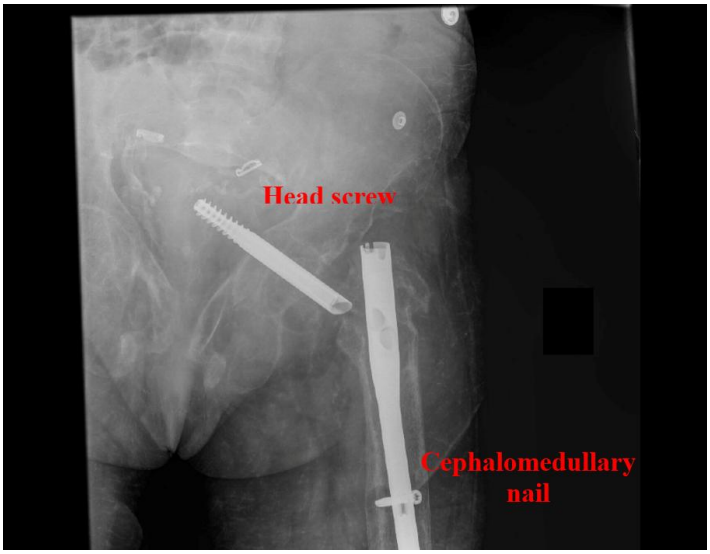


Image 1. Anteroposterior radiograph of the left hip revealed postsurgical changes of open reduction internal fixation with displacement of cephalomedullary nail through the left acetabulum into the pelvis.

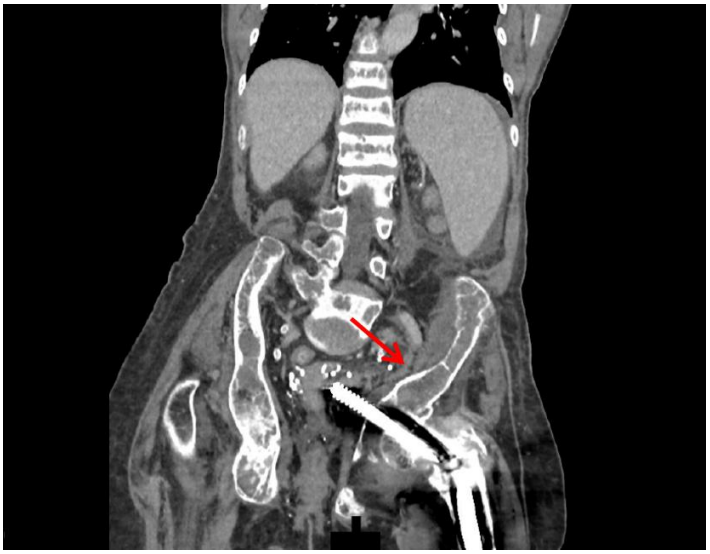


Image 2. A coronal computed tomography angiogram of the abdomen and pelvis revealed postsurgical changes of left hip open reduction internal fixation with displacement of cephalomedullary nail through the left acetabulum into the pelvis with associated mixed density lesion suggestive of an extraperitoneal hematoma (arrow).

nonspecific and may mimic common gastrointestinal and gynecologic conditions.[4,5]

This case emphasizes several critical teaching points for emergency physicians. Any patient with a history of hip/pelvic hardware presenting with acute or acute-on-chronic abdominal or pelvic pain should prompt consideration of hardware-related complications in the differential diagnosis, regardless of how benign the presentation appears, or the time elapsed since surgery. Hardware migration may present with vague, nonspecific symptoms and normal vital signs due to a slowly progressive hematoma or hardware tamponade. Hardware migration may present along a clinical spectrum before catastrophic intrapelvic displacement occurs. Earlier signs include chronic hip or groin pain, decreased range of motion, pain with weight-bearing, or limb-length discrepancy, which are symptoms that may prompt repeat imaging and orthopedic referral before acute decompensation.[6] Furthermore, this patient's wheelchair dependence following open reduction and internal fixation, along with risk factors such as female sex and chronic kidney disease contributing to poor bone quality, warranted further evaluation of hardware placement.

Contrast-enhanced CT of the abdomen and pelvis is the diagnostic test of choice in a stable patient with acute-onset abdominal pain and history of pelvic hardware placement, as it allows for precise localization of possible complication/hardware migration, identification of contrast extravasation indicating arterial injury, and estimation of hematoma volume.[7] While artifact secondary to hardware is a consideration, CT still provides critical diagnostic information regarding major pathology.[8]

Once intrapelvic hardware migration has been identified, ED management focuses on stabilization and preparation for definitive surgical intervention. Patients should be kept nonweight-bearing and immobilized to prevent further hardware displacement or vascular injury. In patients with signs of hemorrhagic shock, resuscitation should be initiated immediately with consideration of activation of a massive transfusion protocol for persistent shock, as delays are associated with increased mortality.[9] If imaging demonstrates hardware proximity to bowel, free fluid, or free air concerning for perforation, empiric broad-spectrum antibiotics with Gram-negative and anaerobic coverage should be initiated.[10]

Management requires immediate multidisciplinary consultation with orthopedic surgery, trauma surgery, vascular surgery, and interventional radiology. Given the complexity of these cases and the need for multiple surgical subspecialties, definitive management typically requires transfer to a tertiary or quaternary care center. In this case, the combination of poor bone quality and intrapelvic migration necessitated explorative laparotomy for safe hardware extraction. Persistent or worsening pain after open reduction and internal fixation, particularly in patients who remain nonambulatory beyond expected recovery timelines, should raise concern for potential hardware complications in the ED.

CONCLUSION

Migration of orthopedic hardware should be considered in

ED patients with a history of prior fixation who present with new or persistent pain. In this case, the patient's acute abdominal pain after eating a burrito—a seemingly benign trigger— could have led to diagnostic anchoring on a gastrointestinal cause despite the history of open reduction and internal fixation. Given the variable presentations of hardware migration, careful clinical judgment, thorough physical examination, and detailed history-taking remain essential especially when presenting to the ED. This case reinforces that emergency physicians must obtain a detailed surgical history in all patients with abdominal or pelvic pain and maintain a low threshold for contrast-enhanced CT imaging when prior hardware is present. Early recognition and prompt multidisciplinary involvement are critical to preventing the morbidity and mortality associated with this rare but serious complication.

The authors attest that their institution does not require Institutional Review Board approval. Patient consent has been obtained for publication of this case report. Documentation on file.

Address for Correspondence: Emmelyn J. Samones, CCRP, Loma Linda University Medical Center, Department of Emergency Medicine, 11234 Anderson St., Room A890A, Loma Linda, CA 92354. Email: esamones@llu.edu.

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Status Epilepticus in a Patient Prescribed Theophylline: A Case Report

Cynthia Coyle, DO
Emmelyn J. Samones, CCRP
Tammy Phan, BS, CCRP
Mindi Guptill, MD

Loma Linda University Medical Center, Department of Emergency Medicine, Loma Linda, California

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Introduction: Seizures are a frequent reason for emergency department (ED) evaluation, and new-onset events carry a wide differential in which medication toxicity can be overlooked. Toxicity from seldomly prescribed drugs, such as theophylline, can be particularly challenging to recognize because they can cause nonspecific adverse effects, including palpitations, nausea, vomiting, and seizures. Although theophylline use was widespread in the 1980s, current prescribing patterns are unknown. Nevertheless, theophylline toxicity continues to result in ED visits and poison center calls. Because adverse effects may be acute or chronic, identifying theophylline toxicity remains difficult.

Case Report: A 74-year-old woman presented to the ED with status epilepticus. After thorough history and extensive medical work-up, medication toxicity was suspected as the cause of her seizures. Upon further investigation, the patient was found to have theophylline toxicity. Prompt treatment was initiated resulting in cessation of seizures, highlighting the importance of considering medication toxicity in geriatric patients with new-onset seizures.

Conclusion: Theophylline toxicity can present with vague, multisystem symptoms, with status epilepticus being one of its most severe complications. Rapid identification of the underlying toxic etiology is essential to guide treatment and stop the seizures. Moreover, clinical suspicion should be elevated in the geriatric population related to frequent polypharmacy. In this case, confirming the medication history with a family member raised concerns for medication misuse, enabling timely intervention. Although rarely prescribed, physicians should counsel patients on the risks and adverse effects of theophylline and emphasize close monitoring. [Clin Pract Cases Emerg Med. 2026;10(4):485–488.]

Keywords: *seizure; theophylline; toxicity; status epilepticus; case report.*

INTRODUCTION

New-onset seizure is a common emergency department (ED) presentation, and the differential is broad and varied.[1] While head trauma, hypoglycemia, metabolic derangements, and infection are common culprits, medication toxicity can be overlooked.[2] Toxicity secondary to infrequently prescribed drugs remains a difficult diagnosis. A study done in the ED found that of new-onset seizures presenting to the ED, 6.1% were medication related.[3] Theophylline was once among the top 20 most prescribed drugs with eleven million annual prescriptions in the 1980s.[3] However, usage has sharply declined, and no data are currently available on the prescription frequency for theophylline.[4] While considered an outdated medication and rarely prescribed, theophylline toxicity still accounts for ED visits and poison center calls. In 2022 and 2023, 244 U.S. poison center cases involved theophylline with one death each year attributed to the medication.[5,6] In geriatric patient

populations in particular, medication side effects should always be considered, and a thorough medication review should be performed.

CASE REPORT

A 74-year-old female with past medical history of chronic obstructive pulmonary disease, atrial fibrillation, and heart failure with reduced ejection fraction presented via emergency medical services (EMS) with altered mental status. The patient's spouse reported decreased responsiveness for approximately five hours prior to presentation. At home, the patient experienced five seizures without return to baseline prompting the spouse to call EMS. Emergency medical services noted the patient having active tonic-clonic seizures during their evaluation and administered two doses of midazolam 2.5 mg intravenously (IV). Upon arrival to the ED, the patient was actively seizing. Vitals obtained upon arrival revealed the patient to be hypertensive to 166/87 mm Hg, tachycardic to 151 beats per minute, and saturating well on supplemental oxygen via nasal cannula. On physical exam, there was no external evidence of trauma or injury.

The patient received two doses of lorazepam 4 mg IV and levetiracetam loading dose (4,200 mg) IV without resolution in seizure activity. The patient was intubated for airway protection and started on a propofol infusion. A point-of-care glucose obtained upon arrival was negative for hypoglycemia. A full medical work-up was notable for a negative urine drug screen with the exception of benzodiazepines (likely related to medication given by EMS and in the ED), and no emergent findings on complete blood count, creatine kinase, comprehensive metabolic panel, troponins, lactate, or urinalysis. A computed tomography of the head was without evidence of acute intracranial abnormality.

On review of her medication list, the patient was noted to be on multiple medications, including digoxin and theophylline, raising concerns for medication toxicity as the cause of her seizures. Therefore, digoxin and theophylline levels were obtained. The digoxin level was undetectable at < 0.4 ng/mL (therapeutic range 0.5-1.0 ng/mL) and the theophylline level was elevated at 21.5 µg/mL (8-16 µg/mL; toxic range > 20 µg/mL). At that time, the working diagnosis was theophylline toxicity as the cause of her seizures.

Further history obtained from the patient's husband revealed that approximately three weeks prior she had been evaluated in the ED for altered mentation and new-onset seizure activity. She had a witnessed seizure in the ED at that visit. She was given IV levetiracetam in the ED and admitted to the neurology service for new-onset seizures. During that admission, video electroencephalogram did not show evidence of seizure activity. She was subsequently discharged home with a prescription for levetiracetam 1,500 mg per os twice a day.

CPC-EM Capsule

What do we already know about this clinical entity?
The pharmacological agent theophylline has a narrow therapeutic index, and toxicity can cause severe complications, including status epilepticus.

What makes this presentation of disease reportable?
This case is an uncommon modern presentation of status epilepticus secondary to theophylline toxicity in a geriatric patient.

What is the major learning point?
Medication toxicity, including from infrequently prescribed drugs, should remain on the differential for seizures, especially in the geriatric population.

How might this improve emergency medicine practice?
This case highlights the need to obtain a thorough history, including medication reconciliation, to identify toxic etiologies and guide timely, targeted treatment.

The husband also revealed that the patient was taking greater than her prescribed amount of theophylline 300 mg twice a day for the prior month. It was unclear how much the patient was taking; however, the patient's husband recalled the patient doubling or tripling her dose due to a cough she had for the prior month. Poison control was consulted and recommended activated charcoal 1 g/kg with serial theophylline levels every four hours (Figure). The patient was admitted to the neurocritical care unit for status epilepticus.

After admission to the neurocritical care unit, electroencephalogram was initiated and IV levetiracetam was continued. Video electroencephalogram showed diffuse background slowing but no seizures or epileptiform discharges. During her 1.5 month hospitalization, the patient developed multiple complications including gastrointestinal (GI) bleeding and atrial fibrillation with rapid ventricular response. During her hospital course, she had issues with secretion management and dysphagia. She required recurrent intubations for airway protection for altered mentation. The patient's altered mentation was thought to be multifactorial but was not suspected to be secondary to waxing and waning methylxanthine toxicity. The patient was ultimately discharged home on hospice from the intensive care unit.

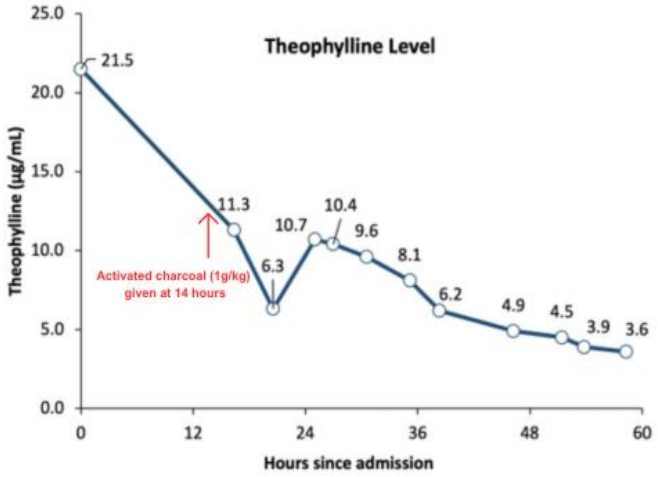


Figure. Graph of serial theophylline levels in a 74-year-old female with theophylline toxicity, measured every four hours over a three-day hospital admission.
µg, micrograms; *mL*, milliliter; *g*, grams; *kg*, kilograms.

DISCUSSION

Theophylline was originally developed in 1922 as a treatment for asthma due to its effect of bronchial dilation and smooth muscle relaxation through β2- adrenergic agonism and increased intracellular cyclic adenosine monophosphate.[7,8] Theophylline works through two separate mechanisms of action; it is both a nonselective adenosine receptor antagonist and a competitive nonselective phosphodiesterase inhibitor, leading to the adverse effects on various systems outside the respiratory system. Theophylline has a narrow therapeutic window and when levels become toxic, most commonly with plasma level > 20 mg/L, adverse effects on the central nervous system, GI system, and the cardiovascular system can occur. Nonspecific symptoms such as GI distress have also been attributed to chronic theophylline overdose, such as the case of a 69-year-old man with chronic obstructive pulmonary disease who presented for medical care with chronic nausea and vomiting. Following an extensive but unrevealing GI evaluation, symptoms were attributed to theophylline toxicity after he was found to have a level of 28.9 µg/mL.[9]

Treatment for theophylline toxicity includes activated charcoal per os or via nasogastric tub. Initial seizure treatment with benzodiazepines is appropriate; however, it may be insufficient due to theophylline's adenosine antagonism. In the case of refractory seizures, escalation to phenobarbital or propofol infusion may be required.[8] Hemodialysis is indicated in the unstable patient with life-threatening arrhythmias or seizures. Hemodialysis may also be necessary for theophylline plasma level greater than 100 mg/L or persistent high levels despite appropriate care.

CONCLUSION

Theophylline toxicity can cause a myriad of nonspecific symptoms involving multiple body systems (neurologic, cardiovascular, respiratory, and GI). Status epilepticus is among the most dangerous adverse effects of theophylline toxicity. Prompt recognition of the underlying cause of the status epilepticus must be identified to address the toxicity and resolve the seizures. Involving the patient's family member to confirm a medication list raised suspicion for theophylline misuse and toxicity, which allowed for prompt treatment. In the case of new-onset seizures or status epilepticus, toxicity of drugs, such as theophylline, should always be on the physician's differential, and a thorough review of a patient's medication list is critical.

The authors attest that their institution does not require Institutional Review Board approval. Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Emmelyn J. Samones, CCRP, Loma Linda University Medical Center, Department of Emergency Medicine, 11234 Anderson St, Loma Linda, CA 92354. Email: Esamones@llu.edu.

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Tension Pneumopericardium from a Gastrojejunal-Pericardial Fistula Causing Tamponade: A Case Report

Catherine Moscinski, MD
Alyssa Altheimer, MD
Lachlan Driver, MD
Nikita Maryanov, BA, NRP
Cassidy Cunningham, DO

Warren Alpert Medical School of Brown University, Department of Emergency Medicine, Providence, Rhode Island

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Introduction: Cardiac tamponade typically develops from the accumulation of blood and other fluids, although it can occur from pneumopericardium.

Case Report: A 62-year-old man presented to the emergency department with chest pain and was found to have a pneumopericardium on computed tomography. He developed signs of obstructive shock with hypotension refractory to intravenous fluids and vasopressors. The patient stabilized with bedside pericardiocentesis and pericardial drain placement.

Conclusion: Gastrointestinal-pericardial fistulae are a rare cause of pneumopericardium. Emergency physicians typically rely on cardiac point-of-care ultrasound to evaluate for tamponade and guide pericardiocentesis. However, air artifacts interfere with images, complicating diagnosis and procedures. [Clin Pract Cases Emerg Med. 2026;10(4):489–492.]

Keywords: *pneumopericardium; cardiac tamponade; gastrojejunal ulcer; pericardiocentesis; case report*.

INTRODUCTION

Pneumopericardium, defined as the presence of air within the pericardial sac, is a rare condition in adults that carries significant morbidity and mortality. While it can be self-limiting, it may progress to tension pneumopericardium, resulting in cardiac tamponade, obstructive shock, and death if not promptly recognized.[1,2] Historical mortality rates approach 57%, and tension physiology confers a 56% mortality even with intervention.[1] Common etiologies include trauma, positive pressure ventilation, iatrogenic causes (particularly atrial fibrillation ablation and cardiothoracic surgery), and, rarely, gastrointestinal-pericardial fistulae.[1–3]

Gastrointestinal-pericardial fistulas are exceptionally rare causes of pneumopericardium; only 65 cases are reported in the modern literature, mostly secondary to prior esophageal or gastric surgery.[4,5] Intestinal-pericardial fistulae involving the jejunum are documented only in isolated case reports, making jejunal-pericardial fistula among the rarest etiologies of pneumopericardium.[4–6]

Emergency physicians frequently rely on cardiac point-of-care ultrasound (POCUS) for rapid evaluation of hypotension and chest pain, which may be the presenting signs of tension pneumopericardium. However, intrapericardial air poses significant diagnostic challenges**.** Air is highly hyperechoic and reflects ultrasound waves, creating characteristic "dirty shadowing," comet-tail artifacts, and loss of visualization of deeper cardiac structures.[7] This acoustic impedance can obscure the myocardium and pericardial layers, impeding assessment of tamponade physiology and potentially delaying diagnosis.[8] The classic sonographic finding is the "air gap" sign—bright spots moving along the pericardial layer during diastole with comet-tail artifacts that disappear during systole.[7] When POCUS is limited by intrapericardial air, computed tomography remains the most sensitive modality for detecting pneumopericardium and delineating fistulous tracts.[4,9]

We present a case of pneumopericardium presenting as

chest pain and hemodynamic instability, ultimately attributed to a jejunal-pericardial fistula. This case highlights the diagnostic challenges posed by intrapericardial air on cardiac POCUS and underscores the importance of maintaining a broad differential diagnosis for pneumopericardium in the emergency department (ED). This case also underscores the critical importance of maintaining proficiency in landmark-based procedural skills, such as pericardiocentesis, when ultrasound guidance is unavailable.

CPC-EM Capsule

What do we already know about this clinical entity?

Pneumopericardium—the accumulation of air within the pericardial sac—has a mortality rate up to 57% and can progress to tension physiology and tamponade.

What makes this presentation of disease reportable?

Pneumopericardium presented as chest pain and hemodynamic instability, ultimately attributed to a jejunal-pericardial fistula, an exceedingly rare etiology.

What is the major learning point?

Intrapericardial air can render cardiac ultrasound nondiagnostic. Emergency physicians must maintain proficiency in landmark-based pericardiocentesis.

How might this improve emergency medicine practice?

This case reinforces the need to recognize ultrasound limits from intrapericardial air and to maintain competency in blind pericardiocentesis for obstructive shock.

CASE REPORT

A 62-year-old male presented to the ED with pleuritic chest pain and dyspnea. The patient's past medical history included atrial fibrillation, a recent pulmonary embolism managed with apixaban, and an incomplete first through fourth cervical spinal cord injury resulting in quadriplegia. Additional history included obesity status post Roux-en-Y gastric surgery, hypertension, gastroesophageal reflux disease, and diabetes mellitus. Initial blood pressure was 85/59 mm Hg, with tachycardia at 110 beats/min (bpm); subsequent vital signs included blood pressure 56/48 mm Hg and tachycardia at 124 bpm. The patient presented awake, alert, and oriented, although ill-appearing, pale, and diaphoretic. No crepitus or pain to palpation was noted to his chest wall or abdomen. Heart sounds were distant, although no obvious Hamman sign was auscultated, and no jugular venous distention was noted. Lung sounds were clear to auscultation bilaterally. His neurological examination was pertinent for incomplete paraplegia with decreased motor function from his chest to his feet with intact sensation, unchanged from his baseline. Lower extremity examination demonstrated bilateral pitting edema; peripheral pulses were 2+ bilaterally.

Initial resuscitation included intravenous fluids (IVF), followed by norepinephrine vasopressor support at 5 mcg/min with subsequent improvement in his blood pressure. A computed tomography (CT) angiography aortic dissection protocol revealed moderate pneumopericardium and a small pericardial effusion without definite findings to suggest cardiac tamponade (Image 1). A POCUS and formal transthoracic echocardiogram were completed with limited views due to his pneumopericardium. Cardiac windows were intermittently lost with reverberation or dirty shadowing consistent with intrapericardial air, limiting assessment for right ventricle diastolic collapse. The ED clinical team consulted cardiac surgery and thoracic surgery services, both of which recommended conservative management. The patient, however, continued to decompensate, requiring additional IVF and continued norepinephrine for vasopressor support. Interventional cardiology performed an emergent bedside pericardiocentesis as tamponade physiology was assumed, and a pericardial drain was placed to gravity (Image 2).

A CT esophagogram was later completed without radiographic evidence of esophageal perforation. The patient had persistent pneumopericardium and developed recurrent hypotension and tachycardia, concerning for recurrent obstructive shock despite interventions. In addition, he developed agonal breathing and was intubated for airway protection. Bedside aspiration from the pericardial drain removed a significant amount of air from the pericardium, with improvement in his blood pressure. The pericardial drain was connected to a low-suction drainage system. The pericardial fluid was sent for culture, which grew mixed aerobic flora, and he was initiated on vancomycin and piperacillin-tazobactam. Bronchoscopy did not reveal evidence of large-airway injury or a fistula. An elevated pericardial fluid amylase level was noted, concerning for a gastrointestinal source of his pneumopericardium. Repeat CT of the patient's chest, abdomen, and pelvis was notable for the appearance of a marginal ulcer at the gastrojejunal anastomosis fistulizing through the diaphragm and into the pericardium, creating a jejunal-pericardial fistula.

During admission, the patient underwent an upper gastrointestinal (UGI) endoscopy, which revealed a large ulcer with an 18-mm defect just distal to the anastomosis with visualization of pericardial tissue (Image 3). The surgical team placed an endoscopic vacuum therapy device (Endovac) with

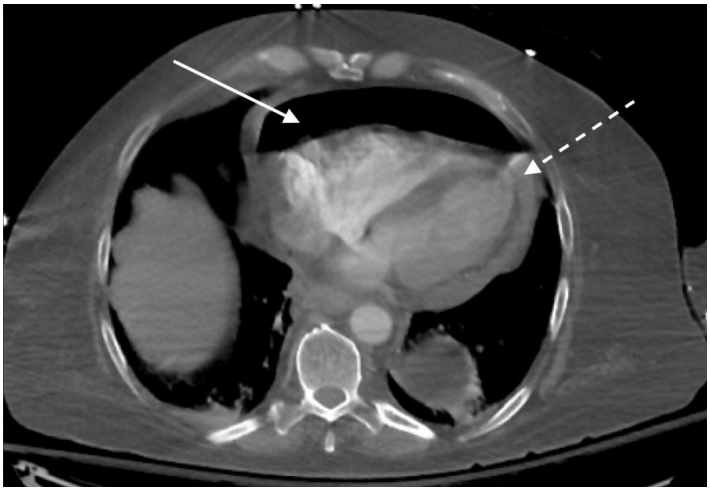


Image 1. Computed tomographic angiography aortic dissection protocol, in lung window, highlighting a moderate-volume pneumopericardium (solid arrow) with associated small-volume pericardial effusion (dashed arrow).

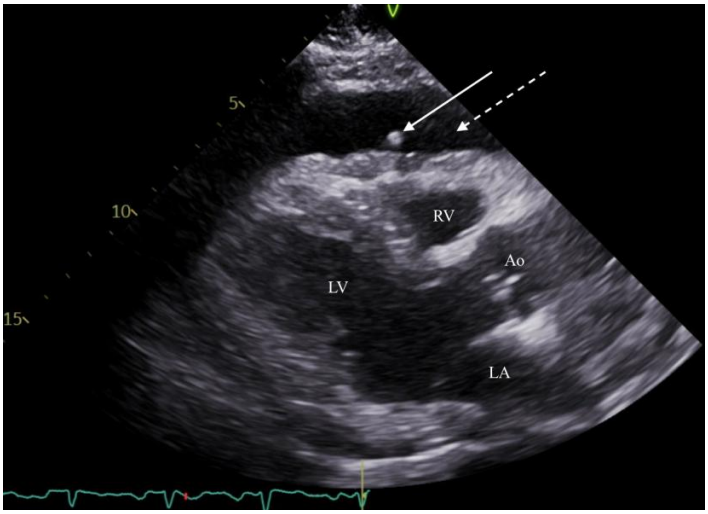


Image 2. Formal transthoracic echocardiogram in the parasternal long axis following emergent pericardiocentesis with aspiration of air and placement of pericardial drain (solid arrow) with residual pericardial effusion (dashed arrow). Ao, aortic outflow tract; *LA*, left atrium; *LV*, left ventricle; *RV*, right ventricle.

a nasogastric tube and surgical sponge attached a wound vacuum over the fistula. Interval UGI endoscopies revealed improvement in the fistula size. Cardiac surgery created a pericardial window with pericardial and pleural drain placement via left anterior thoracotomy. Pericardial fluid samples were obtained and were positive for 1+ yeast (*Candida lambica*) and mold. Antimicrobial coverage was expanded to include micafungin and liposomal amphotericin B. Long-term management included interval Endovac procedures to facilitate fistula closure and completion of a six-week course of antimicrobials.

DISCUSSION

This case highlights tension pneumopericardium as an uncommon but life-threatening cause of obstructive shock in the ED. The key challenges included recognition of tamponade despite limited echocardiographic windows and identification of an underlying source capable of ongoing air entry into the pericardial space.

Cardiac tamponade typically develops from the accumulation of blood and other fluids. However, there is growing evidence that it can also occur from pneumopericardium, similarly impairing diastolic filling and reducing cardiac output. Although rare, it is relatively more frequent in trauma patients and neonates who require positive pressure ventilation. It can also occur from other causes, including infections leading to fistula formation in contiguous organs, esophageal-pericardial fistulas resulting from caustic ingestions, bronchial carcinoma leading to erosion into the pericardium, ulceration of a pulsion diverticulum, development of gastric-pericardial fistulas from perforated gastric ulcers and eroding gastric carcinomas, and from sepsis, where the pericardium was seeded with gas-producing bacteria.[1]

Management of the pneumopericardium depends on the patient's clinical presentation but is typically conservative, including serial examinations and monitoring, analgesics, oxygen therapy, and treatment of the underlying condition.[10] However, patients have a high risk of clinical decompensation, and approximately one in four patients require transfer to the intensive care unit for close monitoring and further interventions.[11,12] In the setting of clinical decompensation, pericardiocentesis is the first-line intervention for immediate decompression and hemodynamic stabilization.[10] As with our patient, it was critical to recognize the progressive development of obstructive shock and advocate for life saving interventions.

Emergency physicians frequently rely on cardiac POCUS for rapid evaluation of hypotension and chest pain and to assist with the pericardiocentesis. However, intrapericardial air poses significant diagnostic and intraprocedural challenges by creating substantial artifacts and impairing visualization of deeper cardiac structures.[7] Specifically, intrapericardial air creates significant acoustic impedance, producing reverberation and dirty shadowing that can intermittently eliminate cardiac windows and obscure visualization of right ventricular collapse. When POCUS assessment is limited by air artifact, the absence of classic findings should not be used to exclude clinically significant tamponade physiology. In this setting, CT can be particularly useful for confirming pneumopericardium, evaluating for an associated effusion and identifying potential etiologies, such as fistula formation.

This case highlights the challenges of stabilization and performance of a pericardiocentesis when there is limited echocardiographic visualization. Although uncommon,

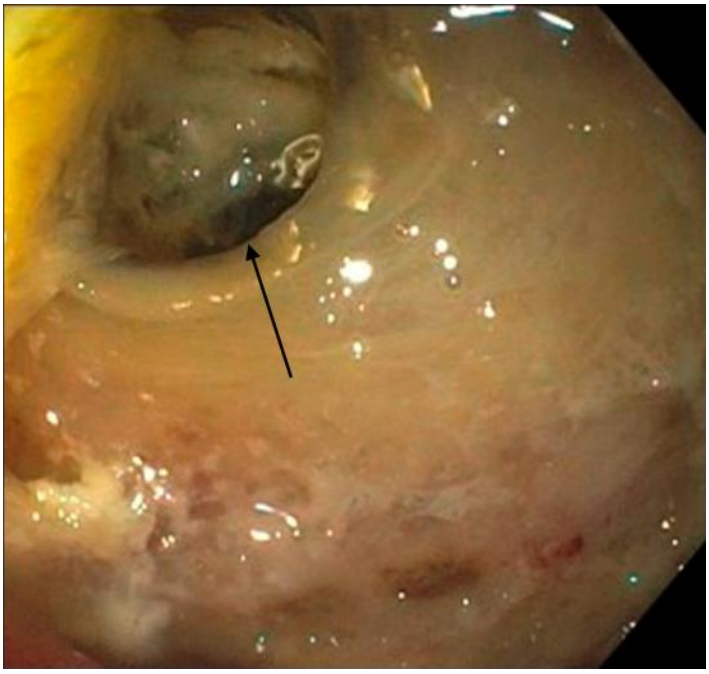


Image 3. Gastrojejunal-pericardial fistula at the anastomosis of Roux-en-Y gastric surgery (arrow) seen on upper gastrointestinal endoscopy.

pneumopericardium warrants inclusion in the differential diagnosis, especially in unstable patients presenting with chest pain and prior gastric surgeries. Patients may require early surgical evaluation and subsequent transfer to a facility with specialized cardiac services. While the pericardial drain was initially attached to a gravity drainage bag with a small amount of pericardial effusion output, pneumopericardium, especially with a fistula as in this case, will likely require the pericardial drain to be attached to low continuous suction to prevent the re-accumulation of air and subsequent recurrence of cardiac tamponade. Given re-accumulation, definitive management often requires source control and urgent surgical evaluation.

CONCLUSION

Tension pneumopericardium is a rare but life-threatening cause of obstructive shock that can mimic classic fluid tamponade. Intrapericardial air may significantly degrade echocardiographic windows, limiting sonographic assessment and complicating ultrasound-guided pericardiocentesis. In patients with pneumopericardium and worsening hemodynamics—especially those with prior foregut surgery—clinicians should maintain a high index of suspicion for tension physiology and consider prompt decompression based on clinical status, even when ultrasound guidance is not feasible. Definitive management requires identifying and treating the underlying source, and persistent or recurrent pneumopericardium may necessitate continuous drainage and multidisciplinary surgical and endoscopic source control.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Cassidy Cunningham, DO, Warren Alpert Medical School of Brown University, Department of Emergency Medicine, 55 Claverick Street, 1st Floor, Providence, RI 02903. Email: ccunningham1@brownhealth.org.

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Intracardiac Placement of Needle Thoracostomy Catheter by Paramedics Due to Anatomic Variation: A Case Report

Brian Strain, DO[*]
Scott Meeker, MD[†]
W. Seth Dukes, MD[*]
Paul Brian Savino, MD, MPH[*]

[*]Loma Linda University Medical Center, Department of Emergency Medicine, Loma Linda, California
[†]Arrowhead Regional Medical Center, Department of Emergency Medicine, Colton, California

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Introduction: Needle thoracostomy is the only procedure in the United States for paramedic treatment of suspected tension pneumothorax within the most recent emergency medical services (EMS) national scope of practice model. We present a rare complication of an out-of-hospital left needle thoracostomy performed by paramedics after intracardiac placement of the catheter. The patient's imaging studies in the receiving emergency department (ED) confirmed that the needle thoracostomy insertion location was correct, but the catheter entered the heart due to the patient's existing atypical anatomy.

Case Report: A 64-year-old unrestrained male driver in a roll-over motor vehicle accident was found in respiratory distress with difficulty auscultating left-sided lung sounds, left chest wall pain on palpation, and confusion. The first-arriving paramedics performed a left needle thoracostomy for suspected tension pneumothorax in advance of helicopter EMS transport. However, pulsatile blood flow from the catheter suggested intracardiac placement, which was confirmed on multiple imaging modalities in the ED. Imaging showed the catheter in the anterior second intercostal space at the midclavicular line. It was also revealed that the patient's heart was prominently superior and nearly extended to the left chest wall with a majority of his left lung positioned posteriorly within the thorax. There were no signs of pneumothorax, hemothorax, subcutaneous emphysema, diaphragmatic injury, sternotomy wires, or other evidence that his atypical anatomy was the result of an exogenous process.

Conclusion: Intracardiac placement of a needle thoracostomy catheter is a rare complication. In this case, the patient's atypical anatomy contributed to asymmetric lung sounds and suspicion of tension pneumothorax. Regardless of which insertion site was chosen, the likelihood of intracardiac placement was potentially high due to the patient's anatomy. Anatomical variation can cause lung sound asymmetry that mimics a pneumothorax, which emergency clinicians and paramedics must consider when assessing for tension pneumothorax. [Clin Pract Cases Emerg Med. 2026;10(4):493–497.]

Keywords: *needle thoracostomy; tension pneumothorax; intracardiac; paramedic; case report.*

INTRODUCTION

Needle thoracostomy is the procedure authorized by the local emergency medical services (EMS) agency for prehospital treatment of suspected tension pneumothorax. This agency authorizes two sites of over-the-needle catheter insertion into the pleural space for decompression.[1] The traditional site is anteriorly at the second intercostal space midclavicular line (ICS2-MCL). More recently, the axillary site at the fourth or fifth intercostal space (ICS4/5) at either the anterior axillary line or at the midaxillary line (MAL) has been adopted after studies indicated decreased chest wall thickness improved likelihood of reaching the pleural space.[2] Prehospital

pleural decompression in the United States is most often performed by needle thoracostomy.

There are alternatives to needle thoracostomy, such as a simple finger thoracostomy, in which a scalpel incision is made at the ICS4/5-MAL and the clinician bluntly dissects or spreads the tissue to access the pleural space with forceps or gloved finger sweeping the pleural space clear of air and/or blood. It is then left open to the atmosphere for continued decompression.[3,4] Another alternative is a chest tube insertion. Both procedures are authorized by the local EMS systems and used only in limited settings by specially trained and qualified prehospital clinicians provided there is medical oversight and appropriate quality assurance.[5]

This case highlights a rare complication of an out-of-hospital needle thoracostomy due to the patient's atypical anatomy mimicking a tension pneumothorax. The treating ground paramedics' suspicion of intracardiac placement after insertion was confirmed at the receiving emergency department (ED). Imaging studies confirmed correct catheter insertion placement but revealed intracardiac extension due to the patient's atypical anatomy.

CPC-EM Capsule

What do we already know about this clinical entity?
Tension pneumothorax is an emergent cause of obstructive shock, treated by immediate pleural decompression.

What makes this presentation of disease reportable?
This presentation demonstrates how anatomic variation can mimic the presentation of tension pneumothorax in a trauma patient.

What is the major learning point?
Anatomic variation may distort lung sounds and mimic the clinical presentation of pneumothorax and tension physiology.

How might this improve emergency medicine practice?
Objective assessment, such as portable ultrasound, prior to needle decompression may clarify the diagnosis and prevent potential complications.

CASE REPORT

A 64-year-old unrestrained male driver with no known medical history presented to the ED via helicopter EMS (HEMS) for blunt traumatic injury after a roll-over motor vehicle accident. Per EMS, the patient was exhibiting respiratory distress with difficulty auscultating left-sided lung sounds, chest wall pain, and confusion. Initial vital signs were as follows: heart rate, 135 beats/min (bpm); oxygen saturation, 78% on room air; respiratory rate, 26 breaths/min; and initial Glasgow Coma Score (GCS) 10 (E2V2M6). Blood pressure was not attempted. The ground paramedic performed a needle thoracostomy using an 8-cm catheter in the left second intercostal space at the MCL for suspected tension pneumothorax. Immediately upon placement, pulsatile blood was noted from the catheter. The catheter was capped and left in place by the transporting HEMS team consisting of a flight paramedic and flight nurse.

Repeat vital signs en route included the following: heart rate, 128 bpm; blood pressure, 160/98 mm Hg; oxygen saturation, 94% on 15 L/min via nonrebreather mask; respiratory rate, 26 breaths/min; and GCS 14 (E4V4M6). The patient received fentanyl 200 mcg and ondansetron 4 mg intravenously en route as well as 1 g of tranexamic acid. The patient continued to have persistent diminished left-sided lung sounds on examination during transport.

Vitals obtained upon arrival to the ED revealed the following: blood pressure, 130/78 mm Hg; heart rate, 97 bpm; temperature, 97.9 °F; respiratory rate, 18 breaths/min; and oxygen saturation, 99% on 15 L/min via nonrebreather mask. The initial extended focused assessment with sonography in trauma exam, radiograph of the chest (Image 1), and computed tomography (CT) of the chest confirmed intracardiac placement of the thoracostomy catheter (Image 2). It was also noted that his heart was prominently superior and nearly extended to the left chest wall with most of his left lung positioned posteriorly within the thorax. Moreover, imaging did not show evidence of pneumothorax, hemothorax, or subcutaneous emphysema. The patient was taken to the operating room where it was again confirmed that the thoracostomy catheter penetrated the pericardium and the lateral left ventricle with subsequent small pericardial effusion without active hemorrhage. The catheter was removed, and the ventricle was repaired. The pericardium was left open, and a Jackson-Pratt drain was placed between the left atrium and left ventricle. Separately, a left-sided chest tube was placed intraoperatively.

The patient was admitted to the intensive care unit for ongoing evaluation and treatment of his injuries. Other significant injuries included unstable sixth cervical vertebra fracture with adjacent spinal epidural hematoma and first lumbar burst fracture. Planned surgical repair was deferred when the patient deteriorated because of progressive traumatic brain injury and sepsis. Based on the poor prognosis and the patient's known wishes against life support, the family elected to proceed with palliative care.

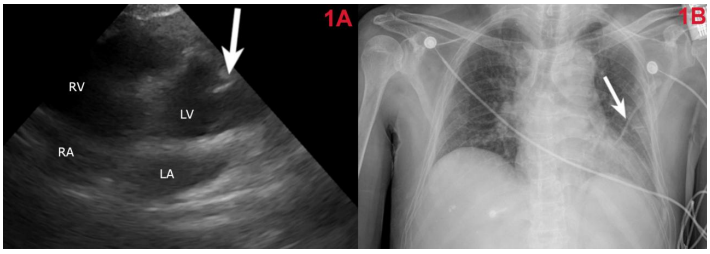


Image 1. (1A) Initial subxiphoid ultrasound image showing catheter tip in the left ventricle of the heart (arrow). (1B) Initial chest radiograph showing the catheter in midclavicular position overlying the cardiac silhouette (arrow).
LA, left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

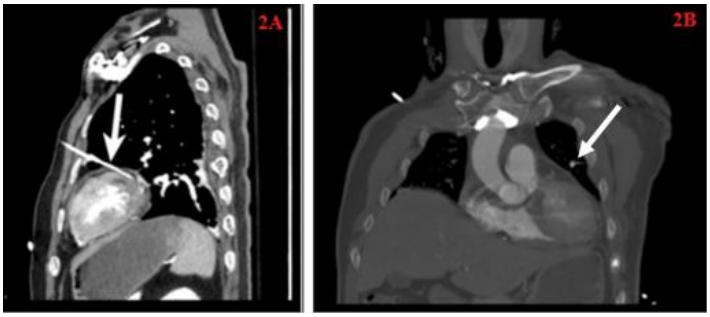


Image 2. (2A) Sagittal image of chest computed tomography (CT) at the approximate left midclavicular line with the catheter (arrow) traversing from entry at the second intercostal space to the superior aspect of left ventricle. (2B) Coronal image of chest CT showing needle thoracostomy insertion at the second intercostal space along the midclavicular line (arrow). The heart is adjacent to the chest wall where needle insertion at the fourth or fifth intercostal space at the midaxillary line would also likely result in iatrogenic injury and/or intracardiac placement.

DISCUSSION

We present a case that highlights the potential iatrogenic injuries that can occur with needle thoracostomy in patients with anatomic variants. This is in contrast with previous reports showing complications secondary to the underlying traumatic injury causing distorted anatomy. In one such case, a patient was victim of a traumatic cardiac arrest following a motor vehicle accident with multisystem trauma. Helicopter EMS performed a left ICS2-MCL needle thoracostomy following return of spontaneous circulation and persistent hypotension without confirmation of a pneumothorax in the ED. The patient was later found to have an intracardiac needle thoracostomy.[6] A retrospective study on prehospital needle thoracostomies identified a case of intracardiac placement. The study did not describe the details of placement; however, review of their CT image supplement revealed an intracardiac ICS2-MCL needle thoracostomy with likely mediastinal shift from visible large right-sided pneumothorax rather than inherent anatomic variation.[7]

Accurate detection and successful treatment of tension pneumothorax in the prehospital setting remains a challenge.[5] Neeki et al emphasizes that traumatic tension pneumothorax is a clinical diagnosis with hallmarks of lung sound asymmetry and hypoperfusion in the setting of chest trauma.[4] The study notes that the actual incidence of tension pneumothorax in the civilian population is not known. Contributing factors include an inability to differentiate iatrogenic pneumothorax caused by needle thoracostomy from pneumothorax resulting from pre-needle thoracostomy traumatic injury. The authors also indicate the difficulty of distinguishing clinical findings attributable to tension pneumothorax from other conditions in the prehospital setting.[4] From review of this case we posit that atypical anatomy can also lead to false-positive identification of tension pneumothorax.

Various studies have demonstrated poor overall needle thoracostomy success rates relating to both insufficient catheter length and improper needle insertion location. Previously, the procedure was routinely performed using 14-gauge intravenous catheters up to 5 cm in length. Increasing the catheter length from 5 cm to 8 cm or greater was advocated after studies that showed many patients had a chest wall thickness greater than 5 cm.[2,5,8] Over-the-needle 10 and 14 gauge/8cm l catheters are commercially available in pleural decompression kits. Changing from the anterior chest ICS2-MCL site to the axillary ICS4/5-MAL site was another strategy, given pectoral muscle mass thickness anteriorly.[2,4,5] After one EMS agency changed its protocol to use the ICS 4/5-MAL as the primary site and increased catheter length to 9.5 cm in 2013, an analysis showed no significant difference in mortality outcomes when comparing cases in the preceding six years and three years after the change.[8]

Variability among clinicians' ability to correctly locate the ICS2-MCL and the ICS4/5-MAL sites has been in reports of prospective simulation and in retrospective case reviews. Inferiorly positioned MCL catheter placement runs the risk of cardiac, diaphragmatic, or intraabdominal injury or placement. Diaphragmatic and intrabdominal placements are also a complication of inaccurate axillary site selection and insertion below the fifth ICS.[9–11] In a study with 33 resident physician and medical student volunteers, the accuracy of correctly marking the ICS4/5 level along the axilla improved from 48% to 91% after brief instruction and addition of ultrasound guidance compared to visual inspection of physical landmarks and palpation alone.[9] Novel approaches to address needle placement challenges now include commercially available template devices to place on the patient for ICS4/5-MAL site confirmation, as well as carbon dioxide detection devices that attach to the catheter to confirm pleural space entry.[12,13] Prehospital use of portable ultrasound could have a role in both confirming tension pneumothorax and aiding in exclusion of tension physiology and reducing unnecessary needle

thoracostomy insertions.[11] Long-term study is needed to determine the extent of patient benefit and whether such strategies should be adopted as standard practice.

Many local EMS protocols require hypotension as evidence of tension physiology prior to performing needle thoracostomy. Many also include empiric bilateral decompression in cases of traumatic arrest with signs of chest trauma. The most recent Tactical Combat Casualty Care guidelines (January 2025) advocate early decompression without awaiting hypotension and advancing the needle/catheter fully with the hub at the skin using an 8-cm catheter.[11] The most recent National Association of EMS Physicians' statement on treatment of traumatic pneumothorax in the 2024 Prehospital Trauma Compendium warns of the increased risk of this technique in the civilian population with catheters longer than 7 cm given the difference in chest wall thickness.[5] The discussion of anatomic variation affecting needle thoracostomy success has been limited to comparison of chest wall thickness. To our knowledge, there is no published research on how native variations in heart and lung position within the chest can impact needle thoracostomy outcomes.

Theoretically, tension physiology should shift vital structures away from the access site on the side of the pneumothorax. Without true tension physiology or when an alternative pathology explains the presentation, needle thoracostomy fails to provide therapeutic pleural volume expansion or protective anatomical shifts. Notably, inadvertent intracardiac placement can occur with other decompression devices as well. One case report details the management of a 44-year-old female pedestrian struck by a vehicle suffering multiple injuries and large left pneumothorax. Decompression attempted via Seldinger technique and with a 14-French pigtail catheter resulted in the catheter transecting a branch of the coronary artery before coiling in the ventricle.[14]

The potential of finger thoracostomy as an alternative to needle thoracostomy for paramedics in the United States is being studied. A retrospective propensity-matched cohort study of 34 patients in Texas suggested higher decompression success by ground paramedics with finger thoracostomy compared to needle thoracostomy. However, because the study was small and not generalizable, it does not support change to finger thoracostomy as the primary method for prehospital pleural decompression.[15] There continues to be wide variability in needle thoracostomy success as well as a high incidence of overtriage of possible tension pneumothorax resulting in needle thoracostomy attempts that are not clinically indicated.[4,5] Thus, a generalizable head-to-head prospective randomized control trial study of needle thoracostomy to finger thoracostomy may not be feasible, setting aside current prehospital scope of practice model limitations.

CONCLUSION

The occurrence of tension pneumothorax in the civilian population remains statistically low. Thus, a paramedic may not encounter a true case of tension pneumothorax during their entire career.[5] Questions remain regarding how to reduce procedural error in the prehospital setting for high-risk, low-frequency and yet potentially life-saving procedures like needle thoracostomy. The lessons of this case may support efforts to include additional assessment technologies such as portable ultrasound and other novel approaches to improve accurate tension pneumothorax recognition and treatment and reduce iatrogenic harm in the prehospital setting.

The authors attest that their institution does not require Institutional Review Board approval. Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Emmelyn Samones, BS, CCRP, Loma Linda University Medical Center, Department of Emergency Medicine, 11234 Anderson St, Room A890A, Loma Linda, CA 92354. Email: Esamones@llu.edu.

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Spontaneous Spinal Epidural Hematoma as a Manifestation of Hypertensive Emergency: A Case Report

Kelly Laird, MD*
Jordan Sell, MD†
Peter England, MD‡
Sacha Montas, MD§
Kevin Walsh, MD§
Carrie Harvey, MD§

*Tacoma Emergency Care Physicians, Tacoma, Washington
†North Memorial Medical Center Emergency Medicine, Robbinsdale, Minnesota
‡University of Michigan, Department of Emergency Medicine, Division of Critical Care, Ann Arbor, Michigan
§University of Michigan, Department of Emergency Medicine, Ann Arbor, Michigan

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Introduction: Uncontrolled hypertension is frequently evaluated in the emergency department (ED), with the goal of identifying those patients with hypertensive emergencies. Spontaneous spinal epidural hematoma is rare and not considered a typical manifestation of hypertensive emergency.

Case Report: A 31-year-old man presenting to the ED with chest pain and hypertension subsequently developed lower extremity neurologic deficits and was diagnosed with a spontaneous spinal epidural hematoma. After thorough testing, no other risk factors for this medical condition were identified, and the hemorrhage was attributed to hypertensive emergency.

Conclusion: Spontaneous spinal epidural hematoma is a rare complication of hypertensive emergency and should be considered when evaluating patients with hypertension, back pain, and neurologic deficits. [Clin Pract Cases Emerg Med. 2026;10(4):498–501.]

Keywords: *spontaneous spinal epidural hematoma; hypertensive emergency; case report*.

INTRODUCTION

Uncontrolled hypertension, or blood pressure greater than 140/90 mm Hg due to untreated or ineffectively treated hypertension, is frequently evaluated in the emergency department (ED). Hypertensive emergency, defined as an acute and marked elevation in blood pressure with associated target-organ damage, is less common, with a prevalence of 0.5% of ED visits.[1,2] Spontaneous spinal epidural hematoma, or blood in the epidural space in the absence of trauma or other iatrogenic cause, is even more rare, with an estimated incidence of 0.1 in 100,00 per year.[3] Spontaneous spinal epidural hematoma has been associated with coagulopathy or vascular malformations, but hypertension is not thought to be a risk factor.[3,4] This rare life-threatening neurosurgical emergency can produce devastating neurologic deficits and requires prompt recognition and intervention to reduce morbidity and mortality.[5] We present a case of a 31-year-old man who developed the medical condition presumed secondary to hypertensive emergency.

CASE REPORT

A 31-year-old man with a history of hypertension presented to the ED with chest pain. The patient presented with severe central chest pain that began several hours earlier. There was no radiation to the back, although he did report mild back pain that he attributed to his job as a house cleaner. He denied shortness of breath, nausea, vomiting, diaphoresis, headache, neurologic symptoms, or preceding trauma. He had no other medical history but noted he had not been adherent to his antihypertensive medications due to inability to access primary care. Social history was relevant for smoking 0.5 packs per day and weekly marijuana use.

Initial vital signs were notable for marked hypertension with blood pressure (BP) 225/134 mm Hg. Temperature, heart

rate, respiratory rate, and oxygenation were normal. On arrival, his physical exam was unremarkable, without cardiac or pulmonary abnormalities noted, and he was able to ambulate without difficulty. It was not immediately clear whether the marked hypertension was the cause or a consequence of his chest pain. Diagnostic testing thus focused on evaluation for serious or life-threatening causes, such as acute coronary syndrome, acute aortic syndrome, pulmonary embolism, pneumothorax, or other end-organ damage from hypertension.

Electrocardiogram showed normal sinus rhythm with likely left ventricular hypertrophy and no other abnormalities. Creatinine was mildly elevated at 1.52 mg/dL (reference range, 0.70-1.30 mg/dL), but complete blood count, prothrombin time, international normalized ratio, partial thromboplastin time and high-sensitivity troponin were all normal. Chest radiograph and computed tomography (CT) of the aorta showed no acute findings or other explanation for his chest pain. There were no abnormal findings within the spinal cord in the final impression or full body of the CT report.

Several hours into his ED course and shortly following CT report finalization, he reported an abrupt onset of numbness to the right lower extremity. Physical examination at that time noted decreased strength to flexion at the right hip and flexion and extension of the right knee and ankle compared to the left, with diminished sensation prompting emergent magnetic resonance imaging (MRI) of the spine with concern for cord compressing process. Over the next 1-2 hours, strength of the right lower extremity decreased, and he developed urinary retention and ascending paralysis into his trunk, although no back pain. Blood pressure was persistently elevated during this time with systolic BP greater than 200 mm Hg. Emergent MRI of his spine noted a posterior thoracic epidural mass at the level of of the first thoracic (T1) to second thoracic (T2) vertebrae measuring 21 mm (Image).

Neurosurgery was consulted due to concern for spontaneous spinal epidural hematoma, and he was taken for urgent open laminectomy with hematoma evacuation. Due to improvement in blood pressure while in the ED during clinical evaluation no antihypertensive medication was provided. Postoperatively, he underwent additional testing to ascertain the etiology of his condition. Spinal angiogram was normal with no arteriovenous malformation or other vascular lesion. Exhaustive workup for underlying coagulopathy was normal, including rotational thromboelastometry, factor XIII, von Willebrand panel, and platelet function testing. The spontaneous spinal epidural hematoma was ultimately ascribed to hypertensive emergency, and he was discharged with near-resolution of his neurologic deficits. Five weeks after surgery, he had no neurologic deficits and had restarted antihypertensive medications.

CPC-EM Capsule

What do we already know about this clinical entity?

Spontaneous spinal epidural hematoma (SSEH) is a rare cause of spinal cord or cauda equina nerve compression that occurs in the absence of trauma or other iatrogenic cause.

What makes this presentation of disease reportable?

Though SSEH is associated with other risk factors, such as vascular malformations or coagulopathy, it is not a typical manifestation of hypertensive emergencies.

What is the major learning point?

Emergency physicians must maintain a high suspicion of this diagnosis when evaluating patients with uncontrolled hypertension, back pain and/or neurologic deficits.

How might this improve emergency medicine practice?

Emergency physicians should consider this rare diagnosis to minimize the risk of devastating neurologic sequelae.

DISCUSSION

Spontaneous spinal epidural hematoma is a rare and emergent condition defined as accumulation of blood in the vertebral epidural space without obvious cause. It typically presents with sudden onset of neck or back pain followed by signs of nerve root or spinal cord compression, such as radiculopathy, progressive numbness or weakness, or cauda-equina syndrome.[3] Symptoms often progress over minutes to hours, although it can be more insidious.[5] Magnetic resonance imaging is the modality of choice, as it can identify location, extent, and chronicity of the hematoma, as well as presence of cord compression and/or spinal cord edema.[6]

Previously described risk factors for spontaneous spinal epidural hematoma include arteriovenous malformations, anticoagulation use, coagulopathy and hemangiomas, though an estimated 40-60% of cases have no identifiable cause.[3] Although the condition has been described in patients with hypertension, a recent meta-analysis did not find this to be an independent risk factor.[4] Additionally, most authors favor epidural veins as the source of bleeding based on anatomy, which would argue against hypertension causing bleeding.[4,5,7] However, while preexisting hypertension may not be a risk factor, it is possible that significantly elevated blood pressure may play an unidentified role in development of spontaneous spinal epidural hematoma.[8,9]

Urgent surgical evacuation within 12-48 hours is the



Image. Sagittal magnetic resonance imaging of the thoracic spine demonstrating a 21-mm posteriorly positioned mass within the thoracic spinal canal at the level of first and second thoracic vertebrae, which results in severe thecal sac narrowing and subtle cord signal alteration at the level of first thoracic. The mass does not demonstrate contrast enhancement on T1-weighted images (A) with peripheral and internal curvilinear hypointensity on T2-weighted images (B), which is most suggestive of epidural hematoma.

definitive treatment, with prognosis most dependent on the severity of the preoperative neurologic deficit.[3-6] Unlike other forms of hypertensive emergency, there is no specific treatment guideline regarding blood pressure management. Cheng et al reported aggressive blood pressure lowering in a patient with cervical spontaneous spinal epidural hematoma and marked hypertension, who was treated nonoperatively with complete neurologic recovery.[10] Small case series in surgical literature have also described increased risk of postoperative spinal epidural hematoma with uncontrolled hypertension (eg, systolic blood pressure > 170 mm Hg.[11,12] Ultimately, prompt surgical consultation or transfer to an appropriate referral center for definitive management is imperative, with additional medical management of coagulopathy and/or uncontrolled hypertension tailored to the individual patient.

In this case, the patient presented with chest pain and uncontrolled hypertension and underwent a typical ED evaluation for these complaints, which was unrevealing. He then suffered a spontaneous spinal epidural hematoma without any clear precipitating cause or risk factors other than his uncontrolled hypertension. Although emergency physicians frequently evaluate patients for hypertensive emergencies, the focus is typically on select target organs that are more commonly damaged, such as brain, heart, and aorta. Thus, spontaneous spinal epidural hematoma secondary to hypertensive emergency is a rare but important consideration in the differential diagnosis for a patient with uncontrolled hypertension and signs and symptoms of spinal cord compression because prompt recognition and intervention are necessary to minimize permanent neurologic damage.

CONCLUSION

Uncontrolled hypertension can lead to highly morbid conditions and is frequently evaluated in the ED. Spontaneous spinal epidural hematoma is a rare complication of hypertensive emergency and should be considered in the emergency evaluation of a patient with uncontrolled hypertension in addition to typical symptoms and signs of spinal cord compression or cauda-equina syndrome.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Carrie Harvey, MD, University of Michigan, Department of Emergency Medicine, 1500 E. Medical Center Dr., Taubman Center B1-354K, Ann Arbor, Michigan 48109-5303. Email: hcarrie@med.umich.edu.

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Exercise-Induced Diaphragmatic Rupture with Intrathoracic Gastric Herniation: A Case Report

Adam S. Berkovits, DO*
Ross P. Berkeley, MD†‡
Gregory P. Daphne, BS, MAPHB§
MacKenzie A. Ellis, BS§
Jeffrey Lin, BS, MS§
Karen P. Chen, BS¶

*Western Arizona Regional Medical Center, Bullhead City, Arizona
†Kirk Kerkorian School of Medicine at UNLV, Department of Emergency Medicine, Las Vegas, Nevada
‡University Medical Center of Southern Nevada, Las Vegas, Nevada
§Touro University Nevada, Henderson, Nevada
¶A.T. Still University, St. Louis, Missouri

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Introduction: While abdominal pain is a common complaint in the emergency department (ED), it is key to obtain a diligent history and maintain a broad differential diagnosis. The development of exercise-induced diaphragmatic hernia is rare but can result in a high risk of morbidity and mortality.

Case Report: This is a case of a 24-year-old man who presented to the ED with acute-onset epigastric abdominal pain that began while performing "burpee" (squat thrust) exercises. Computed tomography of the abdomen and pelvis revealed diaphragmatic rupture with gastric herniation into the left hemithorax. The patient underwent emergent surgical intervention to reduce the intra-abdominal contents and repair the diaphragmatic defect.

Conclusion: There are no prior reports of diaphragmatic hernia due to burpee exercises in the emergency medicine literature. Consideration of exercise-induced diaphragmatic hernia is necessary due to the high morbidity of this condition. [Clin Pract Cases Emerg Med. 2026;10(4):502–505.]

Keywords: *exercise-induced diaphragmatic hernia; spontaneous diaphragmatic hernia; atraumatic diaphragmatic hernia; gastric incarceration; burpee.*

INTRODUCTION

Review of the published literature on exercise-induced diaphragmatic rupture indicates this is a rare occurrence, with no cases describing "burpee" (squat thrust) exercises. Since 2009, there have been sparse reports of exercise-induced diaphragmatic hernias, including three cases involving weightlifting, one involving swimming, one due to deep breathing during Pilates exercise, and one due to lifting heavy garden slabs in a squatting position.[1-6] The clinical presentation of a spontaneous diaphragmatic rupture can be variable, with nonspecific symptoms that may include chest pain, dyspnea, abdominal pain, vomiting, or hematemesis, which can potentially make the diagnosis more challenging to identify.[7] We describe a case of a patient who presented with epigastric abdominal pain secondary to an atraumatic spontaneous exercise-induced diaphragmatic hernia.

CASE REPORT

A 24-year-old man with a history of hypertension was transported via ambulance to the emergency department (ED) of a tertiary-care teaching facility, complaining of two days of constant epigastric pain that radiated diffusely throughout his abdomen and into his testes. The patient reported occasional vomiting but no diarrhea and noted the pain was exacerbated by eating. Upon ED arrival, the patient was afebrile and had a heart rate of 95 beats/min and a blood pressure of 179/94 mm Hg. The only pertinent positive on the physical exam was mild tenderness to palpation of the

epigastrium, with no rebound or guarding, a negative Murphy sign, and no tenderness at McBurney point. The genitourinary exam was unremarkable with no testicular tenderness. Laboratory tests were notable for a hemoglobin level of 17.1 g/dL (reference range, 13.5-17.5 g/dL), with a white blood cell count of 16,300 cells/mm^3 (4,000-11,000/mm^3). Urinalysis demonstrated no signs of infection. Testicular ultrasound was unremarkable with no evidence of testicular torsion. An upright chest radiograph (CXR) interpreted by the radiologist demonstrated "question of some elevation of left hemidiaphragm versus left basilar consolidation, atelectasis, or effusion" (Image 1).

After a thorough workup, the emergency clinician had a low suspicion for underlying pancreatitis, cholecystitis, cholelithiasis, or hollow viscus perforation; neither was there evidence of any free air under the diaphragm on CXR. The patient was feeling improved after five hours of ED observation and was discharged with prescriptions for esomeprazole, ondansetron, and dicyclomine.

The next day, the patient returned to the same ED complaining of severe left upper quadrant abdominal pain with development of multiple episodes of hematemesis in the preceding 12 hours. He denied any rectal bleeding or melena. During this return visit, additional history was obtained that revealed the abdominal pain began acutely while the patient had been performing burpee exercises three days prior. At the time of presentation, the patient's heart rate was 134 beats/minute, and blood pressure was 154/98 mm Hg. The patient appeared diaphoretic, but respirations were nonlabored, with a respiratory rate 20 breaths/min. Cardiac exam was tachycardic without murmurs or rubs, and lungs were clear to auscultation. Abdominal examination was notable for moderate tenderness in the left upper quadrant and epigastric region with associated voluntary guarding but no rebound tenderness. Normoactive bowel sounds were present, and there were no hernias noted of the abdominal wall or inguinal region.

Two large-bore peripheral angiocatheters were established, and the patient received a 2 L bolus of intravenous (IV) crystalloid. Laboratory test results were notable for a hemoglobin level of 17.2 g/dL and a white blood cell count of 22,000/mm^3. An upright CXR (Image 2) by the radiologist demonstrated a dense opacity in the left lung base suggestive of infiltrate with associated effusion. However, the patient denied any recent cough or fevers. Computed tomography (CT) of the abdomen and pelvis with IV contrast was expedited, and the patient empirically received cefoxitin 2 gm IV. The CT abdomen and pelvis revealed a diaphragmatic rupture with gastric herniation into the left hemithorax (Image 3).

Following consultation and bedside evaluation by the surgical team, the patient was transported to the operating room. Exploratory laparotomy revealed a laceration of the lateral aspect of the left diaphragm with herniation and

CPC-EM Capsule

What do we already know about this clinical entity?
Exercise-induced diaphragmatic rupture is rare, may present with non-specific thoracic and/or abdominal symptoms, and can easily be misdiagnosed.

What makes this presentation of disease reportable?
This is the first reported case of diaphragmatic rupture induced by burpee exercises, with non-specific symptoms resulting in delayed diagnosis and gastric necrosis.

What is the major learning point?
Thoracoabdominal pain after exertional Valsalva-type activity should raise suspicion for diaphragmatic rupture with gastric herniation.

How might this improve emergency medicine practice?
Earlier consideration of atraumatic diaphragmatic rupture with timely diagnosis and surgical consultation can help reduce morbidity and mortality.

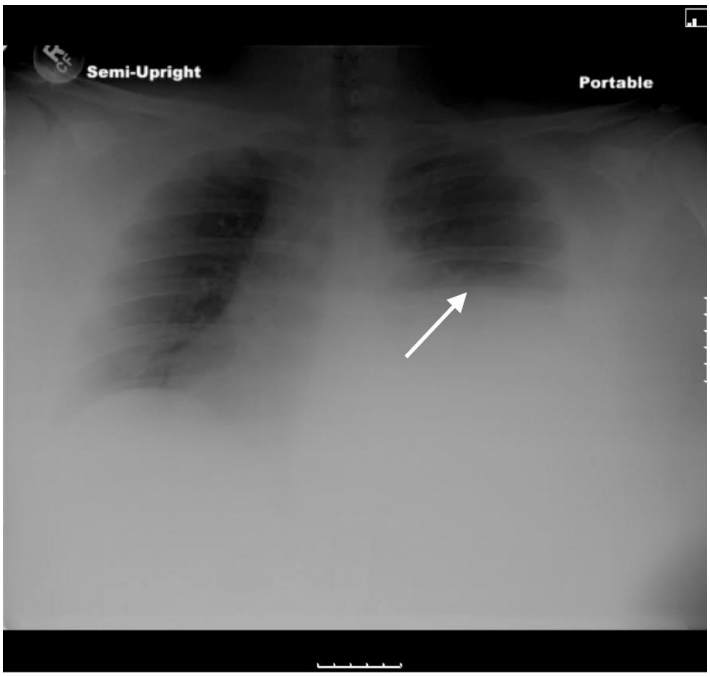


Image 1. Anteroposterior chest radiograph demonstrating elevation of left hemidiaphragm (arrow).

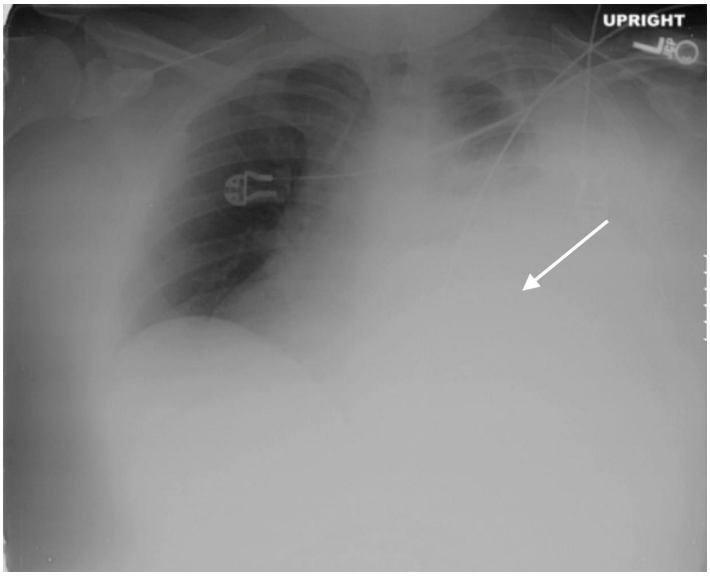


Image 2. Anteroposterior chest radiograph demonstrating a dense left-sided opacity suggestive of intrathoracic gastrointestinal contents, with an associated mediastinum into the right hemithorax (arrow).



Image 3. Coronal view of computed tomography abdomen demonstrating gastrointestinal contents in the thoracic cavity, herniated through a diaphragmatic defect (arrow).

strangulation of the entire stomach and omentum, the majority of which appeared grossly necrotic. The patient underwent diaphragmatic repair with omentectomy and partial gastric resection with primary anastomosis. His postoperative course was uneventful, and he was discharged five days later.

DISCUSSION

Spontaneous diaphragmatic rupture is a rare thoracoabdominal emergency involving a defect in the diaphragm, often complicated by herniation of peritoneal contents; it requires timely identification, treatment and surgical repair.[8] A sudden increase in intra-abdominal pressure from movements similar to the Valsalva maneuver during exercise, weightlifting, or coughing may be sufficient to result in diaphragmatic rupture at the site of a potential pre-existing defect.[7,9,10] Acute diaphragmatic rupture is seen after blunt or penetrating trauma in 5% of cases.[11-15]

Chest radiography may reveal unilateral diaphragmatic elevation, pulmonary consolidation or pleural effusion, hemithoracic density, and/or obvious intrathoracic displacement of abdominal organs. In this case, although the history and physical examination were nonspecific during the initial ED visit, the elevated white blood count and CXR abnormality should have prompted an expansion of the differential diagnosis with consideration for further imaging, as well as a deeper dive into the history and circumstances surrounding the onset of the symptoms. Diagnosis of an acute diaphragmatic rupture may be more challenging in those patients who present with nonspecific symptoms or without pathognomonic findings on plain radiography, and it is therefore key to maintain an index of suspicion based on the history and mechanism, with CT the most useful imaging modality. Reconsideration of the differential based on the mechanism and diagnostic findings may have led to an earlier diagnosis and helped avoid the partial gastric resection that was ultimately required for this patient. A key takeaway is that nonspecific thoracoabdominal pain following an exertional Valsalva-type activity should raise the clinician's suspicion for acute diaphragmatic rupture with gastric herniation. While this case emphasizes the consideration of this differential, a congenital or acquired diaphragmatic defect could have increased this patient's susceptibility to developing a diaphragmatic rupture.

Definitive management involves surgical intervention to reduce the herniated intra-abdominal contents and repair the diaphragmatic defect. Delayed or missed diagnosis can lead to a worsened prognosis with potential for strangulation with ischemia, tissue necrosis, and/or hollow viscus perforation.[7,9,10] As such, this case serves as an example of the importance of expanding the differential for atypical abdominal complaints following exercise.

CONCLUSION

To our knowledge, this is the first report in the literature of a spontaneous diaphragmatic rupture due to burpee exercises. The infrequent occurrence of this entity may lead to misdiagnosis and delay of definitive treatment. Due to the significant morbidity involved with spontaneous diaphragmatic hernia, it is key to consider this entity in the

context of symptom onset during certain exercises and physical activities that may result in a sudden increase in intra-abdominal pressure.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Ross P. Berkeley, MD, Kirk Kerkorian School of Medicine at UNLV and University Medical Center of Southern Nevada, Department of Emergency Medicine, 901 Rancho Lane, Suite 135, Las Vegas, NV 89106. Email: ross.berkeley@unlv.edu.

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The Stellate Ganglion Nerve Block—A Novel Use for Electrical Storm in the Emergency Department: A Case Report

Allison Smith, MD
Louisa Weindruch, DO
Rebecca Mayo, DO
Jonathan Brewer, MD, FPD-AEMUS

Baylor University Medical Center, Department of Emergency Medicine, Dallas, Texas

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Introduction: Electrical storm is an emergent medical condition in which a patient experiences recurrent episodes of ventricular tachycardia or ventricular fibrillation. Traditional management includes intravenous antiarrhythmics and/or electrical cardioversion. However, in some cases, cardiac arrhythmias may persist despite these interventions. In rare circumstances, traditional therapies in the emergency department (ED) may prove to be futile. In such cases, a stellate ganglion nerve block (SGNB) can be considered.

Case Report: We present a novel case in which a SGNB was performed in the ED on an awake patient experiencing recurrent cardioversions from his implantable cardioverter defibrillator shocks for ventricular tachycardia.

Conclusion: In the setting of refractory ventricular dysrhythmias, sympathetic blockade of the left stellate ganglion can be a useful adjunct to conventional management in the ED. [Clin Pract Cases Emerg Med. 2026;10(4):506–509.]

Keywords: *electrical storm; regional anesthesia; ventricular tachycardia; stellate ganglion block; case report.*

INTRODUCTION

Electrical storm is defined as three or more recurrent episodes of a malignant cardiac arrhythmia such as ventricular tachycardia (VT) or ventricular fibrillation (VF) in a 24-hour period. The etiology is varied, but it is theorized that sympathetic activation lowers the threshold for these malignant arrhythmias.[1] Initial treatment in hemodynamically stable patients is intravenous (IV) antiarrhythmic drugs. If the patient is hemodynamically unstable or refractory to antiarrhythmics, cardioversion is the treatment of choice. When a patient is refractory to both medications and cardioversion, a stellate ganglion nerve block (SGNB) can be considered. The SGNB is postulated to function by temporarily blocking the cervical sympathetic chain.[2] It has been largely used in the inpatient setting by cardiologists and anesthesiologists. However, in recent years, there has been emerging evidence on its utility in the emergency department (ED) for patients in cardiac arrest. These data are still limited and exist mostly as case reports and case series on patients in cardiac arrest. In this case, we present the novel use of the nerve block in the ED for treatment of refractory VT and electrical storm in an awake and hemodynamically stable patient with recurrent cardioversions from his automated implantable cardioverter defibrillator (AICD). To date, our case marks the first of its kind reported within the United States.

CASE REPORT

A 60-year-old male with an extensive past medical history including end-stage renal disease, prior cerebrovascular accident, hypertension, recurrent pericardial effusions status post pericardial window, VT/VF arrest status post AICD

placement, coronary artery disease status post coronary artery bypass graft, and heart failure with reduced ejection fraction of 30-35% who presented to the ED after his AICD delivered two episodes of cardioversion that day. The patient was well-appearing and had the following initial vital signs: blood pressure, 181/107 mm Hg; pulse, 82 beats/min (bpm); respiratory rate, 22 breaths/min; oxygenation,95% on room air; and temperature, 98.5 ºF. He was placed on continuous cardiac monitoring and an electrocardiogram initially demonstrated sinus tachycardia at 107 bpm. Intravenous access was established. A chest radiograph showed cardiomegaly without other acute abnormalities.

While the laboratory evaluation was pending, an episode of VT was noted on the telemetry monitor and coincided with the patient experiencing two more episodes of synchronized cardioversion from his AICD. His vital signs remained unchanged. He continued to have episodes of monomorphic VT and was witnessed to have nine additional appropriate synchronized cardioversions from his AICD within a matter of minutes, causing him significant pain. Medical management was attempted with IV amiodarone (150 mg) followed by a magnesium infusion (2 g). However, the patient continued to have recurrent episodes of VT with appropriate cardioversion and overdrive pacing attempts by his AICD. Given the failed pharmacologic interventions and patient's extreme discomfort, SGNB was considered. The risks and benefits of the procedure as well as other possible interventions were discussed with the patient, and he elected to proceed with SGNB.

The patient was positioned to sit upright with his neck in slight extension and rotated to the right, contralateral to the side of the planned procedure. His left anterolateral neck was prepped and draped in sterile fashion. The linear ultrasound probe was placed in transverse orientation at the level of the cricoid cartilage, which is approximately at the level of the sixth cervical vertebra (C6). While the stellate ganglion typically spans from the seventh cervical vertebra (C7) to the first thoracic vertebra (T1), the vertebral artery arises anterior to C7, making C6 a safer landmark for puncture.[3] After moving slightly laterally and caudally, the C6 and C7 transverse processes were identified. This included identification of the anterior tubercle of C6, also known as the Chassaignac tubercle. The vasculature was visualized using color Doppler, including the internal carotid artery, vertebrobasilar arteries, and internal jugular vein.

An in-plane approach with a 22-gauge block B-Braun needle was performed. The needle was inserted laterally and advanced medially until the hyperechoic tip was visualized at the prevertebral fascia of the longus colli muscle, between the posterior aspect of the carotid artery and the Chassaignac tubercle. Five milliliters of 1% lidocaine without epinephrine were injected incrementally and were visualized elevating the prevertebral fascia, just below the stellate ganglion (Images 1 and 2, Figure). The needle was removed without complication. The patient developed miosis and ptosis within 5-10 minutes

CPC-EM Capsule

What do we already know about this clinical entity?
Stellate ganglion nerve blocks (SGNB), traditionally used in the inpatient setting, now have growing evidence for utility in patients with cardiac arrest.

What makes this presentation of disease reportable?
We present the novel use of a SGNB for electrical storm in an awake and hemodynamically stable patient in the emergency department (ED).

What is the major learning point?
When a patient in electrical storm is refractory to both medications and cardioversion, a SGNB should be considered.

How might this improve emergency medicine practice?
The SGNB can be a useful adjunct in the management of refractory cardiac arrhythmias in the ED.

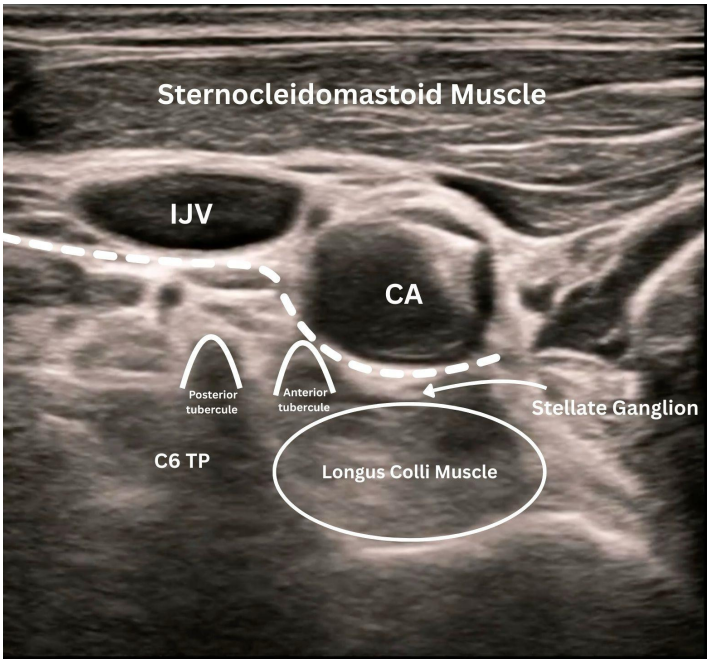


Image 1. Ultrasound image of neck anatomy, with the dotted white line indicating prevertebral fascia.
C6, sixth cervical vertebra; *CA*, carotid artery; *IJV*, internal jugular vein; *TP*, transverse process.

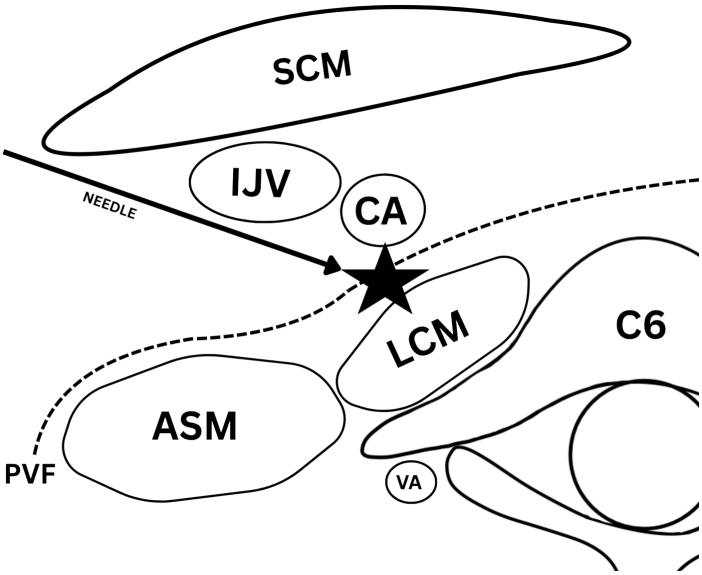


Figure. Diagram of neck anatomy. The star indicates stellate ganglion.
ASM, anterior scalene muscle; *C6*, sixth cervical vertebra; *CA*, carotid artery; *IJV*, internal jugular vein; *LCM*, longus colli muscle; *PVF*, prevertebral fascia*; SCM*, sternocleidomastoid muscle; *VA*, vertebral artery.

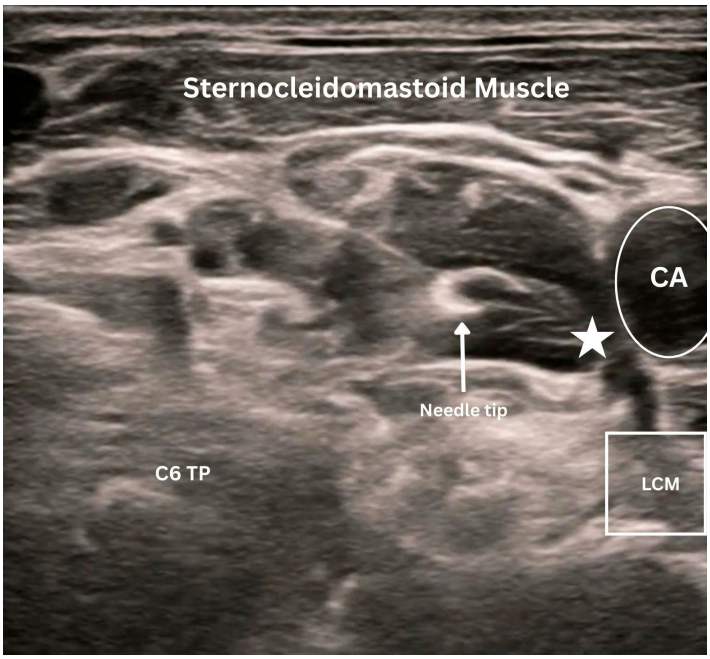


Image 2. Ultrasound image of a needle injecting lidocaine. The star indicates lidocaine injectate traversing underneath the carotid artery; the white arrow points to the needle tip.
C6, sixth cervical vertebra; *CA*, carotid artery; *LCM,* longus colli muscle; *TP*, transverse process.

of the SGNB, suggesting effective coverage of the nerve region.[4] There were no further shocks delivered by the AICD and no further episodes of VT while in the ED. The patient was then admitted to the cardiology service for further management. During his hospitalization, he had no additional episodes of VT. He declined any additional procedures and was discharged on oral amiodarone on hospital day 4.

DISCUSSION

The stellate ganglion is a group of sympathetic nerve fibers in the anterior neck. It is formed by fusion of the inferior cervical ganglion with the first thoracic ganglion at the level of C6 and C7.[5] While it can be found bilaterally, it is believed that the left stellate ganglion plays a more significant role in autonomic regulation of the heart; hence, the left-sided blockade is generally performed.[5] In the 1970s, researchers began to explore the connection between sympathetic activation and cardiac arrhythmias, with one of the first stellate ganglion blocks for antiarrhythmic purposes being reported in 1976.[4,6] Since then, the technique has been used by cardiologists and anesthesiologists for management of arrhythmias refractory to traditional management. In recent years, cases have emerged revealing its use in the ED, particularly in the setting of cardiac arrest.[7] Our case represents a novel use for the stellate ganglion block in the ED in a patient who was awake and hemodynamically stable.

Emergency physicians must be able to identify and quickly treat dysrhythmias, as recurrent ventricular arrhythmias can have a mortality rate up to 97%.[7] Electrical storm is common in patients with AICDs, occurring in 5% of those placed for primary prevention and 25% of those placed for secondary prevention.[4] Recent literature has highlighted SGNB as a successful antiarrhythmic in electrical storm. In a meta-analysis of 15 studies with over 500 patients in electrical storm, 70% had complete resolution of their ventricular arrhythmias after SGNB.[8] In a prospective study of 115 patients, 92.2% had a reduction of at least 50% in the number of treated arrhythmic episodes following SGNB.[4] Complications are rare, the most common being transient hoarseness and lightheadedness.[9]

The benefits of the SGNB in our case are multifactorial. Not only did the SGNB quickly relieve the patient's severe discomfort from the repeated AICD shocks, it decreased the likelihood of hemodynamic instability and eliminated the need for further interventions in the ED. With the widespread use of ultrasound-guided procedures and the increasing prevalence of ultrasound-guided regional anesthesia, we propose that the SGNB is feasible for emergency physicians. Landmarks can easily be visualized on ultrasound, and the procedure requires minimal time and resources—the ideal scenario for a busy ED. Ultimately, this case demonstrates how the SGNB can be used as an adjunct for termination of recurrent ventricular arrhythmias in awake patients in the ED.

CONCLUSION

When conventional measures fail for refractory ventricular dysrhythmias, left stellate ganglion sympathetic blockade is a useful emergency department adjunct.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Allison Smith, MD, Baylor University Medical Center, Department of Emergency Medicine, 3500 Gaston Ave, Dallas, TX 75246. Email: allisonharmsworth@gmail.com.

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Prehospital Ultrasound to Identify Endotracheal Tube Dislodgment in an Infant: A Case Report

Nicholas A. Wright, DO, NR-P* *Edward Via College of Osteopathic Medicine, Blacksburg, Virginia
Ryan Lucas, NR-P† †Christiansburg Emergency Medical Services, Christiansburg, Virginia
Peyton Bailey, PA-C, NR-P†

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Introduction: Point-of-care ultrasound (POCUS) is well established as a useful adjunct for airway management in both adult and pediatric patients, including confirmation of successful endotracheal intubation. While suprasternal ultrasound of the trachea is the most described method for confirming endotracheal tube (ETT) placement, a focused lung exam can be used to rapidly identify appropriate ventilation in otherwise apneic patients.

Case Report: This case involves a one-month-old male infant who experienced an out-of-hospital cardiac arrest secondary to suspected sudden infant death syndrome. During resuscitation, an accidental extubation occurred after an attempt at securing the ETT with a commercial pediatric tube holder. Loss of end-tidal carbon dioxide ($ETCO_2$) waveform was recognized immediately; however, emergency medical services clinicians questioned the device's accuracy due to earlier failures of the $ETCO_2$ monitor and the ETT remaining secured at an expected depth. A rapid POCUS lung examination demonstrated absence of bilateral lung sliding. The patient was immediately extubated, revealing a kinked ETT in the oropharynx with the distal tip positioned above the vocal cords. A supraglottic airway device (SGA) was placed with a successful return of $ETCO_2$ waveform, followed shortly by successful reintubation because of difficulty ventilating through the SGA.

Conclusion: Lung ultrasound is a rapid and effective alternative for identifying ETT dislodgment when conventional verification methods are unreliable or unavailable. As ultrasound becomes increasingly available in prehospital and resource-limited settings, it may be effectively used by nonphysician clinicians to confirm airway placement and detect airway complications. [Clin Pract Cases Emerg Med. 2026;10(4):510–513.]

Keywords: *point-of-care ultrasound; airway management; out-of-hospital cardiac arrest; emergency medical services; pediatric resuscitation.*

INTRODUCTION

Point-of-care ultrasound (POCUS) is an increasingly used adjunct for airway management in both adult and pediatric patients. Established applications include confirmation of successful endotracheal intubation, detection of esophageal intubation, and assessment of adequate lung ventilation.[1,2] Although suprasternal ultrasound of the trachea is the most described sonographic method for confirming endotracheal tube (ETT) placement, lung ultrasound can also be used to provide rapid confirmation of effective ventilation through visualization of bilateral lung sliding in an otherwise apneic patient.[3,4]

Prehospital access to ultrasound continues to expand, offering a valuable method for identifying airway complications, particularly in resource-limited environments when standard confirmation techniques fail. We report a case of accidental ETT dislodgement during the out-of-hospital cardiac arrest of an infant in which lung POCUS enabled rapid

diagnosis when conventional confirmation methods were unreliable.

CPC-EM Capsule

What do we already know about this clinical entity?
Point-of-care ultrasound (POCUS) is an established adjunct for confirming endotracheal tube (ETT) placement, though infant and prehospital data remain limited.

What makes this presentation of disease reportable?
This case highlights the use of prehospital ultrasound to rapidly diagnose accidental ETT dislodgement in a one-month-old infant in cardiac arrest.

What is the major learning point?
Lung ultrasound can be used by nonphysician clinicians to rapidly detect tube dislodgment when conventional confirmation methods are unreliable.

How might this improve emergency medicine practice?
Prehospital POCUS may improve recognition of ETT dislodgment when conventional confirmation methods are unreliable.

CASE REPORT

In July 2025, emergency medical services (EMS) responded to an emergency call for a "newborn not breathing." On arrival, a law enforcement officer was performing cardiopulmonary resuscitation on a 1-month-old male infant who was pulseless and apneic. The arrest was presumed to be secondary to sudden infant death syndrome (SIDS), because a parent was co-sleeping with the patient and awoke to find him unresponsive. It was unknown how long the patient had been in arrest; however, no immediate contraindications to resuscitation were identified.

Resuscitation was initiated in accordance with the EMS agency's standards of care and American Heart Association guidelines. Compressions and ventilations were performed at a 3:1 ratio until an appropriately sized supraglottic airway device (SGA) was placed. Following intraosseous (IO) access epinephrine (0.01 mg/kg) was administered at appropriate intervals. At some point during the resuscitation, it was noted that the initial IO had infiltrated, and a second IO was successfully placed in the left distal femur.

Following SGA placement, it was noted that there was poor compliance with the device and copious amounts of frothy sputum were accumulating in the device and the bag-valve-mask (BVM). Despite aggressive suctioning, compliance did not improve. The decision was made to perform ET intubation with a 3.0-mm uncuffed ETT. Intubation was successful, confirmed by continuous waveform end-tidal carbon dioxide ($ETCO_2$) consistent with BVM ventilations, absence of epigastric sounds, and presence of bilateral lung sounds. Shortly thereafter, secretions occluded the $ETCO_2$ sampling line, causing intermittent loss of waveform. The device was exchanged for a new one, which corrected the problem.

During securement of the ETT with a commercial pediatric tube holder, the ETT was inadvertently withdrawn from the oropharynx. The clinician readvanced the ETT, suspecting that the tube had not been fully withdrawn from the trachea, but a sudden loss of $ETCO_2$ occurred, raising concern for ETT dislodgment. Some clinicians attributed waveform loss to recurrent $ETCO_2$ device occlusion, and secretions were once again visualized in the sample tubing. Lung sounds were auscultated and interpreted as "diminished" by the clinician.

Given the uncertainty of conventional confirmation methods and immediate availability of an ultrasound device, a rapid bilateral anterior thoracic examination was performed during the next rhythm check. The ultrasound examination along the midclavicular lines demonstrated an absence of bilateral lung sliding (Video).

The patient was immediately extubated, and direct visualization of the oropharynx revealed a kinked ETT with the distal tip positioned above the vocal cords, most likely caused by blind readvancement of the ETT. The SGA was immediately reinserted. Ventilation through the SGA once again demonstrated poor compliance and inconsistent $ETCO_2$ waveforms, prompting repeat intubation. A second clinician successfully performed ETI using a 3.5 mm uncuffed ETT. Waveform $ETCO_2$ returned and remained consistent with BVM ventilations. Chest compressions were not halted for either intubation attempt.

After 40 minutes of advanced on-scene resuscitation without return of spontaneous circulation, the closest emergency department (ED) was contacted regarding patient disposition. The attending physician directed the continued administration of epinephrine and transport of the patient. The patient was moved to the ambulance and transported without complication. Once at the receiving facility, the ED staff continued the resuscitation for a brief period prior to terminating efforts.

The medical examiner determined the cause of death to be SIDS and identified a tracheal injury assumed to have been caused by the mother compressing the patient's neck while co-sleeping. This may have contributed to the observed ventilation difficulty while the SGA was in place.

DISCUSSION

Rapid confirmation of correct ETT placement is critical

whenever ETI is performed but particularly in infants where any amount of hypoxia drastically worsens outcomes.[5] Continuous waveform $ETCO_2$ monitoring is considered the gold standard of confirming tracheal intubation and for detecting misplacement; however, unreliable readings may occur due to device failure or occluded sample tubing. It has also been established that other methods of ETI confirmation, such as auscultation of lung sounds and visualization of condensation in the ETT, are less effective and not sufficiently reliable to confirm correct ETT placement.[6,7]

In prehospital and austere environments; limited resources, volatile scenes, and frequent staffing shortages pose challenges to clinicians in ensuring continuous confirmation of ETT placement. Facilitating patient movement or performing tasks with a solo clinician that are traditionally performed with several people in the in-hospital environment (such as ETT securement) poses a serious risk for ETT dislodgement. When combined with the limitations of continuous monitoring in the prehospital environment—such as lung auscultation or colorimetric end-tidal capnometry, unrecognized ETT dislodgment is a known risk. Even though the clinicians had access to the waveform $ETCO_2$ capnography, it still proved to be ineffective because of suspected equipment failure. These challenges are amplified in pediatric ETI due to the high-acuity, low-frequency nature of the procedure.

In recent years, decrease in cost and widespread access to educational materials has resulted in a substantial increase in POCUS implementation throughout EMS systems in the United States. As previously discussed, ultrasound has been established as a reasonable and accepted method to verify ETT placement in both adult and pediatric patients. While most of the studies in this area focus on direct visualization of the ETT within the trachea for confirmation, some have also described using thoracic ultrasound to visualize pleural sliding as evidence of effective ventilation.[8] One study found that "use of the ultrasound-guided lung sliding sign was more accurate than auscultation" for ETT positioning.[9]

With minimal training, non-physician prehospital clinicians can accurately determine the presence or absence of lung-sliding using thoracic ultrasound.[10,11] Thoracic ultrasound is a simple, cost-effective, and non-invasive procedure that, in the setting of emergent pediatric intubation and equipment failure, may be utilized as an adjunct assessment technique to confirm sustained tracheal placement of an ETT. Although well described in hospital settings, reports of prehospital clinicians using thoracic ultrasound in infants to verify ETT position are lacking. This case illustrates how lung POCUS can rapidly identify ETT dislodgement when traditional methods fail or the environment is not conducive to traditional confirmation methods.

CONCLUSION

This case outlines three important points: 1) Thoracic-lung ultrasound is a rapid and effective alternative for identifying ETT dislodgment in pediatric patients when conventional verification methods are unreliable or unavailable; 2) in resource-limited or high-acuity environments, ultrasound may improve airway safety; and 3) as ultrasound becomes increasingly available in prehospital and austere settings, it may be effectively used by nonphysician clinicians to confirm airway placement and detect airway complications. Additionally, while suprasternal tracheal sonography has been established as an effective method for confirming endotracheal tube intubation, many clinicians already perform thoracic ultrasound in routine practice, and these same methods can be readily applied to airway management.

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Video. Bilateral anterior thoracic lung exam performed in the sagittal plane along the midclavicular lines. The initial window is of the patient's left thorax, followed by the right. Lung sliding is absent bilaterally (arrows).

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Nicholas A. Wright, BS, NR-P, Edward Via College of Osteopathic Medicine, 2265 Kraft Dr. SW Blacksburg, VA 24060. Email: nwright@vcom.edu.

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Unna Boot Therapy Reduces Emergency Department Use in an Unhoused Patient: A Case Report

Ariana Coba Clementel, BS* *New York University Grossman School of Medicine, New York City, New York
Frank L. Ross, MD† †New York University Grossman School of Medicine, Department of Surgery, New York City, New York

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Introduction: Venous leg ulcers disproportionately affect people experiencing homelessness and contribute to recurrent emergency department (ED) use. Standard wound care protocols often require daily dressing changes and leg elevation, which are unrealistic in unstable housing environments.

Case Report: A 71-year-old male with a 10-year history of venous leg ulcers and 15 ED visits achieved complete wound epithelialization after four months of weekly Unna boot compression therapy, despite eviction during treatment. He has not returned to the ED for this condition.

Conclusion: Emergency department-initiated compression therapy with structured wound care referral may reduce recurrent ED use among unhoused patients with venous leg ulcers. [Clin Pract Cases Emerg Med. 2026;10(4):514–518.]

Key Words: *wound care; people experiencing homelessness; venous leg ulcers; ED utilization; case report.*

INTRODUCTION

Chronic venous insufficiency is a common and morbid condition that frequently leads to venous leg ulcers, which are chronic wounds characterized by delayed healing and high recurrence rates.[1] Venous pathology accounts for 70% of chronic lower extremity wounds, reflecting the central role of impaired venous return and sustained inflammation in the disruption of healing.[2,3] Consequently, venous leg ulcers contribute substantially to patient morbidity and healthcare utilization. They are cause for substantial financial strain in developed nations, accounting for $148.65 billion in US health expenditures in 2022.[4] Despite their prevalence, venous leg ulcers require ongoing compression therapy and reliable follow up, which are often not feasible within fragmented healthcare systems. Without reliable outpatient management, the emergency department (ED) often becomes the default site of care for patients with chronic wounds. This episodic model of care addresses acute symptoms without correcting the underlying venous pathology, contributing to recurrent visits. This pattern is especially pronounced among people experiencing homelessness, who face higher rates of peripheral vascular disease and disproportionately rely on the ED for care.[5] Venous leg ulcers account for up to 20% of medical complaints among people experiencing homelessness, likely reflecting a combination of inadequate nutrition, limited rest, poor hygiene, prolonged standing or sitting, and restricted access to longitudinal care,[6] and they contribute significantly to repeat ED use (1.63-18.75 times that of housed individuals).[7] Therefore, venous leg ulcers are a frequent source of repeat ED use for people experiencing homelessness, reflecting both disease burden and a gap in accessible, sustained treatment.

Compression therapy remains the cornerstone of venous leg ulcer management, with evidence supporting its effectiveness in healing wounds through improved venous return and reduction of venous hypertension.[6,8] The Unna boot, a zinc oxide–impregnated compression bandage, provides barrier protection and sustained graduated pressure. It can be maintained for up to one week, reducing the need for frequent

dressing changes. This makes it a practical option for patients with limited access to consistent wound care.

Previous studies have consistently demonstrated that the Unna boot is an effective, low-cost compression therapy for venous leg ulcers, supporting its role in conservative management.[9] Systematic reviews and randomized studies show comparable or improved healing outcomes to other compression modalities; however, data were derived from outpatient clinics and do not evaluate outcomes related to ED utilization.[10,11] Despite its established use in outpatient and wound care settings, ED-initiated Unna boot therapy and impact on ED utilization remains poorly described in the literature. This represents a potential gap in care delivery for people experiencing homelessness who frequently rely heavily on episodic ED visits for management of chronic wounds.

We present a case of an unhoused individual with recurrent ED use for venous leg ulcer management, in whom consistent outpatient clinic Unna boot therapy was associated with successful wound healing and cessation of repeat ED visits. This case highlights a potential underused strategy for bridging gaps in care and reducing avoidable ED use in a high-risk population.

CPC-EM Capsule

What do we already know about this clinical entity?

Venous leg ulcers are common, slow to heal, and often lead to repeat Emergency Department (ED) visits when compression therapy and follow-up are not sustained.

What makes this presentation of disease reportable?

An unhoused patient with years of recurrent ED use had venous leg ulcer healing and no further ED visits after consistent Unna boot therapy.

What is the major learning point?

Unna boot compression is a practical, low-barrier treatment for venous leg ulcers that can work even when housing instability limits ideal wound care.

How might this improve emergency medicine practice?

Earlier ED recognition and initiation of compression therapy may reduce repeat visits and connect high-risk patients to definitive wound care.

CASE REPORT

A 71-year-old male with a history of hypertension, chronic obstructive pulmonary disease, asthma, obstructive sleep apnea, chronic venous insufficiency with prior iliac vein stent placement, and chronic venous leg ulcers presented to the outpatient wound care clinic with worsening ulceration of the right lower extremity. His symptoms included pruritus, erythema, mild pain, and serous drainage. The patient's history of venous leg ulcers extended back approximately 10 years, beginning in 2015, during which time he required 15 total ED visits, for complications such as cellulitis, ulcer drainage, and severe leg pain. Over a five-year period from 2020 to 2025, he was treated intermittently by the vascular surgery service, which attributed his condition to venous insufficiency. Arterial Doppler studies ruled out arterial disease, and venous Doppler studies were negative for deep vein thrombosis. In 2020, he underwent placement of a right external iliac venous stent. Despite this, his management remained largely conservative, consisting of intermittent courses of oral antibiotic therapy, dressing changes with silver-impregnated hydrofiber dressings (Aquacel® Ag), compression bandaging, and instructions to elevate the lower extremities at home.

Because of the persistent and refractory nature of the ulcer, he was referred to the outpatient wound care clinic for further management. At his initial evaluation on October 16, 2025, physical examination revealed warm lower extremities without pallor or cyanosis, although both legs demonstrated woody edema, brown hyperpigmentation, and erythema with a maculopapular rash consistent with stasis dermatitis. Closer inspection of the right medial ankle revealed a full-thickness ulcer with a granular base and light fibrinous exudate (Image 1). The wound was slightly moist but lacked purulence, odor, or exposure of deeper structures such as bone or tendon. The ulcer measured 6 × 3 × 0.2 cm, and there was no evidence of surrounding cellulitis. Palpation of peripheral pulses was limited due to significant edema.

Initial treatment focused on establishing a consistent wound care and compression regimen. After cleansing the ulcer with normal saline, topical silver alginate (Silvercel ™) was applied, followed by placement of a paste Unna boot extending from the base of the toes to just below the tibial tuberosity. A light layer of Coban™ was used to secure the dressing without excessive tension. The procedure was well tolerated, and the patient was instructed to return weekly for reassessment and reapplication. He was also advised to elevate his lower extremities above heart level when resting. This approach aimed to both protect the wound and provide sustained compression, thereby improving venous return and promoting ulcer healing.

Over the following weeks, the patient received consistent weekly Unna boot compression at the clinic. However, his course was complicated in early January 2026 by a significant social setback, as he reported eviction from his home and transition to living in a shelter. This change severely limited



Image 1. First presentation of right venous leg ulcer in a patient with chronic venous insufficiency.



Image 2. Complete epithelization of right venous leg ulcer after 15 weeks of Unna boot compression.

his ability to elevate his legs and maintain optimal hygiene. Despite these challenges, he continued to attend weekly wound care appointments.

By February 2, 2026—four months after initial presentation—physical examination of the right lower extremity demonstrated complete epithelialization of the ulcer, with no residual open areas, drainage, odor, or signs of infection (Image 2). Although chronic woody edema and xerosis persisted, there was no evidence of cellulitis, and the right foot remained warm and well perfused.

Ultimately, successful healing of the venous leg ulcer was achieved without complication. Notably, the patient has not required further ED visits for this condition since completing the treatment.

DISCUSSION

This case highlights the practical utility of Unna boot compression in an unhoused patient with a venous leg ulcer, initiated in the outpatient setting and with subsequent impact on ED utilization. The patient reported difficulty ambulating, elevating his lower extremities, and maintaining basic hygiene because of access to showers and clean clothing. Standard wound care recommendations, particularly those requiring daily dressing changes and prolonged leg elevation, are therefore unrealistic for people experiencing homelessness. This population faces numerous barriers to healing, including inadequate nutrition, limited rest, restricted access to exercise and outpatient follow-up, mistrust of healthcare clinicians, and difficulty obtaining medications and pain control.[12] Critically, people experiencing homelessness are often unable to maintain consistent leg elevation, a cornerstone of venous leg ulcer management.[13]

Unna boot compression offers a practical alternative that accounts for these constraints. The Unna boot delivers sustained, graduated compression that improves venous return while also functioning as a protective dressing, thereby reducing the need for frequent dressing changes and hygiene. By consolidating compression and wound coverage into a single application that can be maintained once weekly, this modality significantly reduces dependence on daily wound care, stable hygiene, self-management, and infectious risk.

Although therapy in this case was initiated in the outpatient setting, its subsequent implementation was associated with a termination of recurrent ED utilization. This

suggests that earlier initiation of compression therapy may interrupt the cycle of repeated ED presentations. Therefore, the ED represents a critical potential point of intervention for earlier initiation of therapy. Patients with venous leg ulcers present to the ED for pain management, infectious complications, or dressing failure. Given the simplicity and speed of Unna boot application, ED initiation of therapy is feasible and could be incorporated into existing wound care workflows without significant disruption. Earlier initiation in the ED may therefore shorten the time to definitive compression therapy and reduce reliance on episodic, palliative ED care.

Within the ED, several steps may facilitate implementation. First, increased recognition of venous leg ulcers among unhoused patients is essential to ensure appropriate identification and referral. Second, emergency physicians can initiate Unna boot compression at the point of care because of its brief application time and compatibility with standard wound care workflows. Third, structured referral pathways to accessible outpatient wound care services remain critical for ongoing management. As demonstrated in a narrative review and program evaluation, community-based wound care clinics provide low-threshold, cost-effective longitudinal care, facilitate connection to social services, and reduce stigma associated with traditional healthcare settings.[14] Together, this strategy addresses the core pathophysiology of venous leg ulcers through compression therapy while accommodating the realities of unstable housing. Early initiation in the ED may further enhance this effect by shortening time to definitive treatment and reducing episodic, fragmented care.

This report contributes to the literature by demonstrating that a low-barrier, compression-based wound care strategy initiated in the outpatient setting reduced downstream ED utilization in an unhoused patient. It also highlights the ED as a potential site for earlier initiation of therapy to prevent recurrent visits. To our knowledge, the link between timing of compression therapy and ED use in people experiencing homelessness has not been well described.

LIMITATIONS

Although patient follow-up was limited by housing instability, the observed termination in ED use suggests meaningful downstream system-level impact. While single-case observations limit generalizability, this report supports the hypothesis that earlier initiation of compression therapy, potentially in the ED, may reduce frequent ED use for people experiencing homelessness.

CONCLUSION

Wound care for venous leg ulcers is often poorly adapted to the realities of homelessness, contributing to recurrent ED utilization for pain control, infection, and repetitive dressing changes. In this case, Unna boot compression was associated with cessation of recurrent ED visits, despite unstable housing, limited leg elevation, inconsistent hygiene, and restricted access to nutrition and medications. Given inconsistent availability of wound care infrastructure, scalable ED-integrated strategies such as Unna boot initiation is urgently needed and tailored to people experiencing homelessness. Further study is warranted to evaluate the effectiveness of ED initiated compression therapy on wound healing and ED use.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Ariana Coba Clementel, BS, New York University Grossman School of Medicine, 334 E 26th St., Unit 5K, New York, NY, 10010. Email: Ariana.CobaClementel@nyulangone.org.

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Bezold Abscess: A Case Report

John Wahhab, MD*
Ani Oganesyan, BS*
Peter Pham, BS*
Evan L. Jackson, DO†

*Chicago Medical School at Rosalind Franklin University of Medicine and Science, Department of Medicine, North Chicago, Illinois
†University of Illinois Chicago, Department of Emergency Medicine, Chicago, Illinois

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Introduction: A Bezold abscess is a neck abscess characterized by inflammation and infection of the mastoid process that can invade deep into local anatomical musculature and vasculature.

Case Report: We highlight the case of a 62-year-old man who presented with ongoing left-sided neck pain, headache, and tinnitus that started two weeks prior to arriving at the emergency department. Physical examination showed tenderness to the left trapezius muscle, left-sided mastoid tenderness, and pearly tympanic membranes bilaterally. A computed tomography angiogram of the head and neck was performed due to concerns of vertebral dissection, which eventually led to a diagnosis of Bezold abscess.

Conclusion: Although Bezold abscess has been described in the medical literature, it is rarely encountered in modern clinical practice because the widespread use of antibiotic therapy prevents acute mastoiditis from progressing to abscess formation. We highlight the complexity of clinical presentation that can easily be mistaken for other medical conditions, such as occurred with our patient who presented with symptoms more consistent with vertebral dissection: neck pain; tinnitus; and headache, without associated ear pain or discharge. [Clin Pract Cases Emerg Med. 2026;10(4):519–522.]

Keywords: *Bezold abscess; mastoiditis; neck abscess; computed tomography; case report*.

INTRODUCTION

Bezold abscess is characterized by infection and inflammation of the mastoid process tip that often produces a suppurative collection of pus in the digastric muscle.[1] If Bezold abscess is left untreated, purulent material will accumulate in the mastoid bone causing osteitis and osteonecrosis of fragilized pneumatized air cells.[2] Pneumatized air cells are cavities of air-filled spaces through the eustachian tube and middle ear that are responsible for offsetting pressure changes in the middle ear and permitting gas exchange within the submucosal capillary network.[3] Purulent secretion from severe mastoiditis can extend into deeper and surrounding anatomical structures that may cause significant morbidity. However, the use of antibiotics therapy has significantly altered the clinical course of mastoiditis, rendering Bezold abscess a rare complication in modern practice.[1]

Risk factors for Bezold abscess include cholesteatoma, recurrent otitis media, or recurrent mastoiditis, which are conditions more common in the pediatric population.[1] Normal growth into adulthood elongates the skull base and the eustachian tube becomes more oblique and better drained, decreasing infection risk.[4] Additionally, pneumatized air cells decline with age and rapidly decrease during the seventh decade, reducing pathways for infection to spread into the mastoid.[5] Consequently, the likelihood of Bezold abscess formation in elderly patients is significantly reduced, which can cause underrecognition of Bezold abscess as a differential diagnosis and consequently create delays in treatment.

Since Bezold abscess is not commonly encountered in clinical practice, we chose to inform physicians about this rare diagnosis and its treatment to prevent morbidity and mortality.

This case report highlights the importance of considering Bezold abscess in the differential diagnosis of neck pain, even in patients who fall outside the typical pediatric age range and lack traditional risk factors.

CASE REPORT

A 62-year-old man presented to the emergency department with two weeks of left-sided neck pain, headache, and tinnitus that began while shoveling snow. His past medical history included previous subarachnoid hemorrhage and alcohol use disorder. His initial vital signs showed the following: blood pressure, 128/84 mm Hg; heart rate, 103 beats/min; oxygen saturation, 95% on room air; respiratory rate, 18 breaths/min; and temperature, 36.1°C. His physical examination showed mild tenderness to palpation in the left trapezius muscle and the midline of the neck, mild left-sided mastoid tenderness, and pearly tympanic membranes bilaterally. Cerebellar function was intact to finger-to-nose and rapid alternating movements.

The patient's left-sided neck pain, tenderness, and tinnitus prompted concern for vertebral artery dissection. Differentials such as ischemic stroke, intracranial hemorrhage, infection, and other neurological catastrophe were also considered due to his mastoid tenderness and previous history of subarachnoid hemorrhage. Therefore, computed tomography (CT) angiogram of the head and neck was performed, which demonstrated asymmetric soft tissue fullness with central hypoattenuation in the high cervical soft tissues underlying the left mastoid, appearing to extend into the left lateral epidural space (Images 1-3). These findings were concerning for otomastoiditis with associated Bezold abscess given concomitant osseous erosion. Otolaryngology was consulted and recommended intravenous antibiotic therapy and transfer to a tertiary care center for a higher level of care. The patient received vancomycin (1,750 mg), ampicillin-sulbactam (3g) intravenously and was transferred to an academic center.

At the university hospital, the patient was evaluated by the otolaryngology service, which recommended further imaging with magnetic resonance imaging and consultation of infectious diseases and interventional radiology. The patient was admitted to the surgical intensive care unit and started on vancomycin (1,500 mg) every 8 hours and cefepime (2 g) IV every 8 hours.

Magnetic resonance imaging revealed malignant otitis externa, osteomyelitis of the skull base, a 16-mm abscess, and a dural venous thrombosis. Interventional radiology was consulted but determined they were unable to drain the abscess. Otolaryngology performed a left myringotomy with tympanocentesis, and cultures were sent. Culture results yielded the following: *Rothia mucilaginosa; Actinomyces; Streptococcus mitosis; Streptococcus oralis;* and *Prevotella*. The infectious diseases service recommended a six-week duration of antibiotics. Due to the dural venous thrombosis, the patient was started on a heparin drip and eventually

CPC-EM Capsule

What do we already know about this clinical entity?
Bezold abscess is a rare mastoiditis complication causing deep neck infection, classically seen in pediatric patients with otologic symptoms.

What makes this presentation of disease reportable?
An elderly patient presented with neck pain and tinnitus concerning for vertebral artery dissection but was ultimately diagnosed with Bezold abscess.

What is the major learning point?
Bezold abscess can present atypically in older adults; persistent neck pain with mastoid tenderness may warrant advanced imaging.

How might this improve emergency medicine practice?
Recognizing atypical Bezold abscess presentations may reduce diagnostic delays and prevent severe infectious complications.

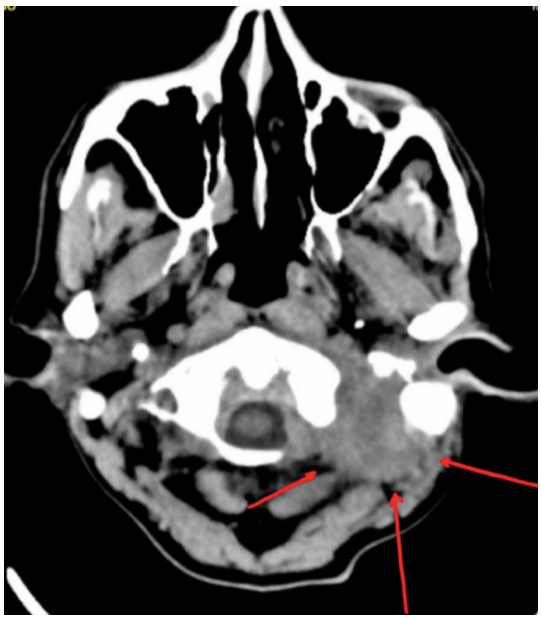


Image 1. Axial computed tomography angiogram of the head and neck demonstrating a 3.0 x 3.7 x 3.1 cm ill-defined soft tissue density (arrows) centered inferior to the medial aspect of the mastoid temporal bone and lateral to the left occipital condyle with disruption of the fat planes and a central hypodensity concerning for Bezold abscess.

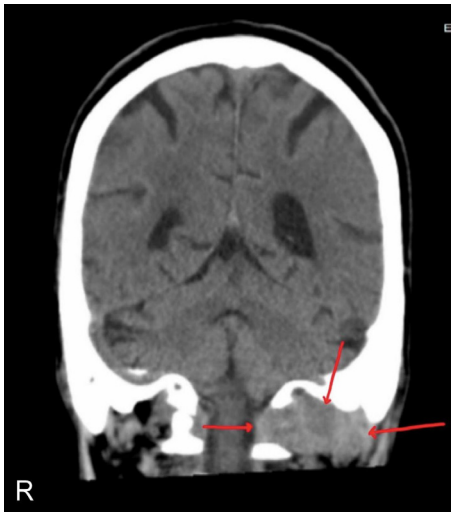


Image 2. Coronal computed tomography angiogram of the head and neck demonstrating a 3.0 x 3.7 x 3.1 cm soft tissue density (arrows) located inferior and medial to the left mastoid process of the temporal bone and extending to the occipital condyle. There is an additional central hypodense region, which is a collection consistent with fluid or pus, concerning for Bezold abscess.



Image 3. Sagittal computed tomography angiogram of the head and neck demonstrating a 3.0 x 3.7 x 3.1 cm (arrows) abnormal soft tissue collection located inferior and posterior to the mastoid process with extension toward the cervical musculature. Within the central region is a hypodense cavity suggestive of fluid or abscess formation, accompanied by adjacent inflammatory changes to fascial planes, concerning for Bezold abscess.

bridged to warfarin.

After a prolonged hospital course, a repeat CT performed on an inpatient basis showed resolution of the fluid collection. The patient was then discharged home to continue a course of amoxicillin-clavulanate (875-125 mg) twice per day. Otolaryngology recommended audiology testing, and results were noted to be unremarkable. The patient followed up with otolaryngology and infectious diseases in the outpatient clinic and noted full resolution of his symptoms.

DISCUSSION

The novelty of this case lies in its presentation of an elderly patient without traditional risk factors and with nonotologic symptoms that initially suggested a vascular cause. Bezold abscess is an exceptionally rare infectious complication of mastoiditis, with fewer than 100 reported cases in the literature.[6] This condition typically presents in children and young adults with tenderness below the mastoid process, neck swelling, restricted neck movement, ear pain and discharge, and fever.[7] In contrast, this patient's symptoms were initially more suggestive of vertebral artery dissection, underscoring the complexity of Bezold abscess in older adults. The patient's physical examination findings of low temperature (36.1 °C) and pearly tympanic membranes did not match the classic Bezold abscess findings of fever and tympanic membrane changes associated with infection.[8] Furthermore, he lacked common risk factors such as a history of cholesteatoma, chronic otitis media, or recurrent mastoiditis.[1]

Advanced imaging with CT angiography was pursued over more conservative initial modalities due to the patient's persistent neck pain over several weeks and associated neurologic findings, including tinnitus, which raised concern for vertebral artery injury. This case highlights the importance of escalating evaluation when symptoms are persistent, progressive, or accompanied by atypical otologic or neurologic features despite an otherwise nonspecific presentation. Physicians should maintain a low threshold for advanced imaging in patients with a history of cholesteatoma, recurrent otitis media, or mastoiditis who present with persistent neck pain in conjunction with mastoid tenderness, hearing changes, neck swelling, or neurologic symptoms, because these findings may indicate serious pathologies such as Bezold abscess.

Although Bezold abscess is traditionally considered rare in older adults due to age-related loss of pneumatized mastoid air cells, emerging reports suggest it may be underrecognized in this population. The condition has been reported in a growing number of elderly male patients, reflecting a need to include it in the differential diagnosis alongside other entities such as deep neck abscesses, suppurative parotitis, coalescent mastoiditis, malignancy, and cutaneous abscesses.[9]

CONCLUSION

The severity and complexity of this patient's findings

required a multidisciplinary approach involving specialists from otolaryngology, infectious diseases, interventional radiology, and hematology. By increasing clinical awareness and prioritizing early collaborative management, physicians can significantly improve patient outcomes and reduce the risk of severe complications associated with Bezold abscess.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Address for Correspondence: Peter Pham, BS, Chicago Medical School at Rosalind Franklin University of Medicine and Science, Department of Medicine, 333 N Green Bay Rd, North Chicago, IL 60064. Email: peter.pham@my.rfums.org.

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Luxatio Erecta with Complex Medical Decision-Making Regarding Reduction: A Case Report

Mitch Daniel, BS* *University of South Florida Health Morsani College of Medicine, Lehigh Valley Campus, Bethlehem, Pennsylvania
Aaron B. Deutsch, DO† †Jefferson Health Lehigh Valley Region, Department of Emergency and Hospital Medicine, Division of Medical Toxicology, Bethlehem, Pennsylvania
Marna R. Greenberg, DO, MPH*‡ ‡Jefferson Health Lehigh Valley Region, Department of Emergency and Hospital Medicine, Bethlehem, Pennsylvania

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Introduction*:* Luxatio erecta is a rare glenohumeral dislocation. The standard treatment is reduction under procedural sedation; however, decision-making becomes more nuanced in patients with comorbidities. This report describes a hyperglycemic patient with type 1 diabetes with luxatio erecta.

Case Report: An 18-year-old man resented with a traumatic inferior shoulder dislocation. Laboratory tests indicated severe hyperglycemia without diabetic ketoacidosis. The emergency department team performed a risk-benefit assessment before proceeding with procedural sedation. Closed reduction was successful, and hyperglycemia was managed with intravenous fluids.

Conclusion: Inferior shoulder dislocations are rare; severe hyperglycemia in diabetics is not an absolute contraindication to procedural sedation, but the patient may need individualized risk-benefit assessment. [Clin Pract Cases Emerg Med. 2026;10(4):523–527.]

Keywords: *inferior shoulder dislocation; luxatio erecta; procedural sedation; risk-benefit; case report.*

INTRODUCTION

Shoulder dislocations are a relatively common orthopedic injury in which the humeral head is forced out of the glenoid socket. They are the most common form of joint dislocation due to the extensive range of motion in multiple planes allowed by the glenohumeral joint allows, with an incidence of 51.2 per 100,000 persons per year.[1] Risk factors for shoulder dislocations are largely trauma-related, especially from contact sports like football and basketball, in which the shoulder is subject to vulnerable positions for dislocation.[2] Treatment for shoulder dislocation comprises prompt closed reduction, paying careful attention to axillary nerve and axillary artery damage, which may present as loss of deltoid strength and lateral shoulder sensation or weak distal pulses, respectively.[3]

Most shoulder dislocations are anterior, occurring after forced external rotation and abduction of the arm and forcing the humeral head to displace anteriorly out of the "socket,"[2,4] This often occurs during contact sports or trauma.[2] However, other forms of shoulder dislocation may occur depending on patient anatomy, mechanism of injury, and comorbidities. For example, epileptic patients are prone to posterior shoulder dislocations because powerful, involuntary contractions of internal rotator and adductor muscles overpower the same involuntary contractions of opposing muscles, forcing the humeral head to displace posteriorly against the glenoid rim.[5,6]

A small subset of patients present with inferior shoulder dislocations, also known as luxatio erecta. This condition refers specifically to an inferior dislocation of the glenohumeral joint in which the humeral head becomes trapped beneath the coracoid process and glenoid rim. It is a very rare subtype, accounting for approximately 0.5% of all shoulder dislocations.[7] One proposed mechanism of action is forceful hyperabduction causing the humeral head to lever down.[8] Another proposed mechanism of action is direct axial

force on an abducted arm.[8]

Inferior shoulder dislocations are typically treated in the same manner as other shoulder dislocations: closed reduction followed by shoulder immobilization and pain management.[2,9] Conscious sedation may involve a combination of benzodiazepines, opioids, and nerve blocks, making management patient-dependent. Many situations exist in which the full medical combination is relatively contraindicated or not recommended, and these decisions are largely based on American Society of Anesthesiologist (ASA) guidelines.[10] For example, patients with significant cardiorespiratory comorbidities undergoing procedural sedation face an increased risk as high as 14.7% for adverse respiratory events, versus 0% with peripheral nerve block alone ($P < .001$).[10] Another relative contraindication is urgent situations in patients at risk for pulmonary aspiration because there is no opportunity for fasting.[11]

The 2018 ASA Practice Guidelines for Moderate Procedural Sedation and Analgesia consider an ASA physical status of 1 or 2 to be appropriate for procedural sedation.[11] An ASA physical status of 1 or 2 refers to a healthy patient with no comorbidities or a patient with mild, well-controlled disease, respectively. Patients with an ASA physical status of 4 (severely compromised or medically unstable) are generally not candidates for procedural sedation, and consultation with an anesthesiologist is recommended. However, ASA status 3 (moderate to severe systemic disease) is a gray area in that procedural sedation is not a hard contraindication, but a careful risk-benefit assessment is necessary beforehand. Examples of a patient who is classified as ASA 3 would be an individual with coronary artery disease with a previous myocardial infarction, or it could be as simple as morbid obesity (Figure).

If a patient is healthy or has only mild disease, procedural sedation in the emergency department (ED) prior to reduction is appropriate. Alternatively, if the patient has severe, systemic disease that is unstable, an anesthesia consultation should be sought.

According to the American Diabetes Association (ADA), procedural sedation in patients with hyperglycemia is reasonable following adequate preprocedural assessment including screening for hyperglycemic emergency. If there are no concerns for hyperglycemic emergency and no underlying organ failure, normalizing periprocedural blood glucose is the main goal.[12] Data on sedation agent selection for hyperglycemic patients are limited, but universal principles of avoiding respiratory depression and maintaining hemodynamic stability apply. Therefore, the choice between procedural sedation and an anesthesia consultation should be individualized. This report highlights a young man with type 1 diabetes (T1D) who presented to the ED with an inferior shoulder dislocation following a motor vehicle collision (MVC). On admission the patient's glucose level was uncontrolled. We present this case as a borderline ASA III example in which the decision to proceed with procedural sedation was made and proved successful.

CPC-EM Capsule

What do we already know about this clinical entity?
Luxatio Erecta is a rare glenohumeral dislocation. The standard treatment is reduction under procedural sedation, however, decision making becomes more nuanced in patients with comorbidities.

What makes this presentation of disease reportable?
An 18-year-old male presented with a traumatic inferior shoulder dislocation. His labs indicated severe hyperglycemia. Risks versus benefits in the approach to reduction are discussed.

What is the major learning point?
Severe hyperglycemia in diabetics is not an absolute contraindication to procedural sedation, but the patient may need individualized risk-benefit assessment.

How might this improve emergency medicine practice?
Physicians benefit from familiarity with the American Society of Anesthesiologist risk categorizations. This case reviews the guidelines of approach for patients requiring sedation.

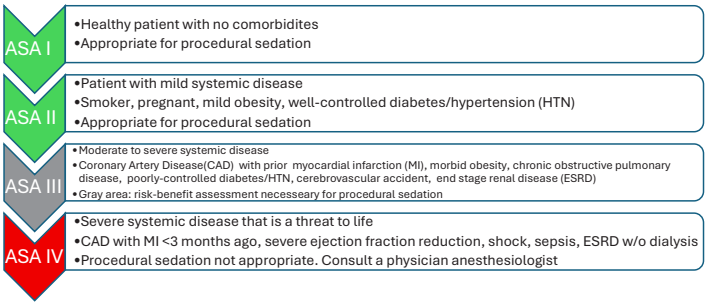


Figure. American Society of Anesthesiologist risk classifications (Practice Guidelines 2018).
ASA, American Society of Anesthesiologists

CASE REPORT

An 18-year-old man with a history of T1D presented with right shoulder injury following a moderate- speed MVC. He was an unrestrained passenger in a truck that swerved off the

road. The patient was able to self-extricate, and there was no loss of consciousness. He presented with severe right shoulder pain and a clearly visible deformity. The patient was placed in a cervical collar, and there were no signs of hemodynamic instability on primary survey.

On secondary survey, there was obvious tenderness to the right shoulder and upper arm. The patient's right arm was held in a position above his head, mimicking a raised hand, and he was unable to voluntarily lower his arm. Grip strength of the upper right extremity was 5/5. Laboratory tests indicated a glucose level of 496 mg/dL (reference range, 70-99 mg/dL), and the patient's mother reported that he was noncompliant with his insulin regimen at home, causing his sugars to run high consistently. Treatment options for hyperglycemia, including intravenous (IV) fluids and insulin administration, were discussed with the patient. He preferred to initiate management with IV fluids and glucose monitoring, noting that he had not eaten for at least six hours. From a clinical standpoint, this approach was considered reasonable, particularly in the setting of anticipated procedural sedation, where avoiding iatrogenic hypoglycemia was a priority. Laboratory tests were otherwise unremarkable, and there were no concerning physical signs of diabetic ketoacidosis. On exam, the patient had no abdominal pain, fruity odor on his breath, rapid/deep or labored breathing (Kussmaul respirations), confusion, or decreased alertness. Two IV lines were placed, and he received IV fluids and 75 mcg of fentanyl for significant pain and discomfort. Chest and right shoulder radiographs revealed a right shoulder inferior dislocation (luxatio erecta) with no evidence of fractures (Image 1).

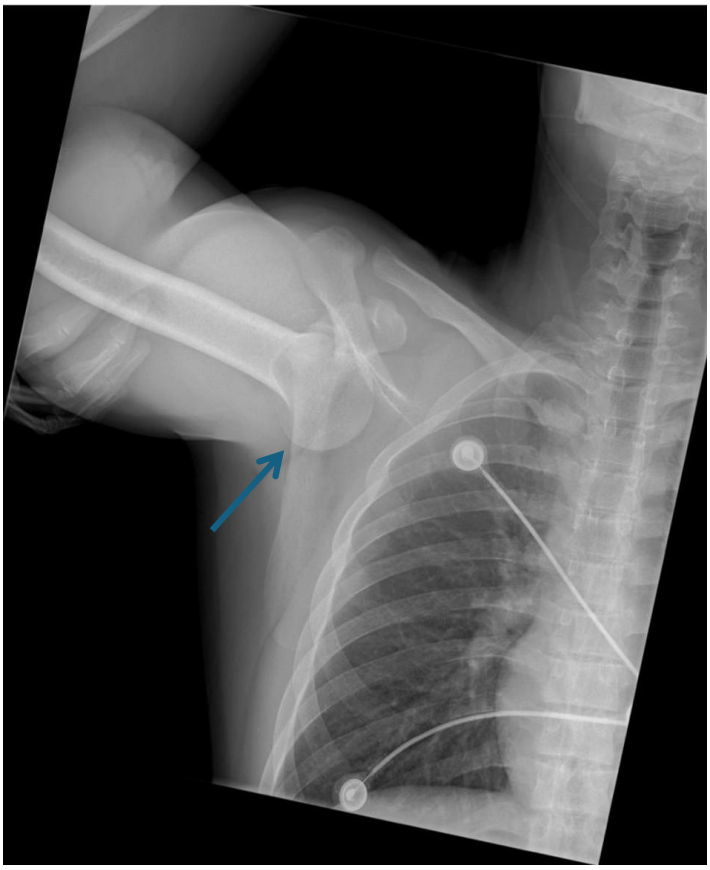


Image 1. Right shoulder radiograph showing inferior shoulder dislocation.

Considering a regional block approach to be safer in context of the scenario, an intraarticular injection of 1% lidocaine (without epinephrine) in the right shoulder was performed. The attempt at reduction after the regional block was unsuccessful, and procedural sedation was initiated with ketamine (60 mg) and midazolam (1 mg). The patient's right shoulder was subsequently reduced using traction-countertraction. The procedure was performed without difficulty or complication. Postprocedure, his right radial pulse was 2+ with a brisk capillary refill; he was able to give a thumbs up and he voiced normal sensation of his right hand. These physical examination findings helped support the presence of intact neurovascular structures. Postreduction computed tomography of the right shoulder suggested successful reduction of the luxatio erecta, but a comminuted greater tuberosity fracture was also discovered (Image 2). The patient was placed in a shoulder immobilizer postreduction.

Two and a half hours postreduction, the patient's glucose level was 340 mg/dL (reference range, 70-99 mg/dL). He was educated on the importance of glucose control, and after consulting orthopedics, he was discharged with an outpatient orthopedic follow-up scheduled.

DISCUSSION

Inferior shoulder dislocation, or *luxatio erecta*, is a rare glenohumeral dislocation, and forceful hyperabduction is one mechanism in which this injury can occur.[7] However, for our patient, direct axial load on an abducted arm that was bracing for impact in the setting of a MVC was likely the case. One interesting point of discussion surrounding inferior shoulder dislocations involves associated secondary injuries. Although understudied, a retrospective case series of 38 cases of luxatio erecta suggests an increased risk of concomitant injuries, potentially even greater than that seen in anterior shoulder dislocations.[14] Associated injuries can include Hill-Sachs lesions, greater tuberosity fractures, rotator cuff injuries, and neurovascular damage. The case series reports associated pathologies in 81% of inferior shoulder dislocations versus 60% of anterior shoulder dislocations and, more specifically, the authors reported an increased rate of associated greater tuberosity fractures in 34% of the patients.[14]

The decision for closed reduction under conscious sedation, which is the mainstay of treatment for shoulder dislocations, was a success. Although conscious sedation is frequently employed, we recommend an individualized approach when foregoing anesthesia consultation in patients who are borderline ASA physical status 3 or greater. This patient's severe hyperglycemia in the setting of T1D raised concern for

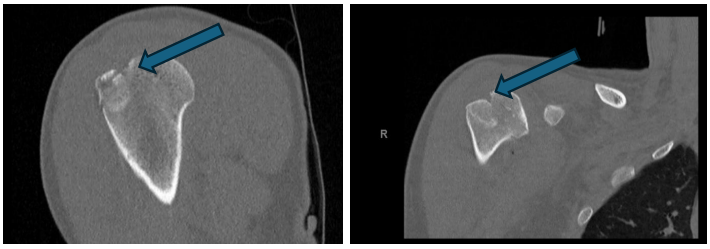


Image 2. Right shoulder computed tomography showing comminuted greater tuberosity fracture.

periprocedural risk. According to ADA and ASA guidelines, hyperglycemia does not contraindicate procedural sedation, provided that hyperglycemic emergencies have been ruled out and the patient is stable. This approach is supported by the American College of Emergency Physicians (ACEP) policy statement on unscheduled procedural sedation guidelines, which uses the ASA risk assessment as a cornerstone of its recommendations.[16] Clearly patients with severe systemic disease (greater than ASA physical status 3) are at greater risk for adverse events.[17-19] The ACEP guidelines endorse that even in high-risk patients, circumstances remain in which time-sensitive sedation is necessary.[16] This approach is particularly appropriate when resources do not permit the timely availability of an anesthesiologist or operating room without the risk of harm to the patient.[16] Additionally, this patient's otherwise normal laboratory values (specifically, the absence of acidosis), young age, and lack of comorbidities supported the decision for sedation in the ED. Ultimately, it was decided that the urgency of treating the dislocation outweighed the case-specific risks associated with conscious sedation for our patient.

Deferring insulin administration for the patient's initial hyperglycemia was clinically appropriate given the anticipated course of procedural sedation and his fasting state of more than 6 hours. Insulin could have been given to treat the hyperglycemia; however, this intervention would have increased the risk of iatrogenic hypoglycemia. Hypoglycemia-related neurological changes could be difficult to distinguish from typical sedation effects. In addition, a postprocedural neurological assessment of the right upper extremity could also be affected by hypoglycemia. Initial management with IV fluids alone can lead to significant glucose reduction by improving renal glucose clearance through increased renal perfusion and volume expansion,[15] as was observed in this patient. His glucose began trending down postreduction after the procedure. This self-resolution with IV fluids and the absence of other laboratory or physical examination abnormalities suggest a few possibilities, the most likely of which is that the patient had isolated hyperglycemia in the setting of insulin nonadherence or stress-induced hyperglycemia.The clinical team appropriately assessed and treated the patient's hyperglycemia in this situation.

From a procedural standpoint, intraarticular lidocaine followed by ketamine and midazolam were appropriate medication choices that aided in a successful reduction without complications. Midazolam was administered to reduce the risk of ketamine-associated emergence reactions. Regarding sedation choices and their impact on hyperglycemia, the published reports conflict. Several studies have shown that patients receiving midazolam and propofol experience increased blood glucose levels; however, at least one study evaluating blood glucose responses in light procedural sedation cataract surgery using either agent with fentanyl found no difference in blood glucose alterations.[20-23] There is consensus across these studies that avoidance of hypoglycemia is crucial, and that procedural stress, pain, and local anesthetics with epinephrine can increase blood glucose levels.[20-23]Although data guiding sedative agent selection in patients with significant hyperglycemia are limited, this case supports the safe use of ketamine with benzodiazepine adjunct, because no adverse events occurred.

CONCLUSION

While rare, luxatio erecta carries the risk of associated fractures and neurovascular injury that may not be initially apparent on imaging. Additionally, this case illustrates that while an ASA physical status of 3 is not an absolute contraindication to conscious sedation, it does necessitate careful assessment of patient stability and sedation strategies. This case demonstrates that closed reduction with conscious sedation can be safely performed in a young, otherwise healthy, severely hyperglycemic T1D patient with an inferior shoulder dislocation. However, individualized sedation decision-making in patients with an ASA physical status of 3 in the ED is recommended.

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The authors attest that their institution does not require Institutional Review Board approval for publication of this case report. Documentation on file. Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Marna R. Greenberg, DO MPH, Jefferson Health Lehigh Valley Region, Department of Emergency and Hospital Medicine, LVH-M-South 5th Floor EM Residency Suite, 2545 Schoenersville Road, Bethlehem, PA 18017. Email: Marna.Greenberg@jefferson.edu.

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Hemopericardium After Cardiopulmonary Resuscitation—Bedside Drainage with a Triple-Lumen Catheter: A Case Report

Michael Gagliardi, MD
Melissa Luu, MD
Rose-Ann Weick, DO
Rebecca White, MD
Mohammad T. Abboud, MD

Hackensack University Medical Center, Department of Emergency Trauma, Hackensack, New Jersey

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Introduction: Hemopericardium from chest compression-related cardiac injury is an underrecognized complication of cardiopulmonary resuscitation, with bleeding risk amplified in anticoagulated patients.

Case Report: A 66-year-old man on apixaban and hemodialysis presented in cardiac arrest. Post resuscitation ultrasound revealed pericardial tamponade. With no pericardiocentesis kit available, a seven French triple-lumen central venous catheter was placed under ultrasound guidance via a subxiphoid approach. Approximately one liter of bloody fluid was drained; the patient died within 24 hours.

Conclusion: Hemopericardium is an underrecognized complication of resuscitation in anticoagulated patients. A triple-lumen central venous catheter offers a familiar bedside drainage alternative when a commercial kit is unavailable. [Clin Pract Cases Emerg Med. 2026;10(4):528–532.]

Keywords: *pericardiocentesis; hemopericardium; cardiac arrest; case report.*

INTRODUCTION

Cardiac tamponade is a life-threatening condition caused by fluid buildup in the pericardial space, compressing the heart and causing hemodynamic instability. Hemopericardium arising from chest compression-related cardiac injury is an underrecognized complication of cardiopulmonary resuscitation (CPR), with risk amplified in patients receiving anticoagulation and those with comorbidities such as end-stage renal disease that further predispose to bleeding.[1,2] Emergency physicians should maintain a high index of suspicion for this etiology during and after prolonged resuscitation, because timely recognition guides both mechanical decompression and reversal of anticoagulation. Regardless of etiology, prompt pericardiocentesis allows drainage of this fluid, temporarily stabilizing the patient until definitive treatment, such as a pericardial window, can be performed.[3,4] While preparations are made for definitive intervention, an initial aspiration of as little as 50-100 mL can be sufficient to restore hemodynamic stability.[6]

In large or rapidly accumulating effusions, this approach may be insufficient; repeated aspirations may be needed to keep pace with ongoing fluid accumulation. In such cases, an emergency physician may consider placing a drainage catheter for larger volume, more frequent drainage.[5] Although commercial kits are available, they are often not accessible in the emergency department (ED), especially in resource-limited settings. Many emergency physicians are also more familiar with central venous catheters, such as triple-lumen catheters, than with specialized kits.

CASE REPORT

A 66-year-old man with hypertension, diabetes, coronary artery disease status post percutaneous intervention, atrial fibrillation on apixaban, and end-stage renal disease on hemodialysis presented to the ED for cardiac arrest. He had two to three days of abdominal pain that worsened suddenly,

leading to collapse, which was witnessed by bystanders who initiated CPR. Emergency medical services performed advanced cardiac life support, and the initial rhythm was noted to be asystole. Return of spontaneous circulation (ROSC) was achieved after 30 minutes of chest compressions and medications, including epinephrine, calcium chloride, sodium bicarbonate, and 250 mL of normal saline. He was intubated in the field using an 8.0-mL endotracheal tube, confirmed by bilateral breath sounds and capnography.

Upon arrival at the ED, the patient again became pulseless, and chest compressions were resumed. He received an additional 3 mg of epinephrine, 1 g of calcium chloride, and 1 ampule of sodium bicarbonate and achieved ROSC after eight minutes of chest compressions. Post-ROSC ultrasound showed a large pericardial effusion and signs of cardiac tamponade.

The patient became increasingly hypotensive, and an emergency pericardiocentesis was planned. Because a pericardiocentesis kit was not immediately available, an 18-gauge spinal needle was used to aspirate pericardial fluid using a 60 mL syringe. Given the large volume of effusion and the likelihood that multiple aspirations might be needed, a seven French (7-Fr) triple-lumen central venous catheter kit was prepared to replace the spinal needle using a guidewire. Using a subxiphoid approach under dynamic ultrasound guidance, 50 mL of bloody fluid was first aspirated through the spinal needle, resulting in immediate hemodynamic improvement. Aortic dissection was a consideration because the aspirate was sanguineous, warranting computed tomography (CT) for further evaluation. The guidewire was threaded through the spinal needle without resistance and visualized within the pericardial space on ultrasound.

After dilating the subcutaneous and muscular tissues, the 7-Fr triple-lumen catheter was placed over the wire. Correct positioning was confirmed by injecting agitated saline through the distal port, which produced echogenic material within the pericardial space without intracardiac opacification. An additional syringe of bloody fluid was drained, and the catheter was sutured in place on the chest wall (Image 1). Chest radiograph showed cardiomegaly with a "water-bottle" cardiac silhouette consistent with pericardial effusion, and the catheter projected appropriately over the cardiac silhouette (Image 2).

During his ED course, the patient experienced several episodes of hypotension that were alleviated by aspirating additional fluid from the central venous catheter. While in the CT scanner being evaluated for aortic dissection as a possible cause of his hemorrhagic pericardial effusion, the patient decompensated into pulseless electrical activity arrest. The emergency physician who accompanied him to the CT suite drained an extra 200 mL of fluid during CPR, and ROSC was achieved. The drainage catheter was connected to a Pleur-Evac on low continuous suction once available, and an additional 620 mL of fluid was drained, with ultrasound showing resolution of the pericardial effusion. The CT confirmed the drain was correctly placed in the pericardium and showed no evidence of aortic dissection (Image 3). The patient received andexanet alfa to reverse anticoagulation after weighing the risks and benefits in the setting of large-volume hemopericardium. He was admitted to the medical intensive care unit in stable but critical condition and ultimately died after 24 hours due to complications related to his illness.

CPC-EM Capsule

What do we already know about this clinical entity?
Cardiac tamponade is treated with urgent pericardiocentesis. Hemopericardium can result from anticoagulation, chest trauma, or cardiopulmonary resuscitation.

What makes this presentation of disease reportable?
Hemopericardium and tamponade developed in an anticoagulated patient after resuscitation, illustrating a potentially under-recognized complication of cardiopulmonary resuscitation (CPR).

What is the major learning point?
Consider hemopericardium in anticoagulated patients with hemodynamic instability after resuscitation. Point-of-care ultrasound guides rapid identification.

How might this improve emergency medicine practice?
Recognize CPR-related hemopericardium early with ultrasound; a triple-lumen catheter can serve as a bedside drainage option when a kit is unavailable.

DISCUSSION

The pericardium is a double-layered sac surrounding the heart, normally containing 10–50 mL of serous fluid. A pericardial effusion occurs when excess fluid accumulates within this space, most commonly due to infectious, metabolic, autoimmune, malignant, or traumatic processes. Clinical presentation varies widely, from asymptomatic to cardiac tamponade, a life-threatening condition marked by impaired cardiac filling that can lead to hemodynamic instability and death.[1-3] The primary treatment for tamponade physiology is urgent pericardiocentesis, performed via apical, parasternal, or subxiphoid approaches, to improve right ventricular filling.[6]

In large or rapidly accumulating effusions, draining a small volume (50–100 mL) may not be sufficient to maintain perfusion. In such situations, placing a drainage catheter allows for continuous decompression.[7,8] Commercial

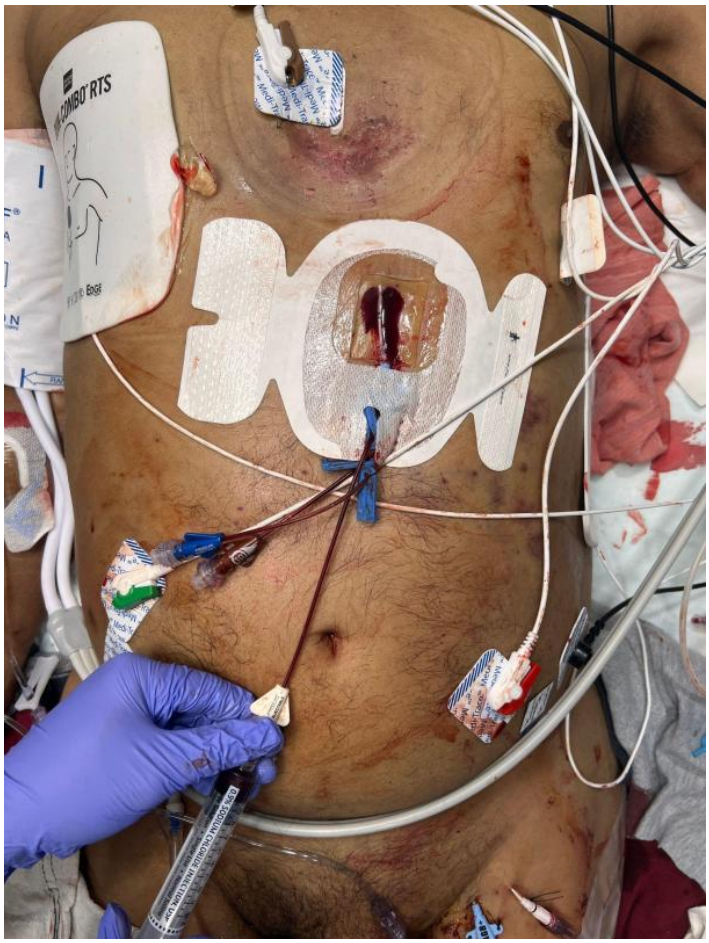


Image 1. Taken with the family's permission. A triple-lumen central venous catheter inserted through the chest wall into the pericardium with free-flow aspiration of bloody fluid through a port into a syringe.



Image 2. Taken with the family's permission. A triple-lumen central venous catheter inserted through the chest wall into the pericardium with free-flow aspiration of bloody fluid through a port into a syringe.

pericardial drainage kits are not always easily accessible, especially in urgent or resource-limited settings. The use of central venous catheters for pericardial drainage has been described in rural and resource-limited settings[9-11] and is a familiar bedside option for emergency physicians. In our case, a triple-lumen central venous catheter served as a practical bedside alternative when a commercial pericardiocentesis kit was not immediately available.

The etiology of hemopericardium in this case warrants consideration. The patient was anticoagulated with apixaban for atrial fibrillation in the setting of end-stage renal disease on hemodialysis, a combination associated with higher steady-state drug exposure and increased bleeding risk compared to patients with preserved renal function.[12] Spontaneous hemopericardium is a rare but increasingly recognized complication of direct oral anticoagulant therapy, with an estimated incidence of 0.02–0.069%, and apixaban has been implicated in a growing case literature.[13,14] The prolonged resuscitation course, however, makes CPR-related cardiac injury the most likely primary or amplifying mechanism. Hemopericardium has been documented in approximately 8–9% of forensic autopsy cases following CPR, and longer chest compression duration has been identified as an independent predictor of thoracic injuries overall.[1, 2]

Approximately 38 minutes of cumulative chest compressions were delivered between the field arrest and the second ED arrest before tamponade was identified. This case raises the possibility that CPR-induced thoracic trauma, which causes pericardial and myocardial injury in up to 17% of autopsy cases,[1] may be sufficient to produce clinically significant hemopericardium in fully anticoagulated patients, even in the absence of gross cardiac laceration. Clinicians should maintain a high index of suspicion for pericardial tamponade when return of spontaneous circulation is not achieved, or hemodynamic instability persists following CPR in anticoagulated patients. Whether hemopericardium preceded the initial cardiac arrest or developed as a consequence of prolonged resuscitation in a fully anticoagulated patient cannot be determined with certainty; however, in either scenario, CPR would be expected to worsen pericardial hemorrhage and accelerate progression to tamponade physiology, underscoring the importance of early echocardiographic evaluation during resuscitation of anticoagulated patients.

The use of a central venous catheter as a pericardial drain offers both advantages and limitations in this context. Unlike a pigtail catheter, which has multiple side ports along its curl to maximize drainage, the triple-lumen catheter has a single

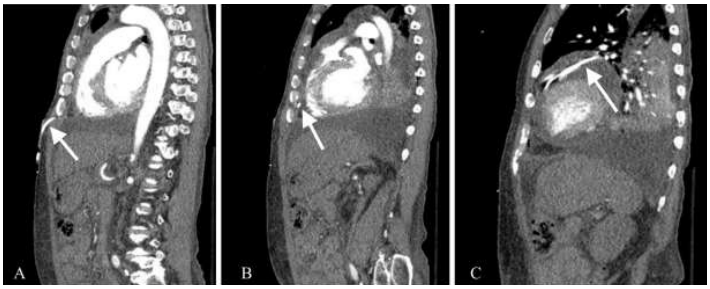


Image 3. Sagittal computed tomography images confirming the location of the drainage catheter highlighted with white arrows entering the skin via a subxiphoid approach (A), entering the pericardium (B), and within the pericardial space (C).

distal-end hole and two staggered side ports that serve its medial and proximal lumens. These openings are configured for venous infusion rather than fluid drainage, which may increase the risk of obstruction from fibrin, particulate debris, or clotted blood, leading to insufficient flow. Nevertheless, its quick availability and operator familiarity make it a useful temporary option, especially when commercial kits are delayed or inaccessible.

As the population of patients with atrial fibrillation, end-stage renal disease, and chronic direct oral anticoagulant use continues to grow, presentations similar to this case are likely to become more frequent in the ED. Future research should clarify the diagnostic and therapeutic approach to cardiac arrest in anticoagulated patients, including the role of point-of-care echocardiography in early identification of CPR-related hemopericardium, the optimal timing of factor Xa inhibitor reversal during ongoing resuscitation, and the comparative effectiveness of bedside drainage strategies when commercial kits are unavailable.

CONCLUSION

This case illustrates hemopericardium as an underrecognized complication of CPR in anticoagulated patients. Early identification with point-of-care ultrasound, prompt pericardial decompression, and reversal of anticoagulation are priorities in management; when a commercial pericardiocentesis kit is not immediately available, a triple-lumen central venous catheter provides a familiar bedside drainage alternative.

TEACHING POINTS

- Consider hemopericardium in anticoagulated patients with hemodynamic instability after cardiopulmonary resuscitation.
- Use point-of-care ultrasound during and after resuscitation to identify pericardial tamponade as a treatable cause of refractory shock.
- Pursue mechanical decompression and anticoagulation reversal in parallel when tamponade is identified in an anticoagulated patient.
- Use a triple-lumen central venous catheter for bedside pericardial drainage when a commercial pericardiocentesis kit is unavailable.

Family consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Mohammad T. Abboud, MD, Hackensack University Medical Center, Department of Emergency Trauma, 30 Prospect Avenue, Hackensack, NJ 07601. Email: mohammad.abboud@hmhn.org.

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Inferior Vena Cava Cement Migration with Pulmonary Cement Embolism: A Case Report

Christina Sneed, MD
Syed Adnan, MD
Jefferson Einstein Montgomery Hospital, Department of Emergency Medicine, Norristown, Pennsylvania

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Introduction: Vertebral augmentation procedures, including vertebroplasty and kyphoplasty, can result in cement extravasation, which is often clinically silent. Less commonly, cement may migrate intravascularly into the inferior vena cava (IVC) and pulmonary arterial circulation, resulting in pulmonary cement embolism, a potentially life-threatening complication. Early recognition in the emergency department (ED) is essential to guide appropriate imaging, specialty consultation, and timely management.

Case Report: A 76-year-old woman with recent third lumbar vertebral augmentation presented to the ED with acute worsening low back pain and burning bilateral radicular symptoms approximately one week after the procedure. Computed tomography (CT) of the abdomen and pelvis with intravenous contrast demonstrated linear hyperdense material extending from the treated vertebral body into the IVC, concerning for intravascular cement migration. Additional CT of the chest demonstrated multiple bilateral subsegmental pulmonary cement emboli. Interventional radiology was consulted emergently, and the patient was transferred for endovascular retrieval of the IVC cement fragment, which was successfully removed. The pulmonary cement emboli were small, peripheral, and asymptomatic and were managed conservatively. The patient remained hemodynamically stable and was discharged after monitored hospitalization.

Conclusion: Inferior vena cava cement migration with pulmonary cement embolism is a potentially life-threatening complication of vertebral augmentation. Emergency physicians should maintain suspicion for this diagnosis in patients with acute pain or new cardiopulmonary symptoms following recent spinal procedures. Early imaging, multidisciplinary consultation, monitored admission, and endovascular retrieval when indicated may reduce morbidity. [Clin Pract Cases Emerg Med. 2026;10(4):533–536.]

Keywords: *pulmonary embolism; vertebral augmentation; kyphoplasty; cement.*

INTRODUCTION

Vertebral augmentation procedures, including vertebroplasty and kyphoplasty, are widely performed for osteoporotic compression fractures. Cement leakage is common, with reported rates of 30–70%, although most cases are asymptomatic and clinically insignificant.[1,2] Venous extravasation through the vertebral venous plexus can result in migration into segmental veins, the inferior vena cava (IVC), and pulmonary arterial circulation.[3] Pulmonary cement embolism is the most recognized serious vascular complication, with reported incidence of 3–23%.[4] While many emboli are clinically silent, larger fragments can cause hypoxia, right ventricular strain, or cardiovascular collapse.[3,5] Although pulmonary cement embolism has been reported after vertebral augmentation, cement migration into the IVC is less frequently described and appears to be limited to isolated case reports and small series, yet it may pose substantial risk of distal embolization requiring urgent intervention.[6,9]

CASE REPORT

A 76-year-old woman with a history of transcatheter aortic valve replacement, atrial fibrillation, congestive heart

failure, and recent third lumbar (L3) vertebral compression fracture status post vertebral augmentation approximately one week prior presented to the emergency department (ED) with acute worsening low back pain and burning bilateral radicular symptoms.

The vertebral augmentation had been performed in an outpatient setting, and procedural records and current medication records were unavailable at the time of presentation. On arrival, vital signs were as follows: temperature, 36.8 °C; heart rate, 117 beats per minute; respiratory rate, 18 breaths per minute; blood pressure, 126/66 mm Hg; and oxygen saturation, 95–99% on room air. Her vital signs remained stable during the ED course prior to transfer, with heart rate ranging from the 90s to a maximum of 120 beats per minute.

The patient was awake, alert, appropriately conversational, nontoxic appearing, and in no acute cardiopulmonary distress. Pulmonary examination demonstrated equal bilateral breath sounds without rales or rhonchi. Cardiovascular examination demonstrated tachycardia with regular rhythm. Physical examination revealed extensive ecchymoses over the abdomen and posterior trunk, midline tenderness of the mid-to-lower lumbar spine, and gluteal tenderness over the hematoma site. Neurologic examination demonstrated intact sensation and 5/5 motor strength in the bilateral upper and lower extremities without saddle anesthesia. Gait assessment was deferred because of pain.

Laboratory evaluation demonstrated hemoglobin of 8.3 g/dL (reference range, 12.0–16.0 g/dL) without prior values available for comparison, limiting determination of chronicity. High-sensitivity troponin levels were minimally elevated but stable on serial testing at 22, 18, and 18 ng/L (≤13 ng/L), and brain natriuretic peptide was elevated at 326 pg/mL (0.0–100.0 pg/mL), concerning for possible right heart strain. Electrocardiogram demonstrated sinus tachycardia without ischemic changes, and the stable serial cardiac biomarkers were not suggestive of acute myocardial ischemia. There were no electrolyte derangements, acute kidney injury, or leukocytosis suggestive of infectious pathology.

Computed tomography (CT) of the abdomen and pelvis with intravenous contrast demonstrated postsurgical changes consistent with prior vertebral cement augmentation at L3 and linear hyperdense material extending from the treated vertebral body into the IVC, concerning for intravascular cement migration (Image 1). The study also demonstrated a 5.7 × 12.1 cm hematoma in the left superior gluteal region with surrounding subcutaneous edema.

Given concern for distal embolization, interventional radiology was consulted emergently. After discussion with the consulting team, CT of the chest without contrast demonstrated multiple branching high-attenuation foci within bilateral subsegmental pulmonary arteries, consistent with pulmonary cement emboli (Image 2). Additional findings included an age-indeterminate 12th thoracic vertebral compression deformity without bony retropulsion and incidental pulmonary nodules. The patient was transferred to a tertiary care center where interventional radiology successfully performed endovascular retrieval of the IVC cement fragment without complication. The pulmonary cement emboli were small, subsegmental, and asymptomatic and were managed conservatively.

Postprocedure, the patient remained hemodynamically stable. Anticoagulation was initially held because of the large gluteal hematoma and extensive ecchymoses. Transthoracic echocardiogram demonstrated a left ventricular ejection fraction of 75%, a dilated right ventricle with moderately reduced right ventricular function, severe mitral stenosis, and a normally functioning transcatheter aortic valve replacement. Cardiology recommended outpatient follow-up for mitral stenosis management. Given her history of atrial fibrillation, warfarin with enoxaparin bridging was later initiated, targeting an international normalized ratio of 2–3. The patient was monitored on telemetry during hospitalization and discharged on hospital day 10. She was subsequently lost to follow-up.

CPC-EM Capsule

What do we already know about this clinical entity?
Pulmonary cement embolism is an uncommon but recognized complication of vertebral augmentation procedures.

What makes this presentation of disease reportable?
Computed tomography showed cement migration from a lumbar vertebral augmentation site into the inferior vena cava and bilateral pulmonary arteries.

What is the major learning point?
Prior vertebral augmentation should prompt consideration of cement embolism, especially when characteristic hyperdense intravascular findings are present.

How might this improve emergency medicine practice?
Awareness of this rare complication may prevent missed diagnoses and facilitate timely consultation and management.

DISCUSSION

Cement leakage is a frequent radiographic finding after

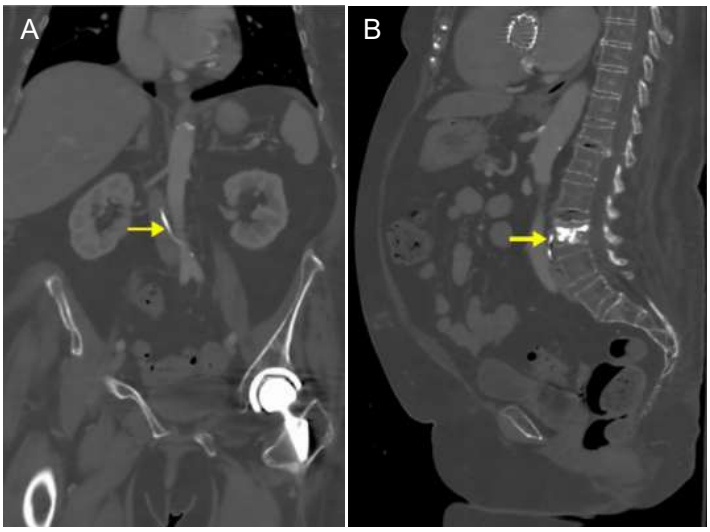


Image 1. A) Coronal and B) sagittal contrast-enhanced computed tomography of the abdomen and pelvis demonstrating linear hyperdense cement extending from the third lumbar vertebral augmentation site into the inferior vena cava (arrow), concerning for intravascular cement migration.

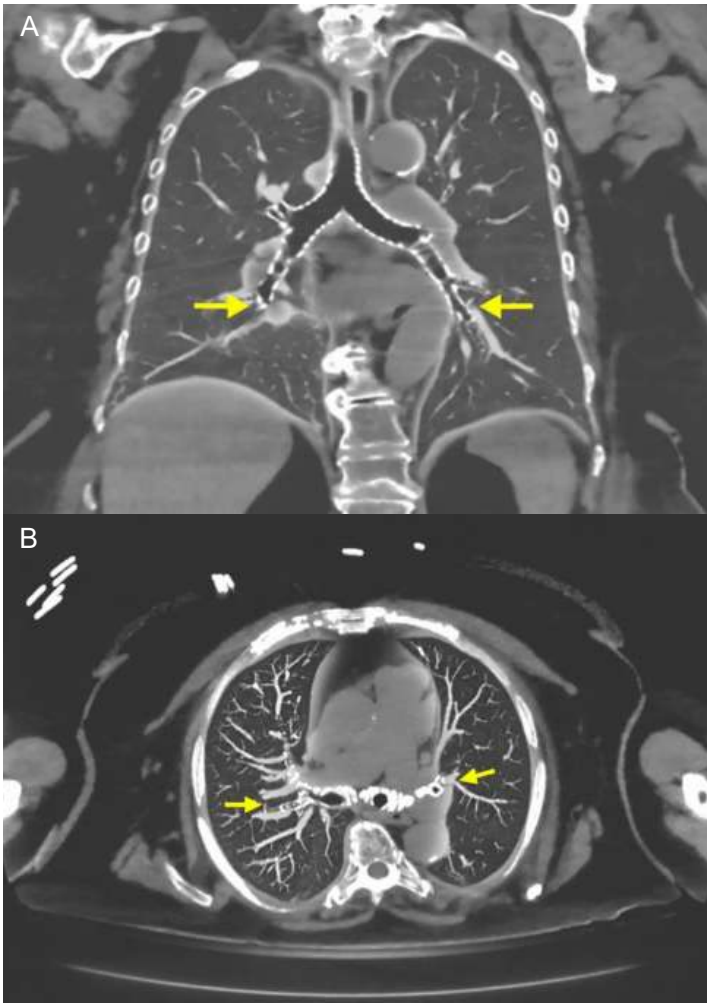


Image 2. A) Coronal and B) axial contrast-enhanced computed tomography demonstrating branching hyperattenuating cement emboli within bilateral subsegmental pulmonary arteries (arrows).

vertebral augmentation, occurring in 30–70% of procedures, although intravascular migration is less common.[1,2] Fragments can travel through the vertebral venous plexus into the IVC, with distal pulmonary embolization reported after vertebral augmentation in the literature.[3-5,7] Pulmonary cement embolism is the most frequently reported serious vascular complication of vertebral augmentation.[3-5] Although many pulmonary cement emboli are asymptomatic, emboli that are larger, central, or multiple can be life-threatening, particularly in patients with pre-existing cardiopulmonary disease.[3,5,8] Mechanistically, embolized polymethylmethacrylate can cause mechanical obstruction of the pulmonary arterial vasculature, increasing pulmonary vascular resistance and precipitating right ventricular strain or hemodynamic compromise. In addition, rigid cement fragments may continue to migrate centrally into the right heart, where they can cause arrhythmia, valvular injury, cardiac perforation, hemopericardium, tamponade, or death. Some reports also suggest endothelial injury and secondary thrombosis may further contribute to cardiopulmonary sequelae.[5,6,8,9]

In this case, the patient's history of transcatheter aortic valve replacement, atrial fibrillation, and postprocedural echocardiographic evidence of right ventricular dilation with moderately reduced right ventricular function increased concern for possible hemodynamic compromise despite the absence of overt hypoxia or instability.

The vertebral level of augmentation may influence the risk and pattern of cement migration due to venous anatomy. The L3 vertebral body drains into the internal vertebral venous plexus, which communicates directly with the IVC, providing a relatively short route for embolization.[3,7] In this patient, the L3 origin may have facilitated migration into the pulmonary arterial circulation. The combination of an IVC cement fragment and multiple pulmonary cement emboli, along with echocardiographic evidence of right ventricular dysfunction, supported prompt interventional radiology consultation, monitored admission, and urgent endovascular retrieval of the intravascular fragment.[6,9]

Management of pulmonary cement embolism depends on symptom burden, embolus location, and hemodynamic status. Small, peripheral, and asymptomatic emboli may be managed conservatively, whereas symptomatic, central, or hemodynamically significant emboli may require anticoagulation or urgent interventional retrieval.[5,8] Endovascular retrieval of IVC cement fragments is increasingly described in the literature, and dual venous access with large-bore sheath protection may reduce the risk of distal embolization during retrieval.[6,9]

This case highlights several important considerations for emergency physicians. Recent vertebral augmentation should

prompt consideration of intravascular cement migration in patients presenting with acute back pain, radicular symptoms, unexplained cardiopulmonary findings, or suspected pulmonary embolic phenomena, particularly because traditional thromboembolic risk stratification tools may not account for nonthrombotic complications such as pulmonary cement embolism. Early CT can identify both the source fragment and distal embolization. Prompt multidisciplinary consultation with interventional radiology and monitored admission are essential when embolization risk exists. Finally, anticoagulation decisions in pulmonary cement embolism should be individualized, particularly when concurrent bleeding risk is present.

CONCLUSION

Inferior vena cava cement migration with pulmonary cement embolism is a rare but potentially life-threatening complication of vertebral augmentation. Emergency physicians should consider this diagnosis in patients presenting after recent spinal procedures with acute pain or unexplained cardiopulmonary symptoms. Early imaging, specialty consultation, and monitored management may reduce morbidity.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Christina Sneed, MD, Jefferson Einstein Montgomery Hospital, Department of Emergency Medicine, 559 W Germantown Pike, Norristown, PA 19403. Email: christina.sneed@jefferson.edu.

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Atypical Presentation of Extensive Stanford Type A Aortic Dissection Initially Misattributed to Aspiration Pneumonia: A Case Report

Margalit Goldgof, MD*†
Ahmad Nama, MD*†
Waseem Mujahed, MD*†

*Hadassah University Medical Center Ein Kerem, Department of Emergency Medicine, Jerusalem, Israel
†Hebrew University of Jerusalem, Faculty of Medicine, Jerusalem, Israel

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Introduction: Acute aortic dissection is a life-threatening emergency that may present with nonspecific symptoms, particularly in older adults, leading clinicians to anchor on more common diagnoses.

Case Report: An 85-year-old man presented with four days of cough, weakness, and constipation after a recent emergency department (ED) visit due to food impaction after he choked on a carrot. A chest radiograph obtained the day before admission showed left basal consolidation, prompting referral for suspected aspiration pneumonia. In the ED, he appeared comfortable with no complaints of chest pain. Physical examination noted mild tachycardia and wheezes bilaterally and treatment was initiated with antibiotics and bronchodilators. A low-voltage electrocardiogram led to immediate point-of-care echocardiography, which demonstrated a large circumferential pericardial effusion, a markedly dilated aortic root, and right ventricular collapse. Computed tomography angiography confirmed an extensive Stanford Type A aortic dissection.

Conclusion: This case highlights how deceptively calm presentations can mask catastrophic disease and why clinicians must be ready to abandon their first diagnosis when the data demands it. [Clin Pract Cases Emerg Med. 2026;10(4):537–541.]

Keywords: *aortic dissection; misdiagnosis; atypical presentation; aspiration pneumonia; case report.*

INTRODUCTION

Acute aortic dissection carries high early mortality, particularly for Stanford Type A dissections, and outcomes depend critically on rapid recognition and surgical management. Classical symptoms include sudden, severe chest or back pain, pulse deficits, and hemodynamic instability, yet up to one-third of patients present atypically, especially older adults. Their complaints may be vague -weakness, dyspnea, abdominal discomfort, or gastrointestinal symptoms - leading to misdiagnosis such as pneumonia, acute coronary syndrome, or gastrointestinal disease.[1,2]

We report an extensive Stanford Type A aortic dissection initially attributed to aspiration pneumonia, emphasizing practical diagnostic lessons for emergency physicians: the need to maintain a broad differential; interpret electrocardiograms (ECG) meticulously (including low voltage), use bedside echocardiography early, avoid anchoring on an initial working diagnosis, and be aware of nuanced presentation in hemodynamic management when structural complications are present.

CASE REPORT

An 85-year-old man with hypertension, hypothyroidism, and benign prostatic hyperplasia presented to the emergency department (ED) with four days of cough, generalized weakness, and constipation. He denied fever, chills, or dyspnea. Ten days earlier, he presented to another hospital with acute chest and epigastric discomfort immediately after

swallowing a carrot, followed by two episodes of vomiting. At that visit, his blood pressure was 170/88 mm Hg; laboratory tests showed mildly elevated inflammatory markers, and a troponin level of 30 ng/L (upper limit of normal) (reference range, 0-35 ng/L). No imaging was performed, and he was discharged after symptomatic treatment.

The day before the current presentation, his family physician obtained a chest radiograph that showed left basal consolidation and blunting of the left costophrenic angle, raising concern for aspiration pneumonia and prompting referral to the ED. On arrival, the following vital signs were measured: heart rate 107 beats/minute (bpm); blood pressure 133/91 mm Hg; respiratory rate 18 breaths/minute; temperature 36.7 °C; and oxygen saturation 94% on room air. He appeared comfortable and cooperative. Cardiovascular examination revealed regular tachycardia and an apical heart murmur. Pulmonary auscultation demonstrated crackles at the right lung base and mild bilateral wheezes. The abdomen was soft, distended, and non-tender; there was no peripheral edema.

Laboratory studies showed no electrolytes abnormalities and stable creatinine 0.7 mg/dL (reference range, 0.55-1.02 mg/dL); liver enzymes were not elevated. Hemoglobin was 10.8 g/dL (down from 15 g/dL five months earlier) (12.4-16.1 g/dL), and platelets were mildly elevated at 478 ×10⁹/L (203-445⁹/L). Troponin was significantly elevated at 107 ng/L (55-58 ng/L). The initial ECG was of poor quality but showed no obvious ischemic ST-segment changes; however, repeat ECG was requested.

Given the presumed diagnosis of pneumonia, intravenous ceftriaxone and bronchodilator therapy were initiated. Because of persistent tachycardia and elevated troponin, computed tomography (CT) pulmonary angiography was ordered. Before imaging, a repeat ECG demonstrated markedly low voltage (Image 1), prompting point-of-care transthoracic echocardiography. The ECG revealed a large circumferential pericardial effusion with radiologic signs of tamponade (right ventricular collapse, video) and a markedly enlarged aortic root causing significant aortic insufficiency.

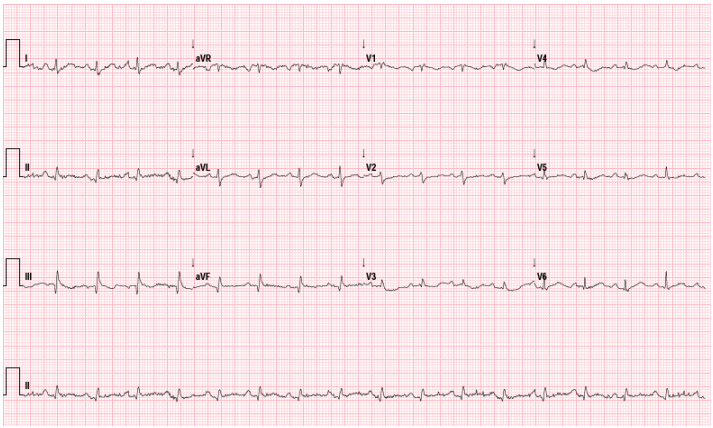


Image 1. Twelve-lead electrocardiogram demonstrating sinus tachycardia with diffuse low-amplitude QRS complexes across limb and precordial leads. The overall low-voltage pattern is consistent with findings that may be seen in pericardial effusion or tamponade physiology.

Emergent CT angiography of the chest, abdomen, and pelvis demonstrated an extensive Stanford Type A aortic dissection originating at the aortic root and extending through the ascending aorta, arch, and descending thoracic aorta, and propagating into the left subclavian artery, celiac and mesenteric arteries, and left iliac artery (Image 2). Despite the extensive dissection, blood pressure remained stable and symmetric in both arms.

Cardiology and cardiothoracic surgery were urgently consulted. The patient underwent emergent surgical repair of the ascending aorta and aortic root. His postoperative course was uncomplicated, and he was discharged home 10 days later.

CPC-EM Capsule

What do we already know about this clinical entity?
Type A aortic dissection often presents atypically in older adults and may mimic common conditions. Subtle symptoms do not exclude life-threatening pathology.

What makes this presentation of disease reportable?
The patient presented with classical symptoms of pneumonia: cough; weakness; and consolidation on chest radiograph; despite extensive Type A dissection with tamponade.

What is the major learning point?
Avoid anchoring on an initial diagnosis and be ready to pivot quickly when new data do not fit the presumed condition.

How might this improve emergency medicine practice?
Broad differentials, access to prior records, and point-of-care echocardiography improve diagnostic accuracy and can rapidly redirect care toward lifesaving intervention.

DISCUSSION

Isolated case reports describe aortic dissection presenting with or mimicking pneumonia, aspiration events, or other pulmonary conditions, including dyspnea-dominant or painless presentations.[3] Reports of dissection temporally

associated with choking or food impaction are rare,[4] but the mechanism is plausible: a sudden rise in blood pressure or intrathoracic pressure during a Valsalva-like event could precipitate dissection in a susceptible aorta. Extensive Type A dissections involving arch vessels, mesenteric arteries, and iliac arteries have been documented,[5] however; they are uncommon. Survival to discharge, as in this case, is notable.

To understand how this diagnosis could have been recognized earlier, it is essential to break down the case and highlight the key clinical clues and lessons that emerged.

Atypical Presentation and Symptom Interpretation

This case illustrates how aortic dissection can masquerade as common conditions in older adults. The patient's presenting symptoms - cough, generalized weakness, and radiographic consolidation - were highly compatible with pneumonia, a far more frequent diagnosis in this age group. However, his earlier episode of abrupt chest and epigastric pain after swallowing food may have represented the onset of dissection rather than simple esophageal irritation.

Importantly, even his "minor" complaints, such as a week of constipation and abdominal distension, are retrospectively explainable by extension of the dissection into the mesenteric and celiac territories, with possible mesenteric hypoperfusion.[6]

Troponin, Hemoglobin, and the First Presentation

At the initial hospital visit, the troponin level was at the upper limit of normal (30 ng/L) in a patient without known coronary disease. In isolation, a borderline troponin in an elderly hypertensive patient with chest/epigastric discomfort is an easy miss; many clinicians would reasonably attribute it to demand ischemia, gastroesophageal disease, or noncardiac causes. However, in retrospect, this value should not be dismissed outright—particularly when combined with severe acute symptoms and elevated blood pressure. Current guidelines emphasize that troponin elevation can occur in proximal aortic dissection and may be an indicator of dissection among patients with atypical symptoms.[7]

Similarly, the drop in hemoglobin from 15 g/dL to 10.8 g/dL over several months, although not dramatic, could be a subtle clue to chronic or subacute blood loss or ongoing pathology. That it was not recognized at the first encounter underscores how easily such trends are missed when prior records are not readily available or not reviewed under time pressure.

Health-Record Fragmentation and Diagnostic Safety

The emergency team that ultimately diagnosed the dissection had access to prior hemoglobin values and the earlier troponin result; the first hospital may not have had the same longitudinal view or may not have had time to integrate it. Interoperable health records and concise, high-yield summaries of prior investigations are critical for pattern recognition and for avoiding missed opportunities in time sensitive conditions.

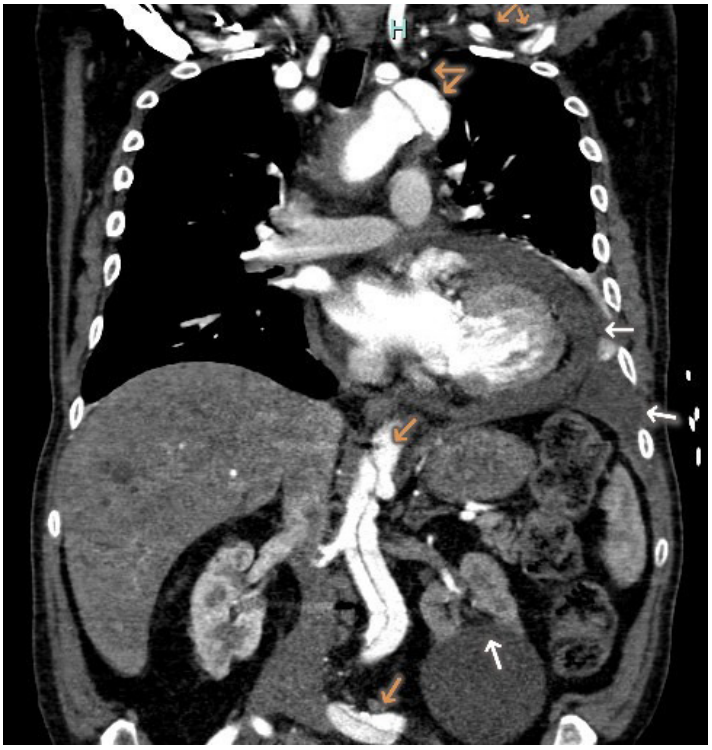


Image 2. Computed tomography angiography image demonstrating an aortic root dissection extending to ascending aorta, left subclavian artery, descending aorta, and left iliac artery (yellow arrows). Circumferential pericardial effusion, left pleural effusion, and poor left renal perfusion were also noted (white arrows).

Electrocardiogram, Low Voltage, and the Role of Point-of-Care Echocardiography

A central learning point from this case is the importance of meticulous ECG interpretation. Low-voltage ECG is often underappreciated but can signal pericardial effusion, obesity, chronic obstructive pulmonary disease, or infiltrative lung disease. In the context of unexplained tachycardia, elevated troponin, and a nonischemic ECG, low voltage should prompt urgent echocardiography to evaluate for pericardial effusion and structural pathology. In this patient, the decision to obtain point-of-care echocardiography based on low voltage was the pivotal step that redirected the diagnostic pathway and led to lifesaving intervention.

Point-of-care ultrasound has been shown to decrease misdiagnosis, as well as expedite diagnosis of ascending aortic dissection.[8,9]

Cognitive Bias and the Need to Pivot from an Assumed Diagnosis

The case also illustrates diagnostic anchoring: once aspiration pneumonia was suspected - supported by cough, consolidation on chest radiograph, and wheezes - subsequent data were initially interpreted through that lens. Yet several features did not fit:

- Unexplained tachycardia in an afebrile, normoxic patient
- Elevated troponin without ischemic ECG changes
- Low-voltage ECG, suggestive of pericardial effusion
- Anemia and prior borderline troponin in a patient with recent chest/epigastric pain

Emergency clinicians must cultivate the habit of asking, "What doesn't fit?" and be willing to rapidly pivot when new information conflicts with the working diagnosis. Literature on diagnostic error in emergency medicine repeatedly identifies anchoring and premature closure as major contributors to missed aortic dissection.[10]

Nuanced Hemodynamic Management

Standard teaching emphasizes reducing aortic wall stress by lowering heart rate (< 60 bpm) and systolic blood pressure (< 120 mm Hg). Opioids can assist early by relieving pain and anxiety, thereby decreasing sympathetic drive. Vasodilators should only be introduced after adequate heartrate control (typically with beta-blockers) to prevent reflex tachycardia;[2] however, in acute aortic regurgitation or tamponade, compensatory tachycardia is essential for maintaining cardiac output and suppressing it may precipitate rapid hemodynamic collapse.[10] In complex presentations such as this, physiological priorities shift from limiting dissection progression to preserving the patient's fragile compensatory state. Accordingly, the cardiothoracic surgeon recommended against hemodynamic manipulation. If hemodynamic collapse had occurred, controlled pericardial drainage (removing very small volumes of less than 30 mL) could have provided temporary stabilization, but definitive treatment remains expedited surgical intervention.[11]

CONCLUSION

This case demonstrates how an extensive Stanford Type A aortic dissection can present with cough, generalized weakness, constipation, and radiographic consolidation - features easily attributed to pneumonia.

Key lessons include the following:

- All chest and epigastric pain and dyspnea should trigger consideration of aortic pathology, especially in older adults.
- Atypical symptoms in older adults - such as weakness, cough, or constipation—do not exclude life-threatening pathology, including aortic dissection.
- Low-voltage ECG should prompt urgent echocardiography, particularly in patients with chest or epigastric pain, dyspnea, or elevated troponin.
- Borderline troponin without ischemic ECG changes warrants a broader differential, including aortic dissection.
- Subtle symptoms such as constipation and abdominal distension may reflect mesenteric involvement in extensive dissections.
- Emergency clinicians must avoid anchoring on an initial diagnosis and be ready to pivot quickly when new data do not fit the presumed condition.
- Point-of-care echocardiography is a decisive tool that can rapidly redirect the diagnostic pathway and facilitate lifesaving intervention.

Early use of point-of-care echocardiography and a disciplined approach to conflicting clinical information can facilitate timely recognition and treatment of this life-threatening condition, as in this case, where the patient survived and was discharged home 10 days after surgery.

Video. Point-of-care five-chamber echocardiographic view illustrating a large circumferential pericardial effusion (white asterisk). The right ventricle demonstrates diastolic collapse (arrow), and the aortic outflow tract is visualized anteriorly (black asterisk).

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Margalit Goldgof, MD, Hadassah University Medical Center Ein Kerem, Department of Emergency Medicine, Kalman Ya'akov Man St., Jerusalem, Israel, 9112001. Email: mgoldgof@hadassah.org.il.

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Polysubstance-Induced Vasoplegia Managed with Venoarterial Extracorporeal Membrane Oxygenation: A Case Report

William J. Bleifuss, MD*
Aaron E. Robinson, MD, MPH†
Beth M. Heather, MSN‡
Hannah M. Brown, PhD§
Tyler G. Floden, MD¶
Jon B. Cole, MD*†

*Hennepin County Medical Center, Minnesota Regional Poison Center, Minneapolis, Minnesota
†Hennepin County Medical Center, Department of Emergency Medicine, Minneapolis, Minnesota
‡Hennepin County Medical Center, Critical Care Nursing and the Extracorporeal Life Support Program, Minneapolis, Minnesota
§Hennepin County Medical Center, Department of Laboratory Medicine and Pathology, Minneapolis, Minnesota
¶Hennepin County Medical Center, Department of Critical Care Medicine, Minneapolis, Minnesota

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Introduction: While the use of venoarterial extracorporeal membrane oxygenation (VA-ECMO) becomes increasingly commonplace in overdose, questions about its role in various shock states remain. The application of VA-ECMO with impaired myocardial function is well established, and use in cardiogenic shock precipitated by poisoning has continued to increase. In poison-induced vasoplegia with preserved myocardial function, the role of VA-ECMO remains undefined. Many atypical antipsychotic and antidepressant agents are known to cause mild hypotension via alpha-1 adrenergic blockade. Intravenous fluids and vasopressors alone are generally effective for single-substance ingestions. In the setting of polysubstance intoxication, synergism leading to bradycardia and refractory vasoplegia refractory to conventional therapies may result, prompting consideration for extracorporeal support.

Case Report: A 71-year-old woman presented to the emergency department (ED) after being found obtunded near bottles of quetiapine, olanzapine, trazodone, mirtazapine, levomilnacipran, eszopiclone, and clonazepam. She underwent prehospital intubation and received push-dose epinephrine for hypotension. In the ED, the patient remained hypotensive with a relative bradycardia, requiring escalating doses of norepinephrine, vasopressin, epinephrine, angiotensin II, and methylene blue infusions. Her extremities remained warm, and point-of-care echocardiography revealed hyperdynamic function, consistent with vasoplegic shock. She subsequently underwent VA-ECMO cannulation, which was followed by a rapid decrease in vasopressor requirements and decannulation on hospital day 3. The patient recovered and was transferred to inpatient psychiatry neurologically intact. Urine mass-spectrometry revealed large peaks for quetiapine, olanzapine, milnacipran, mirtazapine, trazodone, and zopiclone.

Conclusion: While overdose data are limited, levomilnacipran may precipitate or potentiate relative bradycardia with hypotension and a preserved ejection fraction. Significant ingestions and synergism with other agents causing peripheral alpha-1 antagonism may lead to hemodynamic instability refractory to conventional therapies. Extracorporeal life support may aid in recovery. [Clin Pract Cases Emerg Med. 2026;10(4):542–546.]

Keywords: *levomilnacipran; angiotensin II: vasoplegia; ECMO; case report.*

INTRODUCTION

Venoarterial extracorporeal membrane oxygenation (VA-ECMO) is a rapidly deployable modality for hemodynamic support in the setting of cardiogenic shock, although utility in predominantly vasoplegic states remains less well established.[1] Venoarterial extracorporeal membrane oxygenation is a well-described treatment for poisoning, with rates of cannulation consistently increasing in the United States.[2] While cardiogenic shock from poisoning is a relatively common indication, the effectiveness of VA-ECMO in vasoplegia from overdose is poorly defined.[1,2] Multiple antipsychotics and antidepressants have been shown to cause peripheral vasodilation via alpha-1 adrenergic antagonism, which may lead to hypotension.[3] Vasopressors and crystalloid are generally effective, while mechanical circulatory support is rarely required.[3,4]

We present a case using VA-ECMO in the setting of refractory vasoplegia from quetiapine, olanzapine, trazodone, mirtazapine and, more novelly, levomilnacipran, a serotonin–norepinephrine reuptake inhibitor (SNRI) for which overdose data remain limited.

CPC-EM Capsule

What do we already know about this clinical entity?
Atypical antidepressants and antipsychotics may cause hypotension via alpha-1 adrenergic blockade. Effects are usually mild and respond to fluids and vasopressors.

What makes this presentation of disease reportable?
Levomilnacipran has limited data in overdose. Negative inotropy and chronotropy have been reported in animal models, but effects in humans remain poorly described.

What is the major learning point?
Levomilnacipran may blunt the compensatory cardiac output in polysubstance-induced distributive shock. VA-ECMO may be a viable therapy for refractory vasoplegia.

How might this improve emergency medicine practice?
Recognition that levomilnacipran toxicity may cause bradycardia with or without a normal ejection fraction, and require significant hemodynamic support in overdose.

CASE REPORT

An otherwise healthy 71-year-old woman with major depressive disorder and hypertension presented to the emergency department (ED) via paramedics after being found unresponsive by her husband. Empty bottles of quetiapine, olanzapine, trazodone, mirtazapine, levomilnacipran, eszopiclone, and clonazepam were nearby. Amlodipine-valsartan, propranolol and venlafaxine were also noted on her medication list. Initial prehospital vitals were as follows: blood pressure, 89/55 mm Hg; and heart rate, 72 beats per minute. She required bag-valve-mask ventilation due to severe bradypnea and hypoxemia with a peripheral capillary oxygen saturation of 80%. The patient was intubated and received a total of 400 mcg of push-dose epinephrine for hypotension. Vitals on ED arrival were as follows: blood pressure, 71/46 mm Hg; heart rate, 66 beats per minute; temperature, 33.9. ºC; and mechanically ventilated at 14 breaths per minute. A venous blood gas returned with a pH of 7.33 (reference range, 7.31-7.41)' partial pressure of carbon dioxide, 44 mm Hg (41-51 mm Hg); and lactate, 1.0 mmol/L (0.7-2.1 mmol/L), later peaking at 7.3 mmol/L.

Electrocardiogram showed a normal sinus rhythm with a PR, QRS and QTc of 154, 96 and 572 milliseconds, respectively. Her extremities were warm with normal capillary refill, and point-of-care echocardiography revealed hyperdynamic function without tachycardia. Over the following 8 hours, vasopressor requirements escalated to include norepinephrine (0.7 mcg/kg/min), vasopressin (0.08 u/min), and later epinephrine (0.3 mcg/kg/min), and angiotensin II (80 ng/kg/min). With ongoing uptitration of vasoactive agents, a 2 mg/kg bolus of methylene blue followed by a 0.5 mg/kg/hr infusion was also provided with minimal improvement. Due to a national shortage, hydroxocobalamin was not available.[5] High-dose insulin was ultimately deferred given her hyperdynamic cardiac function. Hydromorphone and midazolam infusions were administered for sedation. The medical intensive care unit and regional poison center were contacted to discuss the potential utility of extracorporeal support. Given concerns for further hemodynamic deterioration with ongoing escalation of vasopressor needs for refractory distributive shock, the patient subsequently underwent cannulation for VA-ECMO (Table).[6]

There was expected color change of the circuit, with bright red blood on the return limb and dark blood on the drainage limb. Within minutes, blood in both limbs changed to a dark color. Membrane lung function and cannula positioning were reassessed and appropriate. Colors in the circuit ultimately returned to normal following discontinuation of methylene blue. Transthoracic echocardiogram revealed an ejection fraction of 70% with hyperdynamic function. Hemodynamics continued to improve, and vasopressor needs decreased to norepinephrine and vasopressin by the end of hospital day 1. She was decannulated on hospital day 3, extubated on day 5, and transferred to inpatient psychiatry on day 10 neurologically intact. Urine mass-spectrometry revealed large peaks for

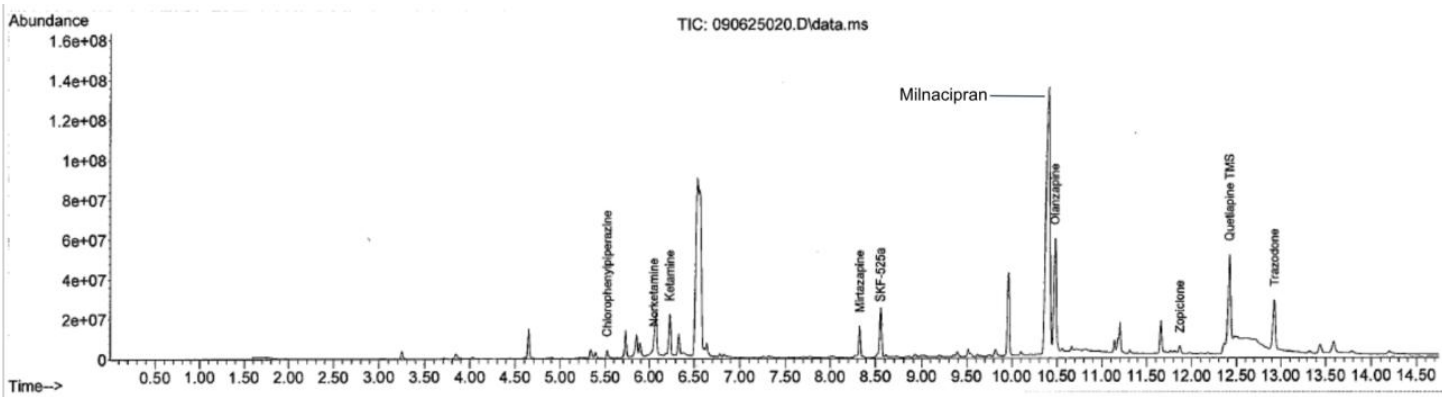


Image. Chromatogram demonstrating a peak consistent with a low concentration of propranolol. Amlodipine and venlafaxine were not detected. (The study was unable to distinguish dextro and levo isomers of milnacipran, zopiclone); neither could specific serum concentrations be obtained.)

quetiapine, olanzapine and milnacipran, additionally noting the presence of ketamine (iatrogenic), mirtazapine, trazodone and zopiclone (Image).

DISCUSSION

As VA-ECMO becomes increasingly available, applications in drug-induced circulatory collapse may continue to expand.[1,2] Utility in vasoplegia remains unclear, with some data suggesting poor survival in comparison to cardiogenic shock.[1] The decision to cannulate for VA-ECMO in these cases remains nuanced. The medications ingested, clinical course, supportive therapies, and patient comorbidities should all be taken into consideration. Consultation with a medical toxicologist, regional poison center, and intensivist or institutional shock team should be used. In more austere community settings, signs of early hemodynamic deterioration signaled by escalating vasopressor needs and clinical evidence of malperfusion should prompt consideration for transfer to a facility with advanced supportive therapies.

Several antipsychotics and atypical antidepressants may cause hypotension via blockade of peripheral alpha-1 adrenergic receptors, although single-substance overdoses are generally well tolerated and rarely require vasopressor support.[3,4] Trazodone and mirtazapine are considered to be generally benign, potentially causing orthostasis, although trazodone has been associated with bradycardia and atrioventricular conduction delay.[7,8] Quetiapine and olanzapine overdoses may cause clinically significant hypotension, the former more so than the latter.[3,9] One case of isolated quetiapine poisoning required VA-ECMO, although this was driven by abnormal myocardial function and ventricular asynchrony.[10] Racemic milnacipran and isomeric levomilnacipran toxicity remains poorly described. A single-substance milnacipran ingestion caused autonomic instability requiring an intermittent norepinephrine infusion, suspected serotonin syndrome, and stress-induced cardiomyopathy.[11] In our patient, the area under the curve for mass spectrometry of quetiapine, olanzapine, trazodone, mirtazapine and milnacipran suggests possibly supratherapeutic concentrations. Notably, the largest peak corresponds to milnacipran, suggesting a significant serum concentration, although this was unable to be specifically quantified.

Our instruments are unable to distinguish between the dextro and levo isomers of milnacipran, hence the reported detection of milnacipran, not specifically the levomilnacipran the patient was prescribed. This same principle applies to the detected zopiclone in the setting of eszopiclone ingestion. While isolated alpha-blockade from any single agent may be effectively managed with vasopressors alone, given refractory hypotension, hyperdynamic myocardial function, and an exam suggestive of vasoplegia, synergism between multiple agents was possible. Trazodone may have contributed to the concurrent bradycardia. Bradycardia is less commonly documented in isolated olanzapine or quetiapine ingestions, as both peripheral alpha-1 blockade and antimuscarinic effects

Table. Venoarterial extracorporeal membrane oxygenation parameters.

Patient Factors	
Height (cm)	160
Weight (kg)	73.9
Extracorporeal Membrane Oxygenation Configuration	
Configuration	Single-lumen cannulae
Cannulation site	Right femorofemoral
Drainage tip position	Inferior cavoatrial junction
Cannula dimensions	V25/55 - A19/15
RPM / Flow	3000 / 3.75 L/min
Sweep gas	1 L/min

Cannula dimensions reflect outer diameter in French sizing and cannula length in centimeters.
A, arterial; *RPM*, rotations per minute; *V*, venous.

generally potentiate tachycardia. The specific effects of levomilnacipran remain unclear. The patient exhibited no clear signs of autonomic instability or serotonergic excess, although this may have been blunted by the administered sedative infusions to a certain degree. A previous report describes initial bradycardia and hypotension, similar to the case of our patient, although with a depressed ejection fraction (30-35%) and subsequent hypertension and tachycardia, which did not develop in our case.[11]

A canine model suggests large doses (10 mg/kg) may precipitate negative inotropic, chronotropic, and dromotropic effects, which may have prevented a reflexive tachycardia in the setting of distributive shock.[12] While similar agents such as venlafaxine are known to cause cardiogenic shock, given evidence of hypercontractile myocardial function and warm peripheral extremities, our patient's presentation was clinically consistent with primarily vasoplegia.[13] Cases with a normal or depressed left ventricular ejection fraction, when combined with signs of peripheral vasodilation, are more suggestive of concomitant cardiogenic shock, particularly when a poison may be blunting compensatory tachycardia. While both trazodone and levomilnacipran may have contributed to concurrent bradycardia, the significantly larger peak of the latter on mass spectrometry suggests levomilnacipran may have played a more predominant role, although this remains inferential in the absence of serum concentrations.

The atypical color change noted in both limbs during cannulation was likely precipitated by the administered methylene blue. Its action as a dye with light-absorption along the orange-red spectrum may directly darken the color of blood.[14] Methylene blue's oxidant effects are also known to precipitate methemoglobinemia, although more commonly at doses approaching 7 mg/kg.[15] While such color discrepancies should otherwise be interpreted as a potential sign of oxygenator failure, similar phenomena have previously been documented following methylene blue administration during VA-ECMO use.[14] The monoamine oxidase inhibition exhibited by methylene blue may also warrant concern for precipitation of serotonergic excess or serotonin syndrome with concomitant selective serotonin reuptake inhibitor or serotonin and norepinephrine reuptake inhibitor use.[16] Signs of excessive serotonergic activity such as worsening tachycardia, encephalopathy or agitation, and lower extremity hyperreflexia and clonus should be monitored, although in the critically ill patient, the more deleterious effects of musculoskeletal rigidity and subsequent hyperthermia may be blunted by sedatives, such as the midazolam infusion in our patient. No signs of serotonergic excess were evident on exam.

CONCLUSION

While clinical experience in overdose is limited, levomilnacipran may precipitate or exacerbate hypotension and bradycardia without a depressed ejection fraction, potentiating malperfusion and shock, particularly when taken in conjunction with other vasoactive medications. Although VA-ECMO is not classically considered as a standard treatment for drug-induced distributive shock in the absence of myocardial dysfunction, it may be considered as a therapeutic option in cases refractory to vasopressor support or other salvage therapies,. Elucidation of definitive benefit remains an area requiring ongoing study.

The authors attest that their institution does not require Institutional Review Board approval for publication of case reports. Written and verbal consent were obtained for publication from the patient. Documentation on file.

Address for Correspondence: William J Bleifuss, MD, Hennepin County Medical Center, Minnesota Regional Poison Center, 701 Park Ave RL.240, Minneapolis, MN 55415. Email: william.bleifuss@hcmed.org.

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Locked-In Syndrome from Delayed Vertebral Artery Dissection After Strangulation: A Case Report

Jennifer R. Snippen, PhD* *Lane Community College, Science Department, Eugene, Oregon
Kien Huynh, MD† †Good Samaritan Regional Medical Center, Department of Internal Medicine, Corvallis, Oregon

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Introduction: Vertebral artery dissection is a rare but life-threatening complication of strangulation. Symptoms are often nonspecific and may not manifest immediately, complicating diagnosis. If prior neck trauma is not elicited during assessment, clinicians may miss a mechanistic link to prompt vascular imaging, leaving patients at risk for irreversible outcomes, as illustrated in this case.

Case Report: A 24-year-old woman presented to a critical access emergency department (ED) with nonspecific neurological symptoms that had largely resolved upon arrival. Her only reported history was vague head and neck pain, which she attributed to stress after leaving an abusive relationship several weeks earlier. Hours later, while still in the ED, the patient experienced abrupt neurological deterioration. She was transferred to a regional medical center, where magnetic resonance imaging eventually revealed vertebral artery dissection with basilar artery hypoperfusion and extensive pontine infarction. Collateral history obtained from family revealed a preceding strangulation event. Police at the scene had documented neck injury, but the patient declined their offer of a medical assessment. By the time vertebral artery dissection was diagnosed, she had developed irreversible neurological damage leading to locked-in syndrome. She survived in this state for years before succumbing to complications of quadriplegia. The death was certified as a homicide due to inflicted blunt force neck injury.

Conclusion: This case underscores the importance of timely, skilled medical assessment after strangulation and highlights the need for targeted screening of patients with vague neurological symptoms or disclosure of intimate partner violence. [Clin Pract Cases Emerg Med. 2026;10(4):547–550.]

Keywords: *vertebral artery dissection; locked-in syndrome; strangulation; medicolegal; case report.*

INTRODUCTION

Early recognition of vertebral artery dissection offers the greatest opportunity to minimize ischemic injury and salvage the viable penumbra.[1] However, diagnosis can be challenging when symptoms are subtle, delayed, or mistaken for more benign conditions.[2] Patients may not readily associate precipitating events with their symptoms and consequently may not disclose the events to clinicians.[3] This case illustrates how failure to elicit disclosure of strangulation and obtain prompt vascular imaging may allow progression from subtle prodromal symptoms to irreversible, catastrophic injury. It highlights the importance of direct screening for strangulation and maintaining a high index of suspicion for vascular injury in patients presenting with vague or evolving neurologic symptoms.

CASE REPORT

A 24-year-old woman with no significant past medical history, presented to a critical access emergency department (ED) by ambulance. To paramedics, she reported waking from

a nap with headache, two bouts of emesis, and transient difficulty moving her left side. She also mentioned transient slurred speech and nonspecific visual difficulties.

Upon arrival in the ED, the original symptoms had largely resolved. The patient denied recent trauma and clinicians explicitly excluded motor vehicle collision, blunt head injury, or fall. She also denied difficulty breathing, swallowing, confusion or any problems with hearing or balance. The patient's sole recent complaint was a history of persistent left-sided neck and shoulder pain, associated with a headache that seemed to worsen when she turned her head. She attributed these symptoms to the stress of leaving an abusive relationship weeks prior and expressed frustration, stating, "How many times do I have to tell this story; it's so hard to talk." Ice, massage, and over-the-counter medications had been used without much relief.

The patient was afebrile with a blood pressure of 133/88 mm Hg. All other vital signs were within normal limits. Physical examination revealed only tenderness in the suboccipital triangle and left levator scapula. Neurological exam was entirely normal with intact cranial nerves, full strength, normal tone, and symmetric deep tendon reflexes. Plantar stimulation elicited a normal flexion response. The neck was supple with no bony tenderness. Routine bloodwork was unremarkable.

The patient was sent for plain cervical films and non-contrast head computed tomography (CT), both of which were negative. Upon return from imaging, she abruptly developed seizure-like activity with decorticate posturing in the left arm and left gaze deviation with nystagmus. She became fully noncommunicative but responded to painful stimuli and periodically seemed to follow commands. Pupillary and gag reflexes remained intact. The patient was stabilized and transferred to a higher level of care for further imaging and neurology consultation. At the receiving facility, clinicians initially focused on ruling out new-onset seizures, viral meningitis, or meningoencephalitis. Meanwhile, her neurological status continued to deteriorate, requiring intubation.

Approximately nine hours after initial emergency medical services (EMS) contact, magnetic resonance imaging was performed, followed by magnetic resonance angiography. Together, these studies demonstrated an acute-to-subacute infarction involving most of the pons, with extension into the medulla and cerebellum, in the setting of a vertebrobasilar dissection and reduced flow in the left vertebral and basilar arteries.

Although the patient remained unresponsive, her family provided a critical detail: She had been strangled several weeks earlier. This disclosure triggered coordination among clinicians and law enforcement. Details emerged of an event, 32 days prior, in which the patient called EMS after being strangled. She reported being forced against a wall by a former partner as he compressed her anterior neck using some combination of his hands and an aluminum bat pressed horizontally across her throat. She experienced neck pain but denied loss of consciousness. Responding officers documented erythema on the anterior neck and offered to summon paramedics; the patient declined, choosing instead to leave and be with family.

The patient's family confirmed that she had been complaining of persistent neck and head pain since the event. They noted that her symptoms had progressively worsened, and she had begun to appear pale and fatigued. Medical care was not sought until the abrupt development of new neurological symptoms. By the time the strangulation history was elicited, the patient had already suffered irreversible brainstem infarction. She was ultimately discharged with locked-in syndrome—cognitively intact but ventilator-dependent and quadriplegic, able to communicate only through vertical eye movements and blinking. With time, she regained limited movement of her lips and was able to mouth words with some effectiveness. The patient survived for several years in this condition.

At the time of her death, the case was referred for medicolegal death investigation. A medical examiner certified

CPC-EM Capsule

What do we already know about this clinical entity?
Nonspecific or delayed symptoms of vertebral artery dissection (VAD) may complicate diagnosis.

What makes this presentation of disease reportable?
First reported case of VAD progressing to locked-in syndrome in an adult strangulation victim.

What is the major learning point?
Disclosure of interpersonal violence should prompt strangulation screening, and nonspecific neurologic symptoms warrant a high index of suspicion for VAD.

How might this improve emergency medicine practice?
Expanded awareness of potential strangulation sequelae may improve medical evaluation and refusal counseling.

the cause of death as urosepsis resulting from complications of an indwelling urinary catheter, as a consequence of quadriplegia due to vertebral artery dissection and pontine infarction, consequent to inflicted blunt force neck injury. The manner of death was classified as homicide.

DISCUSSION

This case illustrates a convergence of multiple levels of failure in the recognition of vertebral artery dissection following strangulation. The patient's initial refusal of medical care, combined with the nonspecific and evolving nature of her symptoms, and delayed disclosure of the strangulation mechanism obscured the underlying vascular injury. Collectively, these factors delayed diagnosis, allowing progression of ischemic injury and culminating in locked-in syndrome. Strangulation events can produce a wide spectrum of physiological effects that may be localized or systemic, transient or delayed, and span a continuum from seemingly insignificant to immediately fatal.[4,5] Among the most serious sequelae is arterial intimal disruption, with carotid artery injury recognized as a cause of acute or delayed infarction and strangulation-related mortality.[5-7]

Vertebral artery involvement has been underrepresented in the strangulation literature, possibly in part because of perceived skeletal shielding afforded by the cervical vertebrae.[5] Forceful neck rotation, flexion, or hyperextension may damage vertebral arteries as they ascend through the transverse foramina or and enter the skull near their basilar confluence.[8] Such injuries have historically been associated with chiropractic manipulation, sports trauma, martial arts, and, more recently, percussive massage devices.[1,3,9] Our review of the literature identified only one prior case of vertebral artery dissection following strangulation, reported in an adolescent victim.[10] To our knowledge, this is the first reported case of vertebral artery dissection in an adult victim of strangulation and the first to progress to locked-in syndrome.

Nonspecific symptoms associated with VAD may precede infarction by days to weeks. These may include headache, neck pain, nausea, visual symptoms, paresthesias, cerebellar signs (eg ataxia, nystagmus), and lateral medullary signs (eg, vertigo, dysphagia, dysarthria).[2,3] Delayed or evolving symptoms may complicate diagnosis, particularly when a precipitating event is not disclosed.[1,3] Clinicians should neither assume that patients will disclose intimate partner strangulation nor expect them to attribute delayed-onset symptoms to a strangulation event without prompting.[11,12] We also note that this patient's evolving neurologic symptoms may have impaired her ability to effectively communicate with clinicians.

For detection of cervical vascular injury, computed tomography angiography (CTA) and magnetic resonance angiography are endorsed—with CTA favored as the initial modality in strangulation cases.[5,13] As this case demonstrates, these critical studies may not be considered when strangulation is not disclosed or when clinicians fail to consider the possibility of underlying vascular injury.

Stabilization of acute strangulation should prioritize airway and cerebral perfusion, followed by evaluation for possible cervical vascular injury.[7,14] Patients who are asymptomatic may be discharged with detailed return precautions; however; soft-tissue injury or neurologic symptoms may warrant observation and advanced screening.[14] If vertebral artery dissection is diagnosed, the prognosis may be favorable.[1,3,9,15] Although treatment approaches lack definitive consensus, current guidelines endorse antiplatelet or anticoagulant therapy, with endovascular or surgical intervention available as increasingly invasive options.[1,10,15]

These findings underscore the need for careful strangulation assessment in the context of reported interpersonal violence, coupled with a high index of suspicion for vertebral artery dissection in patients with nonspecific neurological symptoms. Disclosure of strangulation improves with direct screening, yet research indicates that frontline clinicians lack adequate training and require strangulation-specific protocols to improve recognition and response.[11,12] Strangulation survivors who decline medical assessment should receive refusal counseling that includes an explicit warning about the possibility of delayed stroke. Law enforcement officials, who are often the first point of contact, should receive mandated, standardized training on the full scope of strangulation risks.[11] The circumstances presented in this case indicate that family and community education may also contribute to early recognition and timely referral.

Finally, this case highlights the value of interdisciplinary collaboration among family members, clinicians, law enforcement, and medicolegal death investigators. Although delayed, the eventual coordination enabled accurate mechanism recognition, informed criminal justice proceedings, and ensured appropriate death certification. Standardized protocols and shared training across disciplines may improve patient outcomes, medicolegal accuracy, survivor safety, and strangulation surveillance.

This case report is limited by the inherent constraints of a retrospective single-case observation, including the completeness and availability of medical record data. Because of the time interval between the patient's initial evaluation and submission of this case report, the original digital imaging archives were no longer available. Therefore, all imaging-based conclusions are derived from the written reports.

CONCLUSION

This case illustrates several challenges in the recognition and management of strangulation-related vertebral artery dissection. The findings support routine strangulation screening in patients disclosing interpersonal violence, recognizing strangulation as a high-risk mechanism, and maintaining vigilance for cervical artery injury in patients presenting with

nonspecific neurological symptoms.

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The authors attest that their institution requires neither Institutional Review Board approval nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Jennifer R. Snippen, PhD, Lane Community College, Science Department, 4000 E 30th Ave, Eugene, OR 97405. Email: snippenj@lanecc.edu.

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Subacute Microperforation of Right Atrial Pacemaker Lead Presenting with Shock due to Cardiac Tamponade: A Case Report

Alexander Zirulnik MD, MPH
Briton Bennett, MD
Siham Almousa, DO

Wake Forest University School of Medicine, Department of Emergency Medicine, Winston-Salem, North Carolina

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Introduction: Delayed cardiac tamponade is a rare but life-threatening complication following permanent pacemaker placement and may present with non-specific symptoms.

Case Report: We report the case of a 66-year-old woman on oral anticoagulation who presented with generalized weakness, nausea, and profound hypotension four days after dual-chamber pacemaker placement. Initial evaluation demonstrated atrial fibrillation with rapid ventricular response, a normal chest radiographic findings, prerenal azotemia, and no evidence of device malfunction on interrogation. Point-of-care ultrasound (POCUS) performed in the emergency department revealed a large pericardial effusion with right atrial and right ventricular collapse and a plethoric inferior vena cava, consistent with cardiac tamponade. Emergent pericardiocentesis yielded 400 mL of sanguinous pericardial fluid with rapid hemodynamic improvement. Subsequent imaging revealed a 2-mm protrusion of the right atrial lead into the myocardium, which was suspected to be the source of the effusion, but pacemaker revision was not necessary.

Conclusion: This case highlights the limitations of routine diagnostic testing following permanent pacemaker placement and emphasizes the importance of early POCUS in patients with atypical presentations, particularly in the setting of anticoagulation. [Clin Pract Cases Emerg Med. 2026;10(4):551–553.]

Keywords: *cardiac tamponade; pacemaker lead perforation; point-of-care ultrasound; pericardial effusion; obstructive shock.*

INTRODUCTION

Permanent pacemaker placement is a common procedure with generally low complication rates. However, serious complications such as cardiac perforation and pericardial effusion leading to cardiac tamponade can occur, either immediately or in a delayed fashion. Delayed cardiac tamponade can be particularly challenging to diagnose due to nonspecific symptoms and potentially reassuring initial diagnostic studies, including normal chest radiography and device interrogation.

Emergency physicians must maintain a high index of suspicion for postprocedural complications, especially in anticoagulated patients. Point-of-care ultrasound (POCUS) has emerged as a critical tool in the rapid identification of pericardial effusion and tamponade physiology in unstable patients.

CASE REPORT

A 66-year-old woman with a history of nonobstructive coronary artery disease, Hodgkin lymphoma, breast cancer status postbilateral mastectomy, heart failure with recovered ejection fraction (50%), and persistent atrial fibrillation/flutter on rivaroxaban presented to the emergency department (ED). with generalized weakness and hypotension.

Three days prior to presentation, she underwent dual-chamber permanent pacemaker placement and was discharged

in stable condition. She reported feeling well immediately following discharge until earlier in the day of presentation when she developed progressive generalized weakness and fatigue, accompanied by nausea and one episode of vomiting. She noted home blood pressure readings as low as 60/30 mm Hg, but denied chest pain, dyspnea, syncope, or lower extremity swelling.

On arrival, she appeared toxic and in extremis. Her triage vital signs were notable for hypotension (61/44 mm Hg) and a heart rate of 136 beats per minute. Physical exam was notable for irregular tachycardia, clear lung sounds, pale skin, diaphoresis, healing procedural sites without evidence of infection, and no clear signs of volume overload. Given her hypotension and tachycardia, she received a 1 L bolus of crystalloid resuscitation but remained persistently hypotensive. Subsequently, POCUS demonstrated newly reduced ejection fraction (estimated between 30% and 50%; normal ejection fraction ≥ 50%), a large pericardial effusion with right atrial systolic collapse, right ventricular diastolic collapse, and a plethoric inferior vena cava with minimal respiratory variation, consistent with cardiac tamponade (Images 1 and 2). Cardiology was consulted.

Laboratory studies revealed the following: lactate, 2.7 mmol/L (reference range, 0.5–2.2 mmol/L); white blood cell count, 11.5 × 10^3/μL (4.4–11.0 × 10^3/μL); and creatinine, 1.22 mg/dL (0.70–1.30 mg/dL), increased from 0.70 mg/dL four days prior. Electrocardiogram demonstrated atrial fibrillation with rapid ventricular response. Chest radiograph showed no acute cardiopulmonary abnormalities. Device interrogation in the ED demonstrated no evidence of dysfunction. The patient's vitals marginally improved after fluid support, and after cardiology consultation, she was admitted to the cardiovascular intensive care unit (ICU) to undergo urgent pericardiocentesis.

Pericardiocentesis was performed with removal of 400 mL of sanguinous pericardial fluid and hemodynamics improved significantly. A pericardial drain was placed, and she returned to the cardiovascular ICU.

CPC-EM Capsule

What do we already know about this clinical entity?
Delayed cardiac tamponade after pacemaker placement is rare, may present atypically, and can be difficult to diagnose with routine testing.

What makes this presentation of disease reportable?
Cardiac tamponade developed days after pacemaker placement despite normal chest radiography and device interrogation; rapid point-of-care ultrasound (POCUS) established the diagnosis.

What is the major learning point?
Early POCUS is essential in unstable patients after recent pacemaker implantation to identify cardiac tamponade.

How might this improve emergency medicine practice?
Routine early POCUS in hypotensive patients after pacemaker implantation can expedite diagnosis of tamponade and lifesaving intervention.

She developed pleuritic chest pain immediately following the procedure and was started on nonsteroidal anti-inflammatory drug treatment for pericarditis. Repeat transthoracic echo on postoperative day 1 demonstrated a reduced ejection fraction of 30% without recurrence of pericardial effusion.

Computed tomography of the chest on postoperative day



Image 1. Point-of-care ultrasound using a subxiphoid cardiac view demonstrates a large pericardial effusion (left arrow) with right ventricular diastolic collapse (right arrow). These findings were consistent with cardiac tamponade physiology.

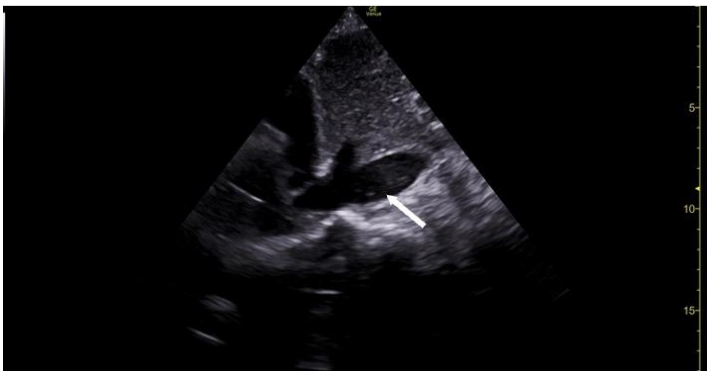


Image 2. Point-of-care ultrasound of the inferior vena cava (IVC) (white arrow) demonstrates a plethoric, dilated IVC with minimal respiratory variation, consistent with elevated right atrial pressure in the setting of cardiac tamponade.

3 revealed approximately 2 mm protrusion of the right atrial lead into the myocardium, and in the setting of the patient's use of a direct-acting oral anticoagulant, this was considered to be the most likely culprit for her pericardial effusion. The electrophysiology consultation determined that lead revision was not indicated. Maintaining the current position may have tamponaded the perforation, and revision could have carried a higher risk of complication. Furthermore, serial device interrogation demonstrated no device malfunction.

Anticoagulation was resumed after stabilization without reaccumulation of the effusion. In total, approximately 1.2 L of fluid was drained before removal on day 4 of her admission. The patient was ultimately discharged in stable condition.

DISCUSSION

Delayed cardiac tamponade following permanent pacemaker placement is an uncommon but well-described complication; however, most are recognized early. Cardiac tamponade causing hemodynamic instability secondary to acute perforation beyond 72 hours accounts for a small minority of presentations.[1]The fact that the patient was on a direct-acting oral anticoagulant likely contributed to both the timing of her presentation and the severity of her tamponade, and patients on direct oral anticoagulants may be at increased risk of hemorrhagic pericardial effusion in the setting of microperforation.[2] In the largest contemporary series, anticoagulation status was an independent predictor of tamponade, and anticoagulated patients had approximately a 22-fold increased risk of developing tamponade when perforation occurred.[3]

The patient's clinical presentation was also atypical. Rather than classic features such as chest pain, which is the most common presenting symptom, she presented with generalized weakness, nausea, and profound hypotension.[3] Such nonspecific symptoms may delay recognition, particularly in patients with competing diagnoses such as arrhythmia or hypovolemia.[4]

This case highlights the limitations of commonly used diagnostic tools. Chest radiography may not detect lead perforation or pericardial effusion, and normal device interrogation does not exclude mechanical complications, such as microperforation.[5] In this patient, both studies were nondiagnostic despite the presence of life-threatening tamponade physiology. However, POCUS played a pivotal role in diagnosis and allowed for rapid identification of tamponade physiology and expedited definitive management. Prior studies have demonstrated that POCUS significantly improves time to diagnosis in undifferentiated hypotension and is highly sensitive for detecting pericardial effusion.[6] Notably, it was not until several days into her hospitalization when microperforation was uncovered.

We believe this reinforces the importance of early POCUS in unstable patients and highlights the ability of emergency physicians to quickly and definitively expose life-threats even when traditional testing may be nonconclusive, nondiagnostic, or superseded by the need to stabilize a critically ill patient.

CONCLUSION

Delayed cardiac tamponade following pacemaker placement can present with nonspecific symptoms and misleadingly normal initial diagnostic studies. Clinicians should maintain a high index of suspicion in recently instrumented and anticoagulated patients presenting with hypotension. Point-of-care ultrasound is a critical tool for early diagnosis and may be life-saving in these scenarios.

The authors attest that their institution requires neither Institutional Review Board approval nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Alexander Zirulnik MD, MPH, Wake Forest University School of Medicine, Department of Emergency Medicine, Meads Hall, 2nd Floor, Medical Center Boulevard, Winston-Salem, NC 27157. Email: Alexander.Zirulnik@wfusm.edu.

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Acute Dystonia Following Ifosfamide Infusion in a Young Woman with Breast Angiosarcoma: A Case Report

William S. Miller, MD, MBA
Alyssa B. Niblett, MD
Charles A. Khoury, MD, MSHA

University of Alabama at Birmingham Heersink School of Medicine, Department of Emergency Medicine, Birmingham, Alabama

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Introduction: Ifosfamide is an alkylating chemotherapeutic agent commonly used in the treatment of sarcomas and other malignancies. Neurotoxicity is a recognized adverse effect, most often presenting as encephalopathy. Acute movement disorders such as dystonia are rare and may be underrecognized in the emergency department.

Case Report: We report a 22-year-old woman receiving combination chemotherapy with doxorubicin, ifosfamide, and mesna for breast angiosarcoma who developed acute right-sided neck spasms and involuntary movements within five minutes of initiating ifosfamide infusion. Vital signs were as follows: temperature, 98.0 degrees Fahrenheit; heart rate, 97 beats per minute; respiratory rate, 18 breaths per minute; blood pressure, 130/88 millimeters of mercury; and oxygen saturation, 99% on room air. Examination revealed sustained contraction of the right sternocleidomastoid and trapezius muscles with involuntary head deviation. She had no exposure to medications commonly associated with dystonia. She was treated with intravenous thiamine, benzodiazepines, intravenous fluids, and baclofen, with subsequent improvement.

Conclusion: Acute dystonia represents a rare manifestation of ifosfamide-induced neurotoxicity. Emergency clinicians should consider chemotherapy-related neurotoxicity in patients presenting with acute movement disorders, as early recognition and supportive management may prevent progression. [Clin Pract Cases Emerg Med. 2026;10(4):554–556.]

Keywords: *ifosfamide; neurotoxicity; acute dystonia; chemotherapy; emergency medicine.*

INTRODUCTION

Ifosfamide is an alkylating agent used in chemotherapy for a variety of malignancies, including sarcomas. It is metabolized by hepatic cytochrome P450 enzymes into active metabolites that exert cytotoxic effects through DNA alkylation. Neurotoxicity is a recognized adverse effect, reported in approximately 5%–30% of patients, and most commonly presents as encephalopathy with confusion, hallucinations, or altered mental status.[1] Movement disorders, including dystonia and myoclonus, are rare manifestations of ifosfamide-induced neurotoxicity and may be unfamiliar to emergency clinicians. With ifosfamide continuing to be used in complex oncologic regimens, emergency physicians may encounter atypical neurotoxic presentations. We describe a case of acute dystonia following ifosfamide infusion to highlight this uncommon but clinically significant adverse effect.

CASE REPORTS

A 22-year-old woman with right breast angiosarcoma was receiving her first cycle of chemotherapy in our institution's cancer treatment infusion center. Approximately five minutes after initiation of the ifosfamide infusion, she developed acute headache and involuntary right-sided neck movements and was referred directly to the emergency department for further evaluation. In the emergency department, she reported associated neck pain and limited range of motion. She denied



Image. Acute cervical dystonia demonstrating sustained involuntary contraction of the right sternocleidomastoid and trapezius muscles with resultant rightward head deviation following ifosfamide infusion.

CPC-EM Capsule

What do we already know about this clinical entity?
Ifosfamide is associated with neurotoxicity, most commonly presenting as encephalopathy.

What makes this presentation of disease reportable?
Acute dystonia is a rare and underrecognized manifestation of ifosfamide-induced neurotoxicity.

What is the major learning point?
Acute dystonia can represent ifosfamide-induced neurotoxicity and may occur shortly after infusion.

How might this improve emergency medicine practice?
Early recognition and supportive treatment, including consideration of thiamine, may prevent progression of neurotoxicity.

similar prior symptoms, recent illness, trauma, or new medications. This represented the patient's first cycle of ifosfamide-containing chemotherapy, and she had no prior exposure to ifosfamide.

Vital signs were as follows: temperature, 98.0 ºF; heart rate, 97 beats per minute; respiratory rate, 18 breaths per minute; blood pressure, 130/88 mm Hg; and oxygen saturation, 99% on room air. No premedications were administered prior to chemotherapy infusion. The patient had not received dopamine antagonists or other medications commonly associated with acute dystonic reactions, including metoclopramide, prochlorperazine, promethazine, haloperidol, or ondansetron. Examination revealed sustained contraction of the right sternocleidomastoid and trapezius muscles with involuntary head deviation to the right (Image). No focal neurologic deficits were present.

The differential diagnosis included acute drug-induced dystonia, seizure, cervical muscle spasm, and acute cerebrovascular event. The close temporal relationship to ifosfamide infusion and isolated focal dystonia supported a medication-induced etiology. The patient was treated with intravenous thiamine and benzodiazepines, with moderate improvement. She was admitted for further evaluation, including electroencephalography and brain magnetic resonance imaging. Her presentation was attributed to ifosfamide-induced acute dystonia.

DISCUSSION

Ifosfamide-induced neurotoxicity includes a spectrum of neurologic complications ranging from mild confusion to severe encephalopathy, hallucinations, seizures, and movement disorders. Encephalopathy is the most frequently reported manifestation; movement abnormalities such as dystonia are rare. Toxic metabolites, including chloroacetaldehyde, are thought to contribute through mitochondrial dysfunction and glutathione depletion.[2-4]

A formal adverse drug reaction assessment using the Naranjo Adverse Drug Reaction Probability Scale yielded a score of 6, consistent with a probable adverse drug reaction. The rapid onset of symptoms following initiation of ifosfamide infusion, absence of alternative etiologies, objective examination findings, and subsequent clinical improvement support a causal relationship. For emergency clinicians, recognition of this entity is important to avoid unnecessary diagnostic testing and to facilitate early targeted management.

Thiamine has been reported as a therapeutic adjunct in ifosfamide-induced neurotoxicity and may improve neurologic symptoms.[2,5] Although evidence is limited, thiamine is low risk and may be considered in suspected cases. Management centers on discontinuation or dose adjustment of ifosfamide, supportive care, and symptomatic treatment with benzodiazepines or antispasmodics. Early recognition may prevent progression to more severe neurologic complications.

CONCLUSION

Acute dystonia is a rare manifestation of ifosfamide-induced neurotoxicity. Emergency clinicians should maintain a high index of suspicion for chemotherapy-related neurotoxicity in patients presenting with acute movement disorders, as early recognition and supportive management may improve outcomes.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Charles A. Khoury, MD MSHA, University of Alabama at Birmingham Heersink School of Medicine, Department of Emergency Medicine, 204 General Services Building, 521 19th St S, Birmingham, AL 35233. Email: ckhoury@uabmc.edu.

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Type I Choledochal Cyst in a Three-Year-Old with Recurrent Vomiting and Pancreatitis: A Case Report

Dominic DiPrinzio, DO* *University of Nevada, School of Medicine, Renown Regional Medical Center, Department of Emergency Medicine, Reno, Nevada
Rance Del Mar, BS† †University of Nevada, School of Medicine, Reno, Nevada

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Introduction: Choledochal cysts are rare congenital dilatations of the biliary tree that may present with nonspecific gastrointestinal symptoms in children, posing diagnostic challenges.

Case Report: We report a 3-year-old girl who presented with recurrent vomiting and abdominal pain. Initial laboratory testing revealed a mildly elevated lipase. An abdominal ultrasound demonstrated a pancreatic cyst; the patient was discharged with outpatient follow-up. She returned with worsening symptoms and a markedly elevated lipase of 2,247 U/L (reference range, < 160 U/L). Computed tomography subsequently revealed a type I choledochal cyst. She underwent operative resection with an uncomplicated postoperative course.

Conclusion: Persistent gastrointestinal symptoms with uptrending lipase in children should prompt consideration of hepatobiliary etiologies. Choledochal cysts may be missed on initial presentation as the classic triad of jaundice, palpable abdominal mass, and right upper quadrant pain is present in a minority of cases. [Clin Pract Cases Emerg Med. 2026;10(4):557–560.]

Keywords: *choledochal cyst; pancreatitis; pediatrics; abdominal pain; case report.*

INTRODUCTION

Choledochal cysts are rare congenital anomalies of the biliary tract with variable presentations, accounting for approximately one in 100,000 to 150,000 live births in Western populations.[1] Clinical symptoms are frequently nonspecific in children, contributing to delayed diagnosis and potential complications such as recurrent pancreatitis, cholangitis, hepatic fibrosis, and malignancy.[2,3] We present a case of a 3-year-old girl whose evolving symptoms led to the eventual identification of a type I choledochal cyst requiring definitive surgical intervention.

CASE REPORT

A previously healthy 3-year-old girl presented to a community emergency department (ED) with two episodes of nonbloody, nonbilious vomiting that were preceded by brief episodes of abdominal pain, which started after she struck her abdomen on a bar of a play structure in a park. The pain resolved after vomiting. There were no associated fevers. Vital signs were as follows: blood pressure, 104/57 mm Hg; pulse, 91 beats per minute; temperature, 36.1 °C; respiratory rate, 26 breaths per minute; and weight, 18.8 kg. The physical examination revealed no abdominal discomfort on deep palpation, and no focal tenderness or palpable masses. Abdominal radiograph revealed a nonobstructive bowel gas pattern. She tolerated oral hydration and was discharged with supportive care instructions.

The patient returned four hours later after another episode of vomiting at home. Abdominal examination was remarkable for right lower quadrant tenderness. Laboratory testing noted a lipase of 182 U/L (reference range, < 160 U/L), and normal complete blood count, C-reactive protein, and comprehensive metabolic panel. Abdominal ultrasound demonstrated a normal appendix and a 2 × 3 cm-anechoic pancreatic cyst; no biliary dilation was documented (Image 1). The patient was given ondansetron, after which she was able to tolerate oral liquids

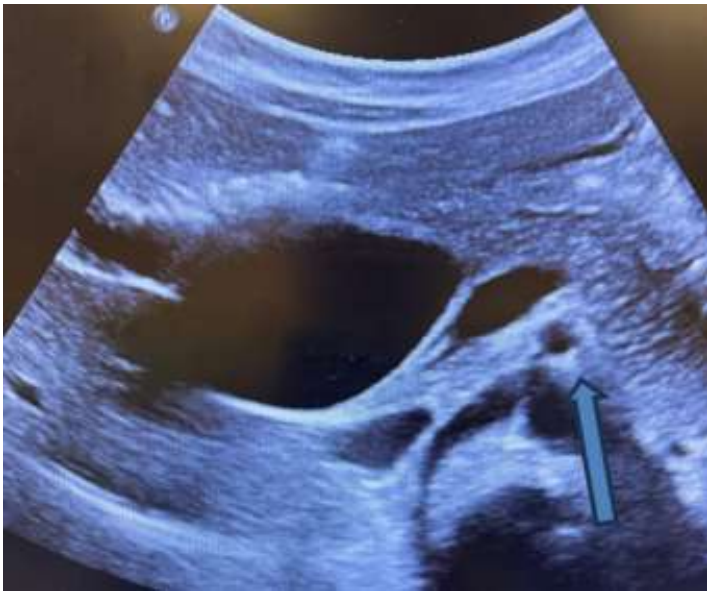


Image 1. 2 × 3 cm-cyst adjacent to gallbladder and pancreas (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Choledochal cysts are rare biliary dilatations causing obstruction, cholangitis, pancreatitis, and malignancy risk.

What makes this presentation of disease reportable?
Symptoms followed minor abdominal trauma with normal initial laboratory studies. Diagnosis was delayed until symptom progression prompted repeat evaluation.

What is the major learning point?
Consider choledochal cysts in recurrent biliary symptoms despite negative initial workup. Repeat assessment and additional imaging may reveal the diagnosis.

How might this improve emergency medicine practice?
Promotes broader differentials, repeat evaluation of persistent symptoms, earlier diagnosis, and timely specialist referral to prevent complications.

and had no further episodes of vomiting or pain. She was discharged with a bland diet and close outpatient follow-up.

Over the next two days, the patient improved clinically while adhering to a bland diet. However, when her diet was advanced, vomiting recurred and was accompanied by right lower quadrant pain. She re-presented to the ED, where examination showed abdominal tenderness in the epigastric and right lower quadrant region. Repeat laboratory evaluation revealed a lipase of 2,247 U/L. Computed tomography (CT) of the abdomen and pelvis demonstrated a cystic structure measuring 6 cm in its greatest dimension, extending from the pancreatic head into the porta hepatis, which was concerning for a choledochal cyst (Image 2).

In addition, there was notable thickening of the terminal ileum with associated prominent right lower quadrant lymphadenopathy and a normal appendix. She was admitted and underwent magnetic resonance cholangiopancreatography. Her pancreatitis was managed, and she was discharged home after three days. Her surgery was scheduled two days after discharge to allow additional time for her lipase to return to normal. She then underwent robotic choledochal cystectomy with a primary hepaticoduodenostomy. Her postoperative recovery was uneventful, and she was discharged home on post operative day 4 with surgical follow-up. At the time of follow-up, she remained asymptomatic with normal diet tolerance.

DISCUSSION

Choledochal cysts are classified into five primary types using the Todani system.[4] Type I choledochal cysts are a cystic or fusiform dilation of the extrahepatic common biliary duct.[4] They are also the most common type, accounting for 50-80% of all cases.[4] Other classifications include type II, which presents as a saccular diverticulum of the common bile duct, type III as a dilation of the distal portion of the common bile duct into the duodenal wall, and type IV as either intrahepatic and extrahepatic dilation of the biliary ducts or multiple sites of dilation in only the extrahepatic biliary tree.[4] Type V choledochal cysts, known as Caroli disease, are characterized by multiple dilations of the intrahepatic bile ducts.[4]

Clinical manifestations vary by age, often contributing to a delay in diagnosis.[2,4,5] Prenatal choledochal cysts can be identified through the presence of infrahepatic lesions, while infants (up to 12 months of age) present with jaundice, hepatomegaly, and acholic stools.[2,3] Children and adolescents (up to 18 years) account for nearly 80% of choledochal cyst cases and typically experience generic symptoms such as vomiting and abdominal pain or show evidence of pancreatitis.[2,5,6] In a recent study, all three symptoms of the classic triad (right upper quadrant abdominal pain, jaundice, and a palpable abdominal mass) were observed in only 4% of pediatric patients.[6] Thus, it is possible for patients to initially be discharged with nonspecific abdominal pain. In these cases, a return visit to the ED often serves as a critical diagnostic inflection point, signaling an evolving pathology and prompting a significant increase in imaging, laboratory tests, and procedures.[7]



Image 2. Coronal computed tomography of the abdomen and pelvis demonstrating cystic structure (white arrow) extending from the pancreatic head into the porta hepatis, concerning for a type I choledochal cyst.

It is hypothesized that abnormalities at the pancreaticobiliary duct junction result in reflux of pancreatic enzymes that weakens the biliary walls, contributing to cyst formation and pancreatitis.[1] Elevations in young children's pancreatic enzymes reflect recurrent chronic inflammation and are associated with precancerous changes within the biliary tract.[8] Given the severity of these underlying pathologies, prompt diagnostic evaluation and early surgical intervention are essential.[3] Ultrasound is a highly sensitive modality for the initial diagnosis of choledochal cysts (71-97%) and can be used to identify them prenatally.[4] However, ultrasound is limited in its ability to detect subtle biliary dilation or definitively map biliary anatomy to identify Todani subtypes.[4,5] Imaging such as CT with contrast or magnetic resonance cholangiopancreatography offer improved sensitivities and a more conclusive diagnosis.[4,5]

Surgical excision with biliary enteric reconstruction is the standard of care due to risks of recurrent pancreatitis, cholangitis, hepatic fibrosis, and malignancy if left untreated, which have been documented even during childhood.[2,9] Minimally invasive techniques, including laparoscopic and robot-assisted approaches, continue to expand and demonstrate favorable outcomes in pediatric patients.[10]

Although choledochal cysts are well documented in the surgery literature, a review of prior emergency medicine case reports demonstrates that they remain a rare and difficult diagnosis in the ED. Initial imaging can mistakenly identify cysts as more common acute biliary pathology such as a distended gallbladder with shadowing stones, requiring additional scanning.[11] Furthermore, rare complications such as spontaneous rupture can complicate the presentation and are difficult to diagnose preoperatively.[12]

This case emphasizes the importance of reconsidering differential diagnoses when symptoms evolve, particularly in pediatric patients with recurrent vomiting and rising pancreatic enzymes. Emergency clinicians should maintain a high index of suspicion for hepatobiliary pathology when initial imaging is nondiagnostic and clinical symptoms progress.

CONCLUSION

Choledochal cysts may present subtly in children and can be initially missed when symptoms mimic common gastrointestinal illnesses. Worsening abdominal symptoms and elevated lipase levels should prompt further imaging and consideration of hepatobiliary disease. Early recognition and surgical management are essential to prevent complications.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Dominic DiPrinzio, DO, University of Nevada Renown Medical Center, Department of Emergency Department, 2620 Antonio Ln, Reno, NV 89523. Email:diprinz2@gmail.com.

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From Staple Removal to Startle—The Unexpected Discovery of a Scalp Pseudoaneurysm: A Case Report

Brendon Stankard, PA-C
John Forrester, DO
Stephanie Jose, DO
Allison Cohen, MD
Mathew Nelson, DO

North Shore University Hospital, Department of Emergency Medicine, Manhasset, New York

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Introduction: Point-of-care ultrasound (POCUS) allows emergency physicians to rapidly diagnose vascular abnormalities, including pseudoaneurysms. Although commonly identified following vascular catheterization, traumatic pseudoaneurysms of superficial scalp arteries are rare.

Case Report: An 85-year-old woman receiving anticoagulation presented to the emergency department (ED) for removal of a scalp staple placed during treatment of a previously bleeding scalp wound. Physical examination revealed a persistent scalp hematoma. Point-of-care ultrasound demonstrated a hypoechoic collection with Doppler flow consistent with a pseudoaneurysm. Computed tomography angiography confirmed the diagnosis. Ultrasound-guided compression attempted in the ED resulted only in transient thrombosis. The patient subsequently underwent elective percutaneous embolization.

Conclusion: Emergency physician–performed POCUS can rapidly identify traumatic scalp pseudoaneurysms and facilitate timely consultation and treatment. [Clin Pract Cases Emerg Med. 2026;10(4):561–563.]

Keywords: *pseudoaneurysm; point-of-care ultrasound; ultrasound; scalp trauma; doppler.*

INTRODUCTION

Point-of-care ultrasound (POCUS) has become an important diagnostic tool in emergency medicine for the rapid identification of vascular abnormalities. Pseudoaneurysms most commonly occur following vascular catheterization, particularly involving the femoral artery, and are frequently diagnosed using ultrasound in the emergency department.[3] Traumatic pseudoaneurysms of superficial scalp arteries are uncommon but represent an important consideration in patients presenting with persistent swelling following head trauma. We report a case of a superficial scalp pseudoaneurysm discovered incidentally during staple removal and diagnosed with POCUS in the emergency department (ED).

CASE REPORT

An 85-year-old woman with a past medical history of hypertension, hyperlipidemia, type 2 diabetes mellitus, and atrial fibrillation treated with apixaban presented to the ED for removal of a scalp staple placed one week earlier. Approximately one month prior, the patient had fallen down three stairs and sustained a head injury. She was initially evaluated in the ED and found to have an abrasion over the right superior occiput without active bleeding and a small underlying scalp hematoma on noncontrast computed tomography (CT). She was discharged home without intervention.

Two weeks later, she returned to the ED after developing bleeding from the area of the hematoma. Repeat noncontrast CT again demonstrated a small scalp hematoma. One staple was placed to control bleeding, and the patient was discharged.

At the most recent visit for staple removal, the patient's vital signs were notable for the following: blood pressure, 179/68 mm

Hg; heart rate, 60 beats per minute; respiratory rate, 20 breaths per minute; temperature, 97.8 °F orally; and oxygen saturation, 98% on room air. The wound appeared completely closed without active bleeding. However, a persistent scalp hematoma was noted on examination. The hematoma was around 3 cm, dome-shaped, and indurated. There was no active bleeding from it at that time. There was no thrill, and the area was slightly tender to palpation. Given this finding and the persistence of a hematoma that would have been expected to be resolving by that point, POCUS was performed to assess for underlying vascular abnormality, including possible pseudoaneurysm formation.

Initial ultrasound demonstrated a hypoechoic fluid collection measuring 2.9 × 1.5 × 2.9 cm (Image 1). Color Doppler imaging demonstrated internal blood flow consistent with a 1.4 × 0.7 × 1.0 cm pseudoaneurysm (Image 2). Computed tomography angiography of the head and neck confirmed a faint blush suggestive of pseudoaneurysm formation (Image 3).

Neurosurgery consult recommended ultrasound-guided manual compression in the ED. Compression produced transient thrombosis but did not achieve durable occlusion. The patient was discharged with follow-up and subsequently underwent elective direct percutaneous embolization of the occipital pseudoaneurysm.

CPC-EM Capsule

What do we already know about this clinical entity?

Scalp pseudoaneurysms are rare delayed complications of blunt trauma and can mimic benign scalp hematomas or soft tissue swelling.

What makes this presentation of disease reportable?

The pseudoaneurysm was discovered incidentally during routine staple removal, with point-of-care ultrasound (POCUS) expediting diagnosis before potentially harmful treatment.

What is the major learning point?

POCUS should be considered for persistent or pulsatile scalp masses after trauma to rapidly identify pseudoaneurysm and avoid inappropriate procedures.

How might this improve emergency medicine practice?

Routine use of POCUS for atypical post-traumatic scalp lesions can prevent misdiagnosis, guide management, and expedite definitive treatment.

DISCUSSION

Pseudoaneurysms develop when disruption of an arterial wall allows blood to escape into surrounding tissues while maintaining communication with the parent vessel. Persistent arterial pressure leads to formation of a fibrous sac containing circulating blood that communicates with the arterial lumen.[1]

Pseudoaneurysms may result from blunt or penetrating trauma, inflammatory processes, infection, or iatrogenic arterial injury during medical procedures.[1] Vascular catheterization is a common cause, with reported incidence rates approaching 7.7%.[1] Although rare, persistent posttraumatic scalp hematoma may indicate underlying pseudoaneurysm formation, with trauma representing the most common cause.[6]

Scalp pseudoaneurysms should be considered in patients presenting with persistent or enlarging posttraumatic scalp hematomas that are not resolving as expected.[6,7] Although pulsatility, bruit, and thrills may be present on physical exam, they are not always noted.[6] In such cases, POCUS provides a rapid, safe, and easily accessible modality for identifying

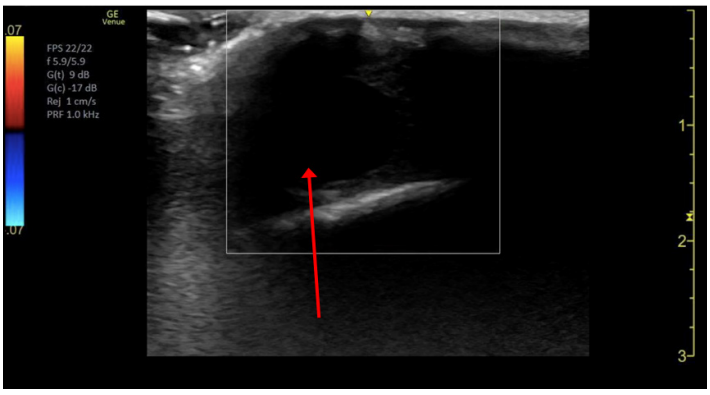


Image 1. B-mode ultrasound demonstrating a hypoechoic fluid collection within the scalp measuring 2.9 × 1.5 × 2.9 centimeter.

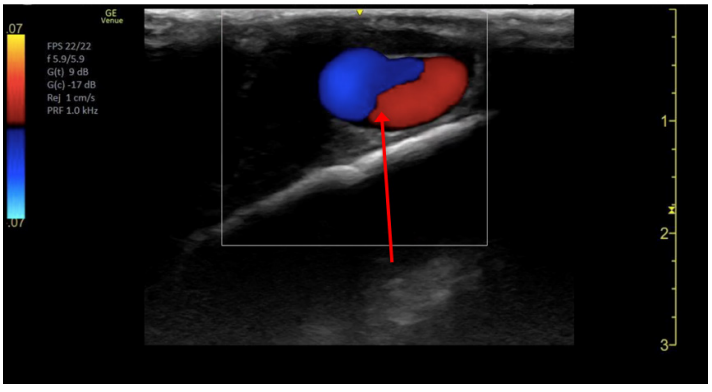


Image 2. Color Doppler ultrasound demonstrating internal swirling blood flow within the lesion consistent with pseudoaneurysm ("yin–yang sign").



Image 3. Computed tomography angiogram of the head and neck demonstrating a hematoma with a faint blush measuring 1.4 × 0.7 × 1 centimeter.

underlying vascular lesions.[6] Point-of-care ultrasound provides a rapid and accessible method for diagnosing pseudoaneurysms in the ED.[6] On B-mode ultrasound, pseudoaneurysms appear as an anechoic or hypoechoic sac adjacent to the affected artery.[1]

Color Doppler imaging typically demonstrates the classic "yin–yang" sign, reflecting swirling bidirectional blood flow within the pseudoaneurysm cavity.[2] The diagnostic hallmark of pseudoaneurysm is identification of a communicating neck between the pseudoaneurysm sac and the feeding artery with a "to-and-fro" Doppler waveform, representing bidirectional blood flow entering and exiting the sac during the cardiac cycle.[1,2]

Ultrasound-guided compression can be used as an initial treatment approach for pseudoaneurysms. Direct pressure is applied with the ultrasound transducer to occlude flow at the pseudoaneurysm neck while preserving distal arterial perfusion. Sustained compression for approximately 10 minutes may promote thrombosis.[3] However, the success of compression therapy may be limited by patient discomfort, operator fatigue, lesion size or depth, and anticoagulation therapy.[1] In such cases, alternative treatments such as thrombin injection or endovascular embolization may be necessary.

CONCLUSION

Persistent hematomas following trauma should prompt consideration of pseudoaneurysm formation. Emergency physician–performed POCUS can rapidly identify superficial vascular injuries and guide timely consultation and treatment.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Brendon Stankard, PA-C, Department of Emergency Medicine, North Shore University Hospital, 300 Community Drive Manhasset NY 11030. Email: bstankard@northwell.edu.

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Idiopathic Left Anterior Fascicular Ventricular Tachycardia in the Emergency Department: A Case Report

Rochelle Kofman, BS* *Mayo Clinic Alix School of Medicine, Scottsdale, Arizona
Connor O'Hare, MD*† †Mayo Clinic Hospital Phoenix, Department of Emergency Medicine, Phoenix, Arizona
Lauren B. Querin, MD, MEd*†

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Introduction: Idiopathic fascicular ventricular tachycardia is a rare form of ventricular tachycardia that is commonly seen in young, otherwise healthy individuals without structural heart disease. This rhythm can be difficult to distinguish from other forms of tachyarrhythmia, including supraventricular tachycardia, yet acute pharmacologic management of this condition varies significantly from more common forms.

Case Report: We describe a 42-year-old woman with no cardiac history who presented with palpitations and lightheadedness. She was found to be in a hemodynamically stable, wide-complex tachycardia. Given no prior cardiac history, supraventricular tachycardia with aberrancy was initially considered as a leading diagnosis; however, a trial of adenosine and lidocaine was ineffective. Her electrocardiogram revealed a regular wide complex tachycardia at 262 beats per minute with a right bundle branch block morphology and right axis deviation, consistent with a rare left anterior fascicular subtype of ventricular tachycardia. She later underwent successful ablation of the left anterior fascicle with no recurrence of symptoms at follow-up.

Conclusion: This report highlights a case of idiopathic fascicular ventricular tachycardia successfully treated with intravenous verapamil and left anterior fascicle ablation, addressing the diagnostic challenges of this arrhythmia. Early recognition by emergency physicians is essential to avoid treatment delays and ensure appropriate definitive management. [Clin Pract Cases Emerg Med. 2026;10(4):564–567.]

Keywords: *idiopathic fascicular ventricular tachycardia; tachyarrhythmia; verapamil; case report.*

INTRODUCTION

Ventricular tachycardia (VT) is a high-risk cardiac arrhythmia that most commonly occurs in the setting of structural heart disease and myocardial scar. However, approximately 10% of patients are young and otherwise healthy with no known cardiac disease.[1-3] These cases are referred to as idiopathic fascicular VT and often stem from re-entrant pathways within the His-Purkinje system.[3] The large majority of these pathways originate from the left posterior fascicle, while the left anterior fascicular subtype represents only 5-10% of idiopathic fascicular VT cases.[4,5] While idiopathic fascicular VT generally has a better prognosis than other forms of VT, it is often difficult to recognize on initial presentation. This diagnostic challenge in the emergency department (ED) arises because its clinical and electrocardiographic features often mimic other arrhythmias such as supraventricular tachycardia (SVT).[6,7] In addition to its diagnostic challenge, idiopathic fascicular VT characteristically responds to verapamil, an agent not commonly used and potentially even contraindicated in other forms of arrhythmia[8]. It is vital that emergency physicians recognize and differentiate idiopathic fascicular VT from other arrhythmias.

CASE REPORT

A 42-year-old woman presented to the ED with palpitations, tachycardia, lightheadedness, and nausea. She had no known past medical history and no personal or family history of heart disease or sudden cardiac death. She reported moderate alcohol intake the night prior, followed by multiple episodes of nonbloody emesis overnight. In the morning, she had another episode of emesis and began experiencing palpitations and lightheadedness. She denied syncope or chest pain. She stated that she has never had symptoms like this in the past and denied illicit drug use.

Upon arrival to the ED, she was tachycardic to 240 beats per minute (bpm) with a blood pressure of 144/123 mm Hg, while other vitals were within normal limits. She appeared fatigued and mildly anxious. Physical examination was notable for tachycardia and dry mucous membranes; she was alert, conversant, and nontoxic in appearance. Laboratory results ordered initially during the patient's evaluation showed mild leukocytosis and mild transaminitis, while electrolytes, thyroid studies, pregnancy testing, and chest radiography were unremarkable.

The initial electrocardiogram (ECG) (Image) demonstrated a regular wide complex tachycardia at 262 bpm with a right bundle branch block-type morphology and right axis deviation. There were no clear capture beats or fusion complexes on the 12-lead ECG. Initial differential diagnoses were SVT with aberrancy versus VT.

CPC-EM Capsule

What do we already know about this clinical entity?
Idiopathic fascicular ventricular tachycardia is a rare subtype that can resemble supraventricular tachycardia.

What makes this presentation of disease reportable?
This highlights a rare subtype, idiopathic anterior fascicular ventricular tachycardia, treated with intravenous verapamil and left anterior fascicle ablation.

What is the major learning point?
This arrhythmia may mimic more common tachyarrhythmias, but early recognition is essential to avoid treatment delays and ensure definitive management.

How might this improve emergency medicine practice?
Prompt identification by clinicians can prevent delays in care, adverse outcomes from inappropriate management, and facilitate definitive management.

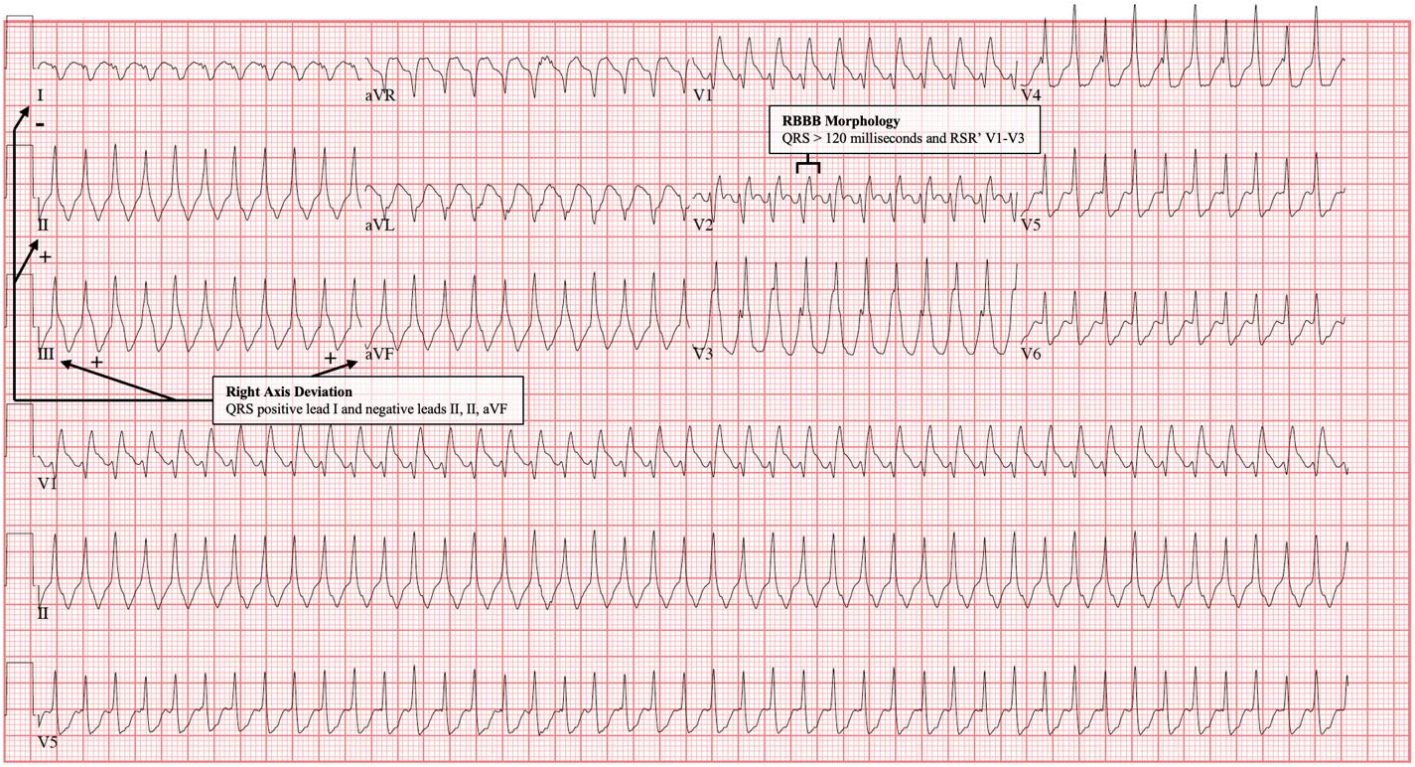


Image. Initial electrocardiogram demonstrating a regular, wide complex tachycardia with a right bundle branch block morphology and a right axis deviation.

A trial of intravenous adenosine (6 mg) had no effect on the rhythm. Subsequent administration of intravenous lidocaine (50 mg bolus followed by infusion) resulted in partial rate reduction to the low 220 bpm but was discontinued due to mild hypotension and poor efficacy. Cardiology consult recommended a trial of verapamil (2.5 mg) intravenously, which resulted in conversion to sinus rhythm.

The patient was admitted to cardiology, and electrophysiology was consulted for mapping and ablation. An echocardiogram showed no structural heart disease with normal biventricular function. The patient later underwent empiric ablation of the left anterior fascicle with noninducible VT following the procedure. A follow-up ECG one month later was normal, and the patient had no recurrence of symptoms.

DISCUSSION

Idiopathic fascicular VT represents approximately 10% of all presenting cases of VT and is due to a re-entrant circuit with ectopic focus most commonly within the left ventricle.[3] This arrhythmia typically affects young, otherwise healthy individuals between the ages of 15-40 years, with a male predominance of 60-80%.[9] It was first described in 1979 by Zipes et al and in 1981, Belhassen et al identified it as verapamil-sensitive VT.[1,10] Like other forms of VT, idiopathic fascicular VT presents as a monomorphic wide complex tachycardia with fusion complexes, atrioventricular (AV) dissociation, and capture beats. The QRS complex duration, however, is usually only modestly widened (100-140 milliseconds) compared with other forms of VT.[11,12] Other characteristic ECG findings include a right bundle branch block morphology and axis deviation which is dependent on the anatomical site of the re-entry circuit.[11] The anatomic location of the majority of re-entry circuits is at the left posterior fascicle, which produces a left axis deviation on ECG. A right axis deviation, as in this patient's case, is seen more rarely (5-10% of cases) when the re-entrant circuit is in the left anterior fascicle, making it a particularly rare clinical entity.[11] Recognition of this variant is important, as its atypical axis may contribute to further uncertainty in the diagnosis of an already rare arrhythmia.

Clinical presentation is variable and may include dizziness, palpitations, chest pain, shortness of breath, or syncope. Given occurrence is primarily in young, healthy individuals, hemodynamic stability is more common than in other forms of VT.3 These clinical features, along with the rhythm's relatively organized appearance, contribute to the diagnostic challenge of the arrhythmia because it mimics other tachyarrhythmias such as SVT with aberrancy.[11,12] It is imperative that emergency physicians be aware of idiopathic fascicular VT and consider it in their differential of tachyarrhythmia since the acute pharmacologic management of this condition varies from other more common forms. The diagnosis can be made by carefully observing specific features of VT, including fusion complexes, capture beats, and AV dissociation, as well as characteristic right bundle branch block morphology and axis deviation.[11]

As seen in this case, idiopathic fascicular VT is often refractory to commonly used pharmacological agents for tachyarrhythmia. It may be unresponsive to adenosine, beta-blockers, and lidocaine; however, it does classically respond to intravenous verapamil, a nondihydropyridine (DHP) calcium channel blocker.[13,14] Although not entirely understood, verapamil is thought to be effective as the re-entrant circuit is calcium channel dependent. Some studies have also demonstrated efficacy of other non-DHP calcium channel blockers such as diltiazem.14 It should also be noted that calcium channel blockers are not typically recommended in other forms of VT.15 However, like other forms of wide complex tachycardia, an unstable patient should be treated with synchronized cardioversion.[10] In cases of diagnostic uncertainty, synchronized cardioversion should always remain a key consideration. Definitive management involves timely coordination with electrophysiology consultation for evaluation of catheter ablation to terminate the aberrant pathway.[10]

A key clinical consideration is the potential for initial misdiagnosis as SVT with aberrancy. As in such cases, adenosine is often administered as first-line therapy, including in this patient's case.[11,12] While generally safe, adenosine is frequently ineffective in fascicular VT and may delay appropriate treatment. Physicians should maintain a high index of suspicion for idiopathic fascicular VT in patients initially presumed to have SVT who do not respond to adenosine. Early recognition is important because verapamil may be both diagnostic and therapeutic in this arrhythmia.

This case highlights a rare presentation of idiopathic fascicular VT arising from the left anterior fascicle, an uncommon subtype that poses an even greater diagnostic challenge. Although most patients presenting with monomorphic VT have underlying heart disease, idiopathic fascicular VT is often seen in young, healthy individuals without structural heart disease. As demonstrated in this case, diagnosis can be challenging because the rhythm can mimic other forms of tachyarrhythmia, including SVT with aberrancy. Despite the diagnostic challenge, early identification of this rhythm is imperative because effective pharmacologic treatment varies from other forms of tachyarrhythmia, including other types of VT.[11,12] Definitive management with targeted cardiac ablation techniques has been highly successful in preventing recurrence and achieving favorable long-term outcomes in idiopathic fascicular VT.[11]

CONCLUSION

Idiopathic fascicular ventricular tachycardia often presents in young, otherwise healthy patients and can closely mimic more common rhythms such as SVT with aberrancy, increasing the risk of misdiagnosis. Unlike typical

management of wide complex tachycardias, this arrhythmia is frequently unresponsive to adenosine and instead responds to verapamil, making early recognition critical for appropriate treatment. Prompt identification can prevent delays in care and facilitate timely cardiology consultation or referral for definitive management with catheter ablation.

The Institutional Review Board approval has been documented and filed for publication of this case report.

Address for Correspondence: Lauren B. Querin, MD MEd, Mayo Clinic Hospital Phoenix, Department of Emergency Medicine, 5777 E. Mayo Blvd., Phoenix, AZ 85054. Email: querin.lauren@mayo.edu.

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A Case Report of Meningitis due to *Borrelia* Species in Arizona

Scott Austin, DO
Tamlyn Tarzia, MD
Griffith Saunders, DO

Valleywise Health Medical Center, Emergency and Trauma Services, Phoenix, Arizona

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Introduction: Lyme disease is most prevalent in the Northeast and Midwest regions of the United States, as well as northern California; however, cases have been reported in nonendemic areas, including the Southwestern United States. Atypical presentations or underreporting can cause delay in diagnosis.

Case Report: We present the case of a 22-year-old previously healthy man, who presented to our emergency department in metropolitan Phoenix with five days of headache, fever, meningismus, and photophobia, and later developed focal neurologic deficits consistent with Lyme meningitis. Cerebrospinal fluid was notable for lymphocytic pleocytosis, and Western blot for *Borrelia burgdorferi* Immunoglobulin-M antibodies was positive, which led us to Lyme meningitis as the unifying diagnosis. He was treated with intravenous ceftriaxone and his symptoms resolved.

Conclusion: This case highlights the importance of considering this pathology in nonendemic areas. [Clin Pract Cases Emerg Med. 2026;10(4):568–570.]

Keywords: *infectious disease; neurology; critical care.*

INTRODUCTION

Lyme disease is caused by *Borrelia burgdorferi* and is the most prevalent vector-borne illness in the United States. The highest incidence is in the Northeast, Midwest, and northern Pacific Coast. However, Lyme disease. has been increasingly detected in areas that have traditionally been considered low risk, including the Southwest, as well as states neighboring high-endemic areas such as Ohio, Indiana, Iowa, and North Carolina.[5]

Lyme meningitis is a rare but serious manifestation of *Borrelia* infection. It typically manifests with headache and photophobia, although cranial neuritis and radiculoneuritis are also seen. It has been well-documented in the Northeast and Midwest but infrequently in other regions. The Wilderness Medical Society confirms that while *Borrelia* infections are rare in the Southwest, increasing travel patterns, climate change, and the spread of the vector species, may be contributing to an expanded geographic range of this pathology.[1] Additionally, other *Borrelia* species such as *Borrelia mayonii* have been implicated in neurologic pathologies across the US, further suggesting that the prevalence of *Borrelia* infections outside endemic areas may be underreported.[3] Continued surveillance is crucial to understanding the epidemiology of Lyme disease and its neurological sequelae in nontraditional areas.

CASE REPORT

A 22-year-old man presented to two separate emergency departments (ED) in the urban metropolitan–Phoenix area with complaints of headache, photophobia, and fever. He was a professional boxer and had sustained a strike to the left parietal-occipital cranium on the day prior to index presentation. Noncontrast computed tomography of the head was negative for acute pathology or evidence of trauma. The patient was thought to have postconcussive headache and was discharged without further workup on both occasions. On the fifth day of symptoms and four days after the initial ED visit, the patient presented to our ED. He complained of persistent symptoms associated with meningismus and vomiting.

His initial vital signs were notable only for a temperature

of 38.8 °C. On examination, he had decreased passive flexion of the neck secondary to pain but was without focal neurologic deficits. There was no evidence of dermatologic abnormalities or tick bites. Complete blood count revealed leukocytosis of 11.3 cells/mL (reference range, 3.4 cells/mL–11.0 cells/mL) with a neutrophilic predominance. Complete metabolic panel was notable for sodium of 126 mmol/L (137 mmol/L–145 mmol/L) and lactic acid of 2.9 mmol/L (0.7mmol/L-1.9 mmol/L). As there was clinical concern for meningitis, the patient underwent lumbar puncture. Initial cerebrospinal fluid analysis revealed increased protein and a pleocytosis with lymphocytic predominance.

The patient was admitted to the medicine floor and initiated on empiric antimicrobials for bacterial meningitis, which included ceftriaxone and vancomycin. Because *Varicella Zoster* and *Listeria* were deemed unlikely, coverage for those were not included. Further cerebrospinal fluid analysis was negative for bacterial and fungal cultures, herpes simplex virus types 1 and 2*,* parechovirus *and* enterovirus, West Nile virus antibodies, and *Coccidioides* antigen and antibodies. Antimicrobials were discontinued, but the patient continued to sustain fevers. On hospital day 6, he developed a left-sided facial droop and left-sided extremity weakness. His level of consciousness declined, and he became oriented only to self. Because there was concern for his ability to protect his airway, he was intubated and transferred to the medical intensive care unit.

Contrast-enhanced magnetic resonance imaging of the brain was unremarkable, but imaging of the cervical spine was notable for meningeal enhancement. Additional serologic studies revealed a positive Lyme antibody, and confirmatory *Borrelia Burgdorferi* immunoglobulin-M by Western blot was positive. Repeat lumbar puncture was negative for *Borrelia* species by polymerase chain reaction, although these results were thought to be affected by preceding antibiotics administration. Further history from the patient's family revealed that that he had frequented mountainous areas in northern Arizona within three months prior to his presentation and was known to interact with wildlife. He had no other travel history. The diagnosis was most consistent with Lyme meningitis, and he subsequently received a 14-day course of intravenous ceftriaxone. His mental status improved, and neurologic deficits resolved.

CPC-EM Capsule

What do we already know about this clinical entity?
The literature regarding Borrelia infections, including the development of Lyme meningitis, is derived from endemic areas.

What makes this presentation of disease reportable?
This case highlights the expansion of Lyme disease into nonendemic areas, including the Southwestern United States.

What is the major learning point?
Clinicians should have a high index of clinical suspicion, even in nonendemic areas, as it changes management of these patients.

How might this improve emergency medicine practice?
The species differential diagnosis for bacterial meningitis could impact the serologies that are initially sent with cerebrospinal fluid samples and lead to earlier diagnosis.

DISCUSSION

We describe the case of a young man who presented to the ED with headache and fever and later developed facial nerve palsy with unilateral weakness, thought to be secondary to Lyme meningitis in a nonendemic area. Guidelines from the Infectious Diseases Society of America and the American Academy of Neurology describe Lyme meningitis as a known early manifestation of disseminated *Borrelia burgdorferi* infection within the first months of exposure. Although Lyme disease is most prevalent in the Northeast, Midwest, and northern California, the Wilderness Medical Society notes that cases have been reported in nonendemic areas, including the southwestern US.

CONCLUSION

Awareness of *Borrelia* infection outside endemic regions is critical to avoid missed or delayed diagnosis, given that the geographic range of vectors continues to expand. Emergency physicians in the Southwest should maintain a high index of suspicion for Lyme meningitis in patients with compatible neurologic complaints, particularly when the patient's travel history is indeterminate. Prompt recognition and initiation of intravenous ceftriaxone (2 g daily for 10–28 days) is associated with favorable outcomes.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Scott Austin, DO, Valleywise Health Piper Pavillion, 2609 East Roosevelt Street, Phoenix, AZ 85008. Email: scottaustin2426@gmail.com.

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Cauda Equina Syndrome Secondary to Seizure-Induced Spinal Fracture During Hyperbaric Oxygen Therapy: A Case Report

Daniel Aloise, MD*
Joel Nash, DO*
Gregory Alfred, MD*
Rahib Iqbal, BA†
Eric Boccio, MD, MPH*‡

*Mount Sinai Medical Center, Department of Emergency Medicine, Miami Beach, Florida
†Nova Southeastern University, Dr. Kiran C. Patel College of Osteopathic Medicine, Davie, Florida
‡New York Medical College-School of Medicine, Department of Emergency Medicine, Valhalla, New York

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Introduction: Hyperbaric oxygen therapy carries a risk of central nervous system oxygen toxicity, which can result in seizures. Seizure-induced spinal fractures are uncommon complications that occur during generalized convulsive activity. While often benign, lumbar vertebral body burst fractures may result in cauda equina syndrome. Additionally, patients with beta-thalassemia may have decreased bone mineral density due to ineffective erythropoiesis and bone marrow expansion increasing the risk for pathologic and traumatic fractures.

Case Report: A 35-year-old man with beta-thalassemia major presented to the emergency department after a witnessed, first-time generalized tonic-clonic seizure during hyperbaric oxygen therapy for chronic venous ulcers. Initial evaluation revealed severe low back pain and midline lumbar tenderness. Computed tomography revealed an acute third lumbar vertebra (L3) burst fracture with retropulsion. The patient rapidly developed progressive right lower-extremity weakness and acute urinary retention. Magnetic resonance imaging revealed 70% loss of height of L3, severe thecal sac stenosis at L2-L3 and L3-L4, and a ventral dural hematoma resulting in stenosis at L4-L5. The patient was diagnosed with cauda equina syndrome and underwent emergent neurosurgical decompression.

Conclusion: This case represents a rare convergence of multiple complications: central nervous system oxygen toxicity during hyperbaric oxygen therapy, seizure-induced lumbar burst fracture with retropulsion, and traumatic cauda equina syndrome in a patient with beta thalassemia. Physicians must maintain a high index of suspicion for vertebral fractures and monitor for evolving neurological deficits when managing the patient with acute back pain following seizure activity. [Clin Pract Cases Emerg Med. 2026;10(4):571–575.]

Keywords: *cauda equina syndrome; seizure-induced spinal fracture; hyperbaric oxygen therapy; beta thalassemia; case report.*

INTRODUCTION

Seizure-induced spinal fractures represent an uncommon but well-documented complication of generalized convulsive activity. The violent muscle contractions generated during tonic-clonic seizures can produce sufficient axial force to cause compression or burst fractures, most commonly affecting the thoracolumbar junction.[1] While most of these are minor compression fractures, severe complications including spinal cord compression or cauda equina syndrome can occur.[1] Hyperbaric oxygen therapy, while an established treatment for chronic wounds, carries a risk of central nervous system (CNS) oxygen toxicity secondary to reactive oxygen species in brain tissue and decreased gamma-aminobutyric acid (GABA) concentrations.[2,3] The risk of fracture is significantly

compounded in patients with beta-thalassemia, who often suffer from multifactorial bone disease. Ineffective erythropoiesis causes bone marrow expansion, leading to cortical thinning and reduced trabecular bone tissue.[4]

This case presents a unique intersection of multiple rare complications involving a patient with beta-thalassemia major who sustained a third lumbar vertebra (L3) burst fracture and subsequent cauda equina syndrome following a seizure while undergoing hyperbaric oxygen therapy. Because clinical features may develop gradually and overlap with other pathologies, prompt recognition and emergent imaging are vital to prevent permanently disabling neurological sequela.

CPC-EM Capsule

What do we already know about this clinical entity?
Hyperbaric oxygen therapy (HBOT) rarely causes oxygen toxicity seizures; however, seizure-induced vertebral fractures may occur.

What makes this presentation of disease reportable?
This rare case of an HBOT-related oxygen toxicity seizure resulted in a third lumbar vertebra burst fracture and cauda equina syndrome in a patient with beta-thalassemia.

What is the major learning point?
New back pain after a seizure warrants evaluation for vertebral fracture and evolving cauda equina syndrome, even without external trauma.

How might this improve emergency medicine practice?
Recognizing seizure-related spinal injuries early can expedite imaging, neurosurgical consultation, and decompression before permanent neurologic deficits develop.

CASE REPORT

A 35-year-old man with a history of chronic venous ulcers, beta-thalassemia major, and ureterolithiasis presented to the emergency department following a witnessed first-time seizure during hyperbaric oxygen therapy. The seizure was generalized tonic-clonic in nature, lasted less than one minute, and resolved spontaneously. There was no history of benzodiazepine or alcohol use, head trauma, known seizure disorder, or previous seizure during hyperbaric oxygen therapy.

On initial evaluation, vital signs were within normal limits. The patient was alert and oriented following the postictal period and only complained of low back pain. Physical examination revealed bilateral tongue lacerations. The head was normocephalic and atraumatic. Extraocular movements were intact, and pupils were equal, round, and reactive to light. The focused cardiac and pulmonary examinations were unremarkable. There was no midline cervical or thoracic spine tenderness. The patient complained of midline lumbar spine tenderness with right paralumbar tenderness. There were no palpable step-offs or muscle spasm. Motor strength was 5/5, sensation was intact in the bilateral lower extremities, and the patient had full range of motion while flexing and extending at the hips and knees bilaterally. There was an approximate 3 × 3-cm venous ulcer over the medial aspect of the right calf of the lower extremity. Given that the seizure occurred during hyperbaric oxygen therapy, oxygen toxicity was considered as a potential etiology. Computed tomography (CT) of the brain without contrast and CT of the abdomen and pelvis without contrast revealed an acute L3 vertebral body burst fracture (Image 1).

Incidentally, well-defined bilateral lobulated soft-tissue masses along the paravertebral regions of the spine consistent with extramedullary hematopoiesis were also observed on the CT abdomen and pelvis series (Image 2).

On reassessment following CT, the patient complained of worsening back pain. Physical examination revealed 4/5 motor strength with right hip flexion and knee extension. The patient was reporting suprapubic abdominal pain, and point-of-care ultrasound revealed a distended bladder, which was concerning for urinary retention. A Foley catheter was placed and emergent magnetic resonance imaging (MRI) of the lumbar spine without contrast was performed. The MRI redemonstrated the L3 vertebral body burst fracture with 70% loss of height and osseous retropulsion resulting in radiographic cauda equina compression (Image 3).

After neurosurgery was consulted, the patient was taken to the operating room for emergent L1-L5 posterior fixation and fusion, L2-L5 laminectomy, and L3 corpectomy with strut reconstruction. On hospital day 14, he developed worsening mid-back pain. Follow-up CT of the thoracic spine demonstrated bilateral L1 pedicle fractures extending into the L1 vertebral body. The patient subsequently returned to the operating room and underwent posterior T-10 pelvis fixation and fusion with cement augmentation of pedicle screws, as well as bilateral sacroiliac fixation and fusion.

The patient was referred to a spinal cord injury rehabilitation unit and discharged to a skilled nursing facility on hospital day 34. At discharge, neurologic examination demonstrated 4/5 strength in right ankle dorsiflexion and plantarflexion, 3/5 strength in right hip and knee flexion, and 2/5 strength in right knee extension. At six-month neurosurgical follow-up, strength had improved to 5/5 in right knee flexion and extension, ankle dorsiflexion, and plantarflexion, with 4/5 strength in right hip flexion. The



Image 1. Computed tomography of the abdomen and pelvis showing an acute comminuted third lumbar burst fracture and severe spinal canal stenosis (arrow).

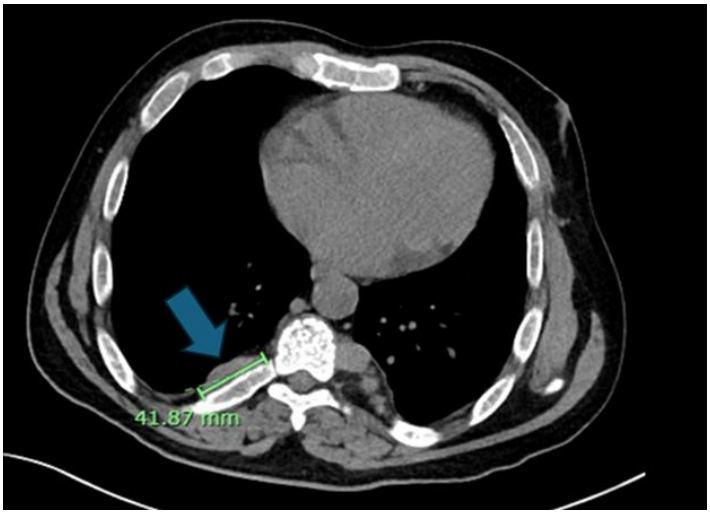


Image 2. Computed tomography of the abdomen and pelvis showing multiple soft-tissue densities in the right parathoracic region, measuring 41.87 millimeters (mm) along its greatest axis (arrow, ruler).

patient denied saddle paresthesia and reported no bowel or bladder dysfunction.

DISCUSSION

Hyperbaric oxygen therapy involves breathing 100% oxygen at pressures exceeding 1 atmosphere absolute (ATA), typically ranging from 1.8 to 3.0 ATA for therapeutic applications.[5] While generally safe when administered according to standard protocols with treatment sessions limited to 120 minutes, hyperbaric oxygen therapy carries the risk of CNS oxygen toxicity, manifesting most dramatically as generalized tonic-clonic seizures.[6] The incidence of oxygen toxicity seizures during hyperbaric oxygen therapy is relatively low. A recent retrospective cohort study of 634 sessions in patients with pre-existing seizure disorders reported only one seizure event.[7] The pathophysiology of CNS oxygen toxicity is multifactorial. Recent evidence demonstrates that elevated partial pressure of oxygen increases the concentration of reactive oxygen species in neural tissues, which irreversibly alters cellular receptors and lowers the activity of glutamic acid decarboxylase, thereby reducing concentrations of the inhibitory neurotransmitter GABA.[6] Additionally, elevated ambient pressure directly inhibits GABA and glycine receptors, contributing to rapid seizure onset.[6]

Central nervous system oxygen toxicity presents initially as a sequence of cardiorespiratory abnormalities including hyperoxic hyperventilation, which ultimately culminates in generalized seizures.[5] The latent period before seizure onset is highly variable between individuals and within the same individual on different occasions, making prediction of toxic indications difficult.[5] When seizures do occur during hyperbaric oxygen therapy, they are typically self-limited and cause no permanent neurological damage when the patient is immediately removed from the hyperbaric environment. However, the violent muscular contractions during these seizures can result in significant injury.

The mechanism of seizure-induced vertebral fractures involves violent forces generated by strong paraspinal muscles during tonic-clonic activity. A systematic review of seizure-related fractures found that thoracic and lumbar vertebral compression fractures accounted for 29% of all seizure-related fractures, second only to bilateral posterior shoulder fracture-dislocations (33%).[8] However, a burst fracture—characterized by disruption of both anterior and posterior vertebral body columns with potential retropulsion of bone fragments into the spinal canal—may be a rare complication. A multicenter video-electroencephalogram study of 626 generalized convulsive seizures found vertebral body compression fractures in seven patients, yielding a risk of 1.1% per seizure (95% CI, 0.5–2.2%) and 1.7% per patient (95% CI, 0.8–3.3%).[9] Importantly, these fractures occurred during the tonic phase of seizures and were accompanied by a characteristic



Image 3. T2-weighted lumbar spine magnetic resonance image showing an acute third lumbar (L3) burst fracture with 70% loss of height, significant retropulsion, and severe thecal sac stenosis spanning L2-L5 secondary to a downstream ventral dural hematoma (arrow).

audible cracking noise, with all affected patients reporting spontaneous back pain and increased pain on palpation of the affected spine.[9]

Several case reports have documented seizure-induced lumbar burst fractures. Youssef et al reported contiguous L1 and L2 burst fractures in a 35-year-old man following a witnessed tonic-clonic seizure while lying on a couch.[10] McCullen and Brown described thoracic burst fractures with bilateral proximal humerus fractures secondary to seizure activity.[11] Napier and Nolan reported a rare case of burst fracture resulting in conus medullaris syndrome following generalized motor seizures, emphasizing that despite the relative frequency of vertebral compression fractures, burst fractures with neurological sequelae are uncommon.[12] Risk factors for seizure-induced fractures include the following: seizure severity and duration; use of antiseizure drugs known to decrease bone density; and family history of fractures.[8] Patients with generalized tonic-clonic seizures have significantly higher injury rates compared to those with other seizure types. The presence of comorbidities, polytherapy drug regimens, and alcohol consumption have also been identified as independent predictors of seizure-related injuries.[13]

Cauda equina syndrome results from compression of the nerve roots in the lumbosacral spinal canal and presents with varying degrees of sensory loss, motor weakness, and bowel and bladder dysfunction. The classic clinical presentation includes urinary retention, saddle anesthesia of the perineum, bilateral lower extremity pain, numbness, and weakness. Decreased rectal tone may be a relatively late finding. The diagnosis of cauda equina syndrome can be challenging, as initial signs and symptoms are often subtle and may not present simultaneously. Cauda equina syndrome may be caused by spinal trauma resulting in injury at or below the level of L1. A study of 214 patients with traumatic cauda equina syndrome found that 52% were able to walk and more than 20% recovered bowel and bladder function by rehabilitation discharge, with median improvement in lower extremity motor score of four points.[14] Shorter time from injury to rehabilitation admission was a significant predictor for improvement in both walking ability and bowel function.[14] A study of 17 consecutive cases of cauda equina syndrome from low lumbar traumatic injuries found that 14 of 17 patients had satisfactory outcomes, with recovery of leg weakness occurring within four months and bladder and bowel function within three months.[15] However, three patients with poor outcomes included two with gunshot wounds, highlighting the importance of injury mechanism.[15] Time to surgery within 24 or 48 hours versus later did not represent a prognostic factor for recovery, and incomplete versus complete cauda equina syndrome did not show better recovery.[15]

Beta-thalassemia is associated with significant bone disease, representing a potential source of morbidity in this population. The pathophysiology is multifactorial, involving ineffective erythropoiesis causing bone marrow expansion with reduced trabecular bone tissue and cortical thinning, excessive iron loading causing endocrine dysfunction with increased bone turnover, and disease complications resulting in physical inactivity with subsequent reduction in optimal bone mineralization. Investigators have documented a high prevalence of osteoporosis in patients with transfusion-dependent beta-thalassemia.

CONCLUSION

This case highlights the potential for severe neurological injury following a seizure induced by hyperbaric oxygen therapy. The patient's convulsive episode, likely secondary to central nervous system oxygen toxicity, resulted in an L3 compression fracture and subsequent cauda equina syndrome. Although seizures induced by hyperbaric oxygen therapy are uncommon, clinicians should remain vigilant for this complication, particularly in patients with predisposing risk factors such as beta-thalassemia. As demonstrated in this case, a stepwise imaging strategy progressing from CT to emergent MRI is essential for identifying spinal cord or cauda equina compression. Fractures associated with neurological deficits require emergent surgical consultation, as timely decompression may remain an important determinant of functional recovery. In patients with underlying conditions such as beta-thalassemia, pretreatment assessment of bone

health and careful monitoring for early signs of oxygen toxicity may help mitigate risk. Emergency physicians should maintain a high index of suspicion for vertebral injury after seizure activity, especially when accompanied by new back pain, focal spinal tenderness or deformity, or new neurologic deficits.

Author Contributions: DA and GA managed the patient's clinical care. DA and JN performed the literature review and drafted the initial manuscript. EB assisted in the diagnostic interpretation and critically revised the manuscript for important intellectual content. EB and GA assisted with the revisions of the final manuscript. All authors have read and approved the final version of the manuscript.

The authors attest that their institution does not require Institutional Review Board approval. Patient consent has been obtained and filed for the publication of this case report. Documentation on file.

Address for Correspondence: Eric Boccio, MD, MPH, Mount Sinai Medical Center, Department of Emergency Medicine, 4300 Alton Road, Miami Beach, FL 33140. Email: bocciomedicalservices@gmail.com.

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A Rare Presentation of Pneumococcal Meningitis with Acute Bilateral Vision Loss: A Case Report

Adam Anderson, MD
Alexander Piszker, DO
Eric Applebach, MD
Reid Mitchell, DO
Aji Yagou Jeng, MD

Trinity Health West Michigan Emergency Medicine Residency, Muskegon, Michigan

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Introduction: Bacterial meningitis affects about 1.2 million people worldwide each year. *Streptococcus pneumoniae* is responsible for the majority of cases of bacterial meningitis in the United States and Europe, with visual impairment seen in 6.3% of patients. The etiology of acute vision loss in the setting of bacterial meningitis remains wide-ranging, most often occurring in the setting of endogenous endophthalmitis, optic neuropathy, and cortical blindness. Retinal necrosis is rare, and typically associated with viral meningitis, but has not been reported in bacterial meningitis.

Case report: A 66-year-old man with headache, malaise, and intermittent blurry vision, initially diagnosed with viral upper respiratory infection and presumed trigeminal neuralgia, presented to the emergency department with altered mental status, meningismus, and progressive bilateral vision loss. He was diagnosed with bacterial meningitis, and subsequent cerebrospinal fluid and blood cultures were positive for *S pneumoniae*. Ophthalmologic evaluation demonstrated bilateral nonreactive pupils, retinal ischemia, optic nerve pallor, and retinal necrosis but no intraocular inflammation. Brain magnetic resonance imaging showed multifocal cortical ischemic infarcts thought to be of embolic source. His hospitalization was complicated by mitral valve regurgitation, endocarditis, and cardiac tamponade requiring extensive surgical repair.

Conclusion: This case represents a rare presentation of bilateral retinal ischemia and necrosis in the setting of *S pneumoniae* meningitis without evidence of endophthalmitis. Awareness of atypical ocular presentations and differential diagnoses is vital in providing early diagnosis, treatment and specialist involvement. [Clin Pract Cases Emerg Med. 2026;10(4):576–579.]

Keywords: *pneumococcal meningitis; bilateral vision loss; acute ischemic optic neuropathy; case report.*

INTRODUCTION

Bacterial meningitis is a life-threatening condition affecting about 1.2 million people worldwide each year.[1] *Streptococcus pneumoniae* is responsible for approximately 66% of all cases of bacterial meningitis in the United States and Europe.[2] Headache is the most common symptom, present in more than 85% of patients; seizures and focal neurologic deficits occur in 25% to 30%.[1] One systematic review and meta-analysis of bacterial meningitis complications found that visual impairment occurred in 6.3% of patients.[3] The differential diagnosis for acute vision loss in the setting of bacterial meningitis is broad, with case reports discussing etiologies ranging from endogenous endophthalmitis to cortical blindness to acute neuritis.[4-6] Acute vision loss due to bacterial meningitis most often occurs in the setting of endogenous endophthalmitis, optic neuropathy, and cortical blindness. Retinal necrosis is a rare

complication, and while it can present as a component of endogenous endophthalmitis it is typically indicative of an underlying viral process. Our case highlights a unique presentation of acute bilateral visual impairment in the setting of *S pneumoniae* bacterial meningitis, with findings of retinal ischemia and necrosis.

CPC-EM Capsule

What do we already know about this clinical entity?
Visual loss is a rare complication of pneumococcal meningitis, usually attributed to endophthalmitis, optic neuropathy, or cortical blindness.

What makes this presentation of disease reportable?
This is the first reported case of bilateral retinal ischemia and necrosis in the setting of pneumococcal meningitis without evidence of endophthalmitis.

What is the major learning point?
Pneumococcal meningitis can cause retinal ischemia and necrosis, even without endophthalmitis.

How might this improve emergency medicine practice?
Recognizing atypical ocular complications of bacterial meningitis can help guide timely diagnosis and treatment.

CASE REPORT

A 66-year-old retired, active man with no pertinent past medical history initially presented to urgent care with five days of upper respiratory symptoms, headache, and intermittent blurry vision. Tests for COVID-19 and influenza were negative. He was diagnosed with viral upper respiratory tract infection, prescribed fluticasone nasal spray, and discharged with instructions for routine primary care follow-up. Four days later, he presented to the emergency department (ED) for headache, jaw pain, and generalized malaise. He was afebrile with normal heart rate and blood pressure. On physical exam, he was well appearing without meningismus or other focal neurologic deficits. Computed tomography of the head was negative, erythrocyte sedimentation rate was mildly elevated at 28 mm/hour (reference range, 0-20 mm/hour), and white blood cell (WBC) count was elevated at 15.8 K/mcL (4.0-10.0 K/mcL). He was diagnosed with suspected trigeminal neuralgia and discharged on prednisone and carbamazepine.

Two days later, he returned to the ED with headache, weakness, altered mental status, and bilateral vision loss. His vitals showed the following: temperature, 37.4 °C; heart rate, 107 beats per minute; and blood pressure, 105/72 mm Hg. He was confused with orientation to name only and had nuchal rigidity and a presumed new heart murmur. Ocular evaluation demonstrated bilateral dilated, nonreactive pupils with severely diminished visual acuity (light perception only). Lumbar puncture with cerebrospinal fluid (CSF) studies revealed elevated protein at 173 mg/dL (reference range, 12-60 mg/dL), normal glucose at 64 mg/dL (40-70 mg/dL), and an elevated WBC count of 1,306 mm^3 (0-5 mm^3) with neutrophilic predominance. Gram stain showed moderate Gram-positive cocci similar to pneumococci. Serum WBC count was elevated at 23.5 K/mcL (reference range, 4.0-10.0 K/mcL). The patient received a 30 mL/kg bolus of lactated Ringer's solution, ceftriaxone, vancomycin, acyclovir, and dexamethasone, and he was admitted to the intensive care unit for empiric treatment of bacterial meningitis.

During admission, ophthalmology was consulted. Their examination documented ongoing bilateral vision loss with 5 mm, minimally reactive pupils. Dilated fundoscopic exam revealed severe central retinal ischemia with scattered hemorrhages and complete pallor of bilateral optic nerves and maculae, suggestive of severe, bilateral ischemic optic neuropathy. Although acute retinal necrosis was initially thought to be related to meningitis, clinicians considered a concomitant giant cell arteritis. Subsequent evaluation by a retinal specialist revealed acute retinal necrosis, which was likely caused by varicella-zoster virus (VZV) or herpes simplex virus (HSV). Neither the ophthalmologist, nor the retinal specialist, found evidence of cell or flare in the anterior or posterior chambers. Additionally, polymerase chain reaction testing of CSF for VZV and HSV were negative. Vitreous culture was negative after five days and sample was insufficient for further polymerase chain reaction **(**PCR) testing.

Encephalitis panel, CSF, and blood cultures returned positive for *S pneumoniae.* Antibiotic therapy was guided by infectious diseases consult with recommendations to continue vancomycin and ceftriaxone. Acyclovir was discontinued. Vancomycin was discontinued after three days as the cultures were adequately sensitive to ceftriaxone.

On hospital day 3, brain magnetic resonance imaging (MRI) revealed a focus of acute ischemia in the left subcortical occipital lobe and an oblong focus of acute ischemia in the right posterior cerebellum, likely secondary to an embolic phenomenon. Neurology was consulted for stroke and possibility of giant cell arteritis, with recommendations for dual antiplatelet therapy for 21 days followed by aspirin monotherapy, as well as a temporal artery biopsy. The general surgery team did not pursue the biopsy because they considered it to be unlikely to change diagnosis or therapy. On hospital day 4, an echocardiogram revealed findings of aortic

stenosis, posterior mitral valve regurgitation, and mitral valve vegetation, consistent with endocarditis. Cardiothoracic surgery consult did not recommend surgical intervention given the patient's unresolved neurologic status.

On hospital day 7, the patient developed chest pain with troponin elevation of 150 ng/L (reference range, < 21 ng/L) with no ischemic electrocardiographic changes. Vitals signs were within normal limits. Computed tomography angiography of the chest showed pericardial effusion with concern for cardiac tamponade. An emergency repeat echocardiogram showed right ventricular dynamic collapse, confirming cardiac tamponade. He underwent emergent pericardiocentesis with removal of 400 mL of blood, resolving the tamponade. The cardiothoracic surgery team concluded that the tamponade developed secondary to mitral valve endocarditis eroding through the aortomitral curtain into a 5.8-cm ascending aortic aneurysm. Specialists at tertiary centers concluded that the patient's disease progression represented a terminal condition given the required extensive surgical interventions. The patient chose to pursue comfort measures. Antibiotics were discontinued, and he was discharged home on hospice.

While in hospice, the patient exhibited unexpected improvement in his clinical condition, leading him to seek a second opinion regarding cardiothoracic surgical options. Two weeks after initial hospital discharge, he underwent aortic and mitral valve replacement, two-vessel coronary artery bypass grafting, ascending aorta replacement, and pericardial patch repair of proximal aortic abscess/fistula. He had a remarkably uneventful postoperative recovery. To date, he is stable and recovering at home but remains blind with no light perception.

DISCUSSION

The incidence of pneumococcal meningitis in the United States is 0.3 per 100,000 people, with reported mortality rate as high as 37%.[2,7] For those who survive, devastating neurologic complications can occur in up to 52% of patients.[2] In the rare case of concomitant acute vision loss, establishing a differential diagnosis provides an opportunity to review the potential causes of vision loss and identify which features of each pathologic process correlate with the patient's presentation.

There was much discussion about the underlying etiology of our patient's bilateral visual impairment. The retinal specialist considered the patient's ocular findings to be consistent with acute retinal necrosis, implying concomitant viral infection in addition to the patient's bacterial meningitis. Acute retinal necrosis is caused by VZV and HSV, characterized by retinal inflammation, uveitis, vasculitis, and necrosis.[8] Fundoscopic examination typically demonstrates sharply demarcated areas of necrosis with evidence of inflammation of the anterior and posterior chambers.[8] Treatment consists of systemic antivirals and, occasionally, intravitreal antivirals.[8] Involvement of both eyes is not uncommon and can lead to permanent bilateral vision loss.[8] This can also be seen in the setting of viral meningitis but has not been reported as a complication of bacterial meningitis.[9] Contrary to ophthalmologist opinion, the infectious diseases specialist maintained that a concomitant bilateral viral retinal process in the setting of a severe bacterial infection would be unlikely. Additionally, CSF PCR was negative for HSV and VZV, and no inflammation was observed in either the anterior or posterior chambers, further supporting an unlikely diagnosis of acute retinal necrosis. The infectious diseases specialist concluded that the overwhelming burden of the systemic bacterial infection directly led to retinal injury and necrosis.

Another important consideration provided by neurology was the possibility of acute cortical blindness, an uncommon but recognized complication of bacterial meningitis.[10] Inflammation and edema are involved in the disease process, and vasculitis leads to cortical injury with focal necrosis.[11] Given that this is a cortical lesion, pupillary light reflexes and extraocular movements typically remain intact, and fundoscopic evaluation remains normal.[12] Although our patient did have evidence of cortical ischemia on MRI, it was likely secondary to septic embolic phenomena, and his loss of pupillary reflexes; therefore, his retinal findings made cortical blindness unlikely to be the primary cause of his vision loss.

Given the patient's presentation with headache and jaw pain, an important differential was acute ischemic optic neuropathy (AION), which can present as either arteritic or nonarteritic. Nonarteritic anterior ischemic optic neuropathy, which is typically idiopathic, accounts for 90% to 95% of cases of AION; investigators hypothesize that circulatory insufficiency to the optic disc ultimately leads to ischemia and painless vision loss.[8] The vision loss is typically mild, with the progressive form worsening over weeks.[8] Arteritic anterior ischemic optic neuropathy is a manifestation of giant cell arteritis, leading to disc ischemia and retinal necrosis, causing severe vision loss, which is typically unilateral but sometimes bilateral and sudden.[8,13] This was deemed unlikely as our patient had an erythrocyte sedimentation rate < 50 mm/hour, which did not rule out giant cell arteritis but made it far less likely.[14]

An uncommon but important diagnostic consideration was central retinal artery obstruction (CRAO). This ocular emergency typically causes painless, unilateral vision loss (98% to 99% of cases).[8] Examination shows a central area of pallor with a persistent cherry-red spot overlying the macula due to its thinner nerve layer.[8] This appearance persists for six weeks, after which the retinal pallor spontaneously resolves without progressing to necrosis.[8] Although our patient was at increased risk for embolic phenomena, his retinal findings were not consistent with CRAO.

CONCLUSION

Ultimately, the etiology of our patient's ocular symptoms and findings remain unclear. To our knowledge, this is the

only reported case of bacterial meningitis-associated retinal necrosis without evidence of endophthalmitis. Due to the severity of disease burden, we suspect a pathophysiologic process with both localized and cortical insults. We postulate that the severe systemic and localized inflammation from pneumococcal meningitis led to vasculitis and retinal necrosis. Although most of the diagnoses considered in this case are beyond the scope of the emergency clinician, general knowledge of their occurrence and basic pathophysiology can be vital in providing early diagnosis, treatment, and specialist involvement. Such early interventions are imperative both to guide initial diagnosis and treatment and preserve vision while decreasing overall morbidity and mortality.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Aji Yagou Jeng, MD, Trinity Health West Michigan Emergency Medicine Residency, 1675 Leahy St, Suite 315A, Muskegon, MI, 49442. Email: aji.jeng@trinity-health.org.

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Atypical Emergency Department Presentation of Acute Motor and Sensory Axonal Neuropathy: A Case Report

Shadman Kabir, DO
Austin Rahman, DO
Sitha Konopack, MD, MPH

University of Florida College of Medicine, Gainesville and UF Health Shands Hospital, Department of Emergency Medicine, Gainesville, Florida

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Introduction: Acute motor and sensory axonal neuropathy is a rare, severe axonal variant of Guillain–Barré syndrome characterized by rapidly progressive motor and sensory dysfunction. Early recognition in the emergency department (ED) is challenging because patients may initially present with subtle neurologic findings, preserved reflexes, and normal cerebrospinal fluid (CSF) studies.

Case Report: A 29-year-old woman with a history of generalized anxiety disorder presented to the ED with progressive distal numbness and mild weakness, with weakness being her chief complaint. Neurologic examination demonstrated preserved reflexes without focal deficits, and she was noted to be anxious. Given the reassuring examination, the patient was discharged with strict return precautions, including instructions to seek immediate reevaluation for worsening weakness, numbness, or progression of neurologic symptoms. She returned 48 hours later with rapidly worsening ascending weakness. The patient's clinical progression prompted neurology consultation and hospital admission despite initially reassuring examination findings. During a five-day hospitalization, electrodiagnostic testing and clinical findings established the diagnosis of acute motor and sensory axonal neuropathy.

This case illustrates how acute motor and sensory axonal neuropathy may initially mimic benign or functional conditions, particularly in patients with underlying anxiety, preserved reflexes, and normal early CSF findings. In the ED, anchoring bias and premature diagnostic closure may delay recognition of serious neurologic disease. Serial neurologic examinations, maintaining a broad differential diagnosis, and early neurology consultation are critical when evaluating patients with progressive neurologic symptoms.

Conclusion: Acute motor and sensory axonal neuropathy may present atypically during the early stages of disease, and initial examination and laboratory findings may be misleading. Clinical progression, rather than isolated examination or diagnostic findings, should guide disposition and prompt reassessment to facilitate timely diagnosis and treatment, with the potential to improve patient outcomes. [Clin Pract Cases Emerg Med. 2026;10(4):580–583.]

Keywords: *acute motor and sensory axonal neuropathy (AMSAN); Guillain-Barré syndrome; emergency department; diagnostic error; case report.*

INTRODUCTION

Guillain-Barré syndrome is an acute immune-mediated polyneuropathy classically characterized by ascending weakness and sensory deficits.[1,6] It is most commonly associated with demyelination of peripheral nerves, although axonal variants such as acute motor and sensory axonal

neuropathy (AMSAN) occur and are often associated with more severe disease.[2,3,6] Guillain-Barré syndrome is frequently preceded by a gastrointestinal or respiratory infection.[2,6]

Early recognition in the emergency department (ED) remains challenging because patients may initially lack the classic findings of areflexia, progressive ascending paralysis, and albuminocytologic dissociation.[4,6,7] This case highlights the importance of serial neurologic examinations, maintaining a broad differential diagnosis, and early neurology consultation when evaluating patients with progressive neurologic symptoms suggestive of Guillain-Barré syndrome despite initially reassuring findings.

CPC-EM Capsule

What do we already know about this clinical entity?
Acute motor and sensory axonal neuropathy (AMSAN) is a rare Guillain-Barré syndrome variant that may initially lack classic findings, delaying diagnosis.

What makes this presentation of disease reportable?
AMSAN presented with preserved reflexes, normal cerebrospinal fluid findings, and subtle upper extremity deficits, leading to a reassuring emergency evaluation.

What is the major learning point?
Serial neurologic examinations and recognition of clinical progression are essential when patients have persistent neurologic symptoms despite reassuring findings.

How might this improve emergency medicine practice?
Recognizing atypical Guillain-Barré syndrome presentations and avoiding anchoring bias can improve emergency department decision-making and timely treatment.

CASE REPORT

A 29-year-old woman with a history of generalized anxiety disorder presented to a community satellite ED with three days of progressive bilateral hand and foot numbness. Her medical history was notable for generalized anxiety disorder. Surgical history included tonsillectomy. Family history was noncontributory except for a cousin with multiple sclerosis. She denied tobacco, alcohol, or illicit drug use. Medications included vitamin D supplementation; she was not taking her prescribed bupropion. She reported a latex allergy (rash).

Her symptoms began approximately one week after a viral upper respiratory infection, initially as three days of intermittent hand paresthesias that progressed to persistent distal sensory deficits bilaterally in her upper arms/hands. She subsequently developed neck and shoulder discomfort and headache, along with early functional impairment, including difficulty lifting her toddler. She denied chest pain, shortness of breath, nausea, vomiting, and bowel or bladder dysfunction.

A focused neurologic examination demonstrated intact strength (5/5) in bilateral upper and lower extremities except for mild weakness (4/5) in finger abduction and adduction bilaterally. Reflexes were 2+ throughout, and Hoffman sign was negative bilaterally. Sensory examination revealed decreased light touch, pinprick, and vibration only in the hands (distal digits, palms) and feet (mid-foot) without a clear dermatomal distribution. Temperature sensation was preserved. Cranial nerve examination was normal, gait was steady, and no focal neurologic deficits were identified. Based on pertinent information at the time of presentation a broad differential diagnosis was considered, which included cervical myelopathy, inflammatory neuropathy including Guillain-Barré syndrome, metabolic derangements, and less likely central nervous system pathology. Many of the differentials had a low pretest probability based on presentation and duration of symptoms. Electrolyte abnormality was ruled out based on objective lab markers.

Laboratory evaluation, including complete blood count, basic metabolic panel, thyroid-stimulating hormone, magnesium, phosphorus, ionized calcium, urinalysis, and urine pregnancy test, were within normal limits. Computed tomography (CT) of the head and imaging of the cervical spine were unremarkable. The presence of bilateral upper distal symptoms following a recent viral illness raised concern for early Guillain-Barré syndrome. She received intravenous fluids, magnesium, nonsteroidal anti-inflammatory drugs, and vitamin supplementation without improvement. Due to diagnostic uncertainty and limited resources, she was transferred to a tertiary care ED for further evaluation and specialty consultation.

At the tertiary care ED, her symptoms persisted, but reflexes remained preserved. Negative inspiratory force measured −40 cm H_2O, above commonly used thresholds for ventilatory support but sufficiently reduced to warrant serial respiratory monitoring (reference range, −60 to −100 cm H_2O). No other studies were pursued at the tertiary care center; however, neurology was consulted. Given preserved reflexes, stable respiratory parameters, and unremarkable diagnostic studies, Guillain-Barré syndrome was considered less likely. In conjunction with neurology, the ED team determined the patient was appropriate for discharge with strict return precautions, which included worsening of numbness or increased weakness in her extremities. A rheumatology and neurology referral was provided to the

patient for further workup in the outpatient settin

She returned to the tertiary ED 48 hours later with worsening symptoms. These included progressive distal weakness (difficulty manipulating zippers and dropping objects), new generalized weakness, and ascending numbness in a stocking-glove distribution. Repeat neurologic examination on this visit showed the following:

- Right upper extremity: Deltoid, bicep, and triceps strength. 5/5; wrist extension, 4/5; finger abduction and finger extension, 3/5; finger flexion, 5/5 L
- Left upper extremity: Deltoid, biceps, and. triceps strength, 5/5; wrist extension, 5/5; finger flexion, 5/5; finger abduction and finger extension, 3/5

Lower extremities were 5/5 bilaterally. She demonstrated normal reflexes, unchanged from prior presentation.

Repeat noncontrast CT of the head and cervical spine showed no acute findings. A lumbar puncture was performed in the ED based on concern for Guillain-Barré syndrome, which showed CSF studies within normal limits (glucose, protein, cell count, clear colorless appearance). Given concern for rapidly evolving neuromuscular disease, magnetic resonance imaging of the brain and cervical spine were performed, which showed benign findings. After consultation with neurology, she was admitted to the neurology service. Corticosteroids were initiated while inflammatory myelopathy remained in the differential diagnosis.

During hospitalization, serial examinations demonstrated progression to symmetric distal greater than proximal weakness, areflexia in both upper and lower extremities, and persistent sensory deficits in her upper extremities (bilateral arms and hands). Repeat laboratory studies remained within normal limits. Serial respiratory monitoring showed negative inspiratory force values ranging from −40 to −52 cm H_2O. On hospital day 3, electromyography and nerve conduction studies demonstrated a severe axonal sensorimotor polyneuropathy consistent with AMSAN, a variant of Guillain-Barré syndrome. Following electrodiagnostic confirmation, corticosteroids were discontinued and intravenous immunoglobulin was initiated. The patient demonstrated improvement in strength while maintaining stable respiratory function without the need for ventilatory support. She was discharged with persistent but improved distal weakness and referred for outpatient neuromuscular follow-up and rehabilitation.

DISCUSSION

Acute motor and sensory axonal neuropathy is a rare axonal variant of Guillain-Barré syndrome characterized by immune-mediated injury to motor and sensory axons, often resulting in more severe clinical outcomes than demyelinating forms.[3] Early recognition in the ED is notoriously difficult; as seen in this case, patients may initially present with subtle neurologic deficits while maintaining preserved deep tendon reflexes and normal CSF studies.[7]

Diagnostic frameworks emphasize that early Guillain-Barré syndrome frequently lacks the classic triad of progressive ascending weakness, sensory symptoms, and areflexia.[4] Guillain-Barré syndrome encompasses multiple clinical variants, further contributing to diagnostic uncertainty. For example, Miller Fisher syndrome presents with ophthalmoplegia and ataxia,[14] while the pharyngeal-cervical-brachial variant involves bulbar and upper extremity weakness rather than the classic ascending pattern.[15] Unlike acute inflammatory demyelinating polyradiculoneuropathy, the demyelinating form of Guillain-Barré syndrome, AMSAN primarily involves axonal injury, which is associated with more severe weakness, slower recovery, and greater residual disability. Early clinical findings, however, may overlap substantially with other Guillain–Barré syndrome variants, making distinction difficult before electrodiagnostic testing. These variants underscore the clinical heterogeneity of Guillain-Barré syndrome and demonstrate why atypical presentations must remain in the differential diagnosis, even when traditional markers are absent.[2,4]

In this case, the patient's history of generalized anxiety disorder potentially contributed to diagnostic anchoring—a cognitive bias where clinicians rely too heavily on initial information or psychiatric history rather than performing a careful neurologic reassessment.[13] Awareness of such biases is essential to avoid premature diagnostic closure, especially since objective abnormalities in CSF and electrodiagnostic studies often lag behind clinical symptom onset.[2,7] Recent literature highlights this variability, reinforcing the importance of prioritizing the clinical trajectory and serial examinations over single time-point assessments.[7,10]

This case underscores the importance of strict return precautions. Emergency physicians are responsible for the disposition of every patient they treat. In this instance, the medical team appropriately transferred the patient to a tertiary center for neurologic evaluation before her symptoms worsened. While clinicians might consider a lumbar puncture during the initial visit, normal CSF findings early in the course of Guillain-Barré syndrome would not have altered the immediate management. Albuminocytologic dissociation develops in only approximately half of patients during the first week of illness; therefore, a normal CSF profile should not exclude Guillain-Barré syndrome early in its course.

Ultimately, providing clear return instructions enabled the patient to seek timely follow-up care as her condition evolved. Emergency physicians must prioritize emphasizing these precautions during the discharge process.

CONCLUSION

This case illustrates that early acute motor and sensory axonal neuropathy may not meet traditional diagnostic expectations despite evolving neuromuscular disease. In such patients, progression over time may be a more reliable diagnostic indicator than isolated examination findings.

Beyond its low incidence, the clinical complexity of this case stemmed from an atypical presentation involving symptoms primarily in the upper extremities, preserved reflexes, and unremarkable CSF. These subtle findings complicated early diagnostic assessment and contributed to initially low clinical suspicion for Guillain-Barré syndrome.

The initial stages of Guillain-Barré syndrome frequently lack traditional diagnostic hallmarks, particularly within the first week of symptom onset, which complicates early identification. Consequently, this report stresses the necessity of performing serial neurologic assessments, obtaining prompt neurology consultation, and maintaining a high index of suspicion for Guillain-Barré syndrome even when initial workups are reassuring. The presence of progressive weakness warrants meticulous reassessment and careful disposition planning, regardless of preserved reflexes or normal CSF parameters.

For the emergency physician, this clinical scenario emphasizes the critical need to mitigate anchoring bias and recognize that unconventional presentations of Guillain-Barré syndrome may appear deceptively benign. Early recognition, close reassessment, and timely initiation of immunotherapy remain critical to reducing morbidity and preventing respiratory compromise in patients with Guillain-Barré syndrome.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file

Address for Correspondence: Shadman Kabir, DO, University of Florida College of Medicine, Gainesville and UF Health Shands Hospital, Department of Emergency Medicine 1329 SW 16th Street, PO Box 100186 Gainesville, FL 32610-0186. Email: Skabir@ufl.edu.

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Acute Myocardial Infarction Following Tenecteplase: A Case Report of Complex Dual Pathology

Devin Howell, DO[*†‡]
Ramses Ramirez Damera, MD[§]
Gaurav Rao, MD[*†¶]

[*]Northwell Health, New Hyde Park, New York
[†]Donald and Barbara Zucker School of Medicine, Hempstead, New York
[‡]Huntington Hospital, Department of Emergency Medicine, Huntington, New York
[§]Nassau University Medical Center, East Meadow, New York
[¶]Huntington Hospital, Department of Cardiology, Huntington, New York

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Introduction: Acute ischemic stroke is a leading cause of morbidity and mortality, with tenecteplase being a cornerstone of acute management. While tenecteplase carries a known risk of bleeding, major thrombotic events such as acute myocardial infarction (AMI) after stroke reperfusion are less commonly described.

Case Report: We present the case of a 67-year-old man who presented with acute stroke symptoms. After ruling out intracranial hemorrhage and managing blood pressure, he received tenecteplase, and all neurological deficits resolved. Soon after, he developed acute chest pain, and his repeat electrocardiogram confirmed a ST-elevation myocardial infarction. He underwent emergent cardiac catheterization, revealing 100% occlusion of the left circumflex artery, which was successfully revascularized with thrombectomy and angioplasty. Intravascular ultrasound further supported the presence of embolic phenomenon. Pathology of the retrieved material identified thrombus alongside atherosclerotic plaque fragments. The patient experienced complete symptom resolution and was discharged after a short hospitalization.

This case highlights the complexities encountered when a patient with acute ischemic stroke who is treated with tenecteplase subsequently develops an AMI. Potential mechanisms include a coincidental plaque rupture, possibly exacerbated by the stress of the acute stroke, a rare paradoxical prothrombotic effect of thrombolysis, or a combination thereof. The pathological finding of mixed thrombus and atherosclerotic plaque underscores the underlying disease contributing to this event.

Conclusion: This case accentuates the importance of maintaining a high index of suspicion for new cardiovascular events in post-thrombolysis patients, especially those with significant cardiovascular risk factors. Rapid and judicious diagnostic evaluation is crucial for timely management of these complex, life-threatening complications. [Clin Pract Cases Emerg Med. 2026;10(4):584–588.]

Keywords: *stroke; STEMI; thrombolytics; tenecteplase; case report.*

INTRODUCTION

Acute ischemic stroke remains a leading cause of long-term disability and death worldwide.[1] Rapid reperfusion therapies, including intravenous thrombolysis with agents such as tenecteplase are critical for improving neurological outcomes by restoring blood flow to ischemic brain tissue.[2] Tenecteplase, a modified recombinant tissue plasminogen activator, offers practical advantages over alteplase, including

a single bolus administration, and has demonstrated noninferiority and potential superiority in certain contexts.[3,4] While tenecteplase is highly effective, its use carries an inherent risk of bleeding complications, most notably intracranial hemorrhage.[2] Less frequently reported are major arterial thrombotic events distant from the initial cerebrovascular occlusion, such as acute myocardial infarction, which can present a significant diagnostic and therapeutic challenge in the post-thrombolysis setting.[5]

This case report describes a unique scenario where a patient achieved complete neurological recovery following tenecteplase administration for acute ischemic stroke, only to subsequently develop an acute posterolateral ST-elevation myocardial infarction (STEMI). The pathology of the retrieved coronary thrombus revealed a combination of thrombus and calcified atherosclerotic plaque, providing critical insights into the potential underlying mechanisms. This report highlights the diagnostic complexities, discusses potential pathophysiological considerations, and emphasizes the clinical vigilance required in managing such intricate cases.

CPC-EM Capsule

What do we already know about this clinical entity?
Tenecteplase is commonly administered for stroke, ST-elevation myocardial infarction (STEMI), and pulmonary embolism (PE).

What makes this presentation of disease reportable?
Thromboembolic events following tenecteplase administration are rare but life-threatening.

What is the major learning point?
Patients receiving tenecteplase should be monitored for hemorrhage and thromboembolic events, including STEMI, PE, or peripheral arterial obstruction.

How might this improve emergency medicine practice?
Thrombolytic administration is common when time to catheterization is >120 minutes. Readers should be aware of this rare complication of tenecteplase.

CASE REPORT

A 67-year-old man with a medical history significant for hypertension, hyperlipidemia, a known cerebral aneurysm status postcoiling, a thoracic aortic aneurysm (stable, nonsurgical), and gastroesophageal reflux disease, presented to the emergency department with acute onset left-sided weakness, slurred speech, and facial droop. His symptom onset was approximately one hour prior to arrival.

Upon presentation, his vital signs were as follows: heart rate, 84 beats per minute; blood pressure, 187/121 mm Hg; respiratory rate, 18 breaths per minute; SpO_2, 98% on room air; and blood glucose 89 mg/dL (reference range, 70–99 mg/dL). Neurological examination revealed a National Institutes of Health Stroke Scale (NIHSS) score of 10, characterized by partial paralysis of the left face (two points), left arm and leg hemiparesis with no effort against gravity (arm = three points, leg = three points), and severe dysarthria (two points). He was activated as a code stroke based on hospital policy.

Noncontrast computed tomography of the brain no evidence of acute intracranial hemorrhage or mass effect. A subsequent computed tomography angiogram (CTA) of the head and neck did not reveal any large vessel occlusion amenable to endovascular thrombectomy, and his known cerebral aneurysm appeared stable. Laboratory workup, including a complete blood count, complete metabolic panel, and coagulation studies, were within reference range, with an initial high-sensitivity troponin being undetectable. Following blood pressure control using a single push-dose of intravenous (IV) labetalol to achieve parameters below 185/110 mm Hg, the patient received a single bolus of tenecteplase (0.25 mg/kg) approximately 95 minutes from symptom onset. An IV infusion of nicardipine IV was initiated to maintain blood pressure below 180/105 mm Hg.

Within 15 minutes of tenecteplase administration, the patient exhibited significant neurological improvement. A repeat neurological assessment demonstrated complete resolution of his left-sided weakness, facial droop, and dysarthria, with a subsequent NIHSS score of 0. Approximately 30 minutes after tenecteplase administration, the patient developed sudden, severe, retrosternal chest pain radiating to his left arm, accompanied by diaphoresis. An immediate 12-lead electrocardiogram (ECG) revealed massive ST segment depressions in leads V1–V6 (Image 1).

A posterior 12-lead ECG confirmed ST-segment elevations in V7–V9, indicating an acute posterolateral STEMI. Serial troponin levels rapidly rose, peaking at over 8,000 ng/L (reference range, < 20 ng/L). Given his history of an aortic aneurysm, an immediate CTA of the chest was performed to rule out aortic dissection, which resulted as negative. The patient was not given antiplatelets or heparin in the setting of recent tenecteplase administration. He was emergently transferred to the cardiac catheterization laboratory for further management.

Coronary angiography revealed a 100% thrombotic occlusion of the mid-left circumflex artery (Image 2).

The culprit lesion was successfully wired, and a small-balloon angioplasty was performed to create a channel. This was followed by aspiration thrombectomy using a catheter to

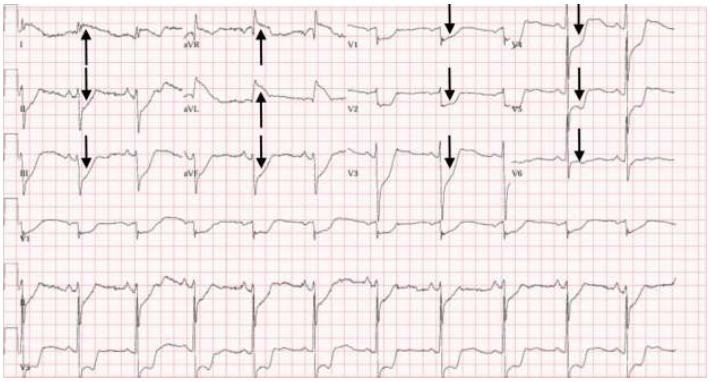


Image 1. Electrocardiogram performed with initial onset of chest pain. Depressions noted in leads II, III, aVF, and V1-V6 with elevation in leads I, aVR, and aVL

remove significant thrombotic material (Image 3), resulting in thrombolysis in myocardial infarction grade 3 flow and ST-segment resolution.

Intravascular ultrasound was used to characterize the culprit site, revealing a lack of significant underlying atherosclerotic burden or evidence of a ruptured fibroatheroma. Due to the favorable angiographic result and absence of a high-grade stenotic lesion, stent placement was deferred. Pathological analysis of the retrieved revealed the presence of a thrombus extensively intermingled with calcified atherosclerotic plaque fragments. This finding strongly suggested mobilization and acute plaque rupture of aortic or proximal arterial debris as the underlying mechanism for the STEMI.

Following successful revascularization, the patient's chest pain resolved. His postprocedure course in the intensive care unit was uneventful. A transthoracic echocardiogram revealed normal left ventricular function without regional wall motion abnormalities corresponding to the lateral and posterior walls, and an ejection fraction of 56%. He was initiated on guideline-directed medical therapy, including aspirin, high-intensity statin, beta blocker, and an angiotensin-II receptor blocker. The patient remained neurologically intact, with no recurrence of stroke symptoms or bleeding complications. He was discharged on hospital day 6 in stable condition with close outpatient follow-up arranged for both his cardiac and neurological conditions.

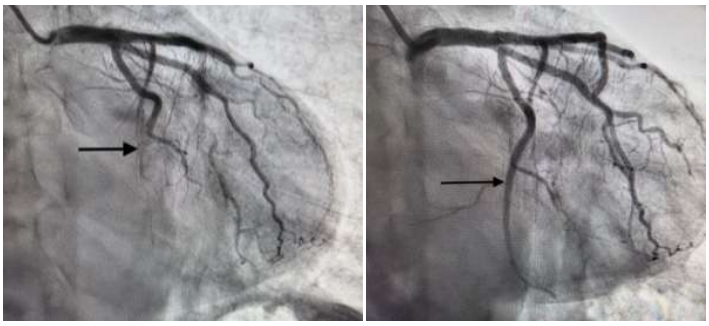


Image 2. Coronary angiography of the left main coronary artery showing 100% occlusion of the left circumflex artery (left). Coronary angiography of the left main coronary artery after thrombectomy with thrombolysis in myocardial infarction 3 flow (right).

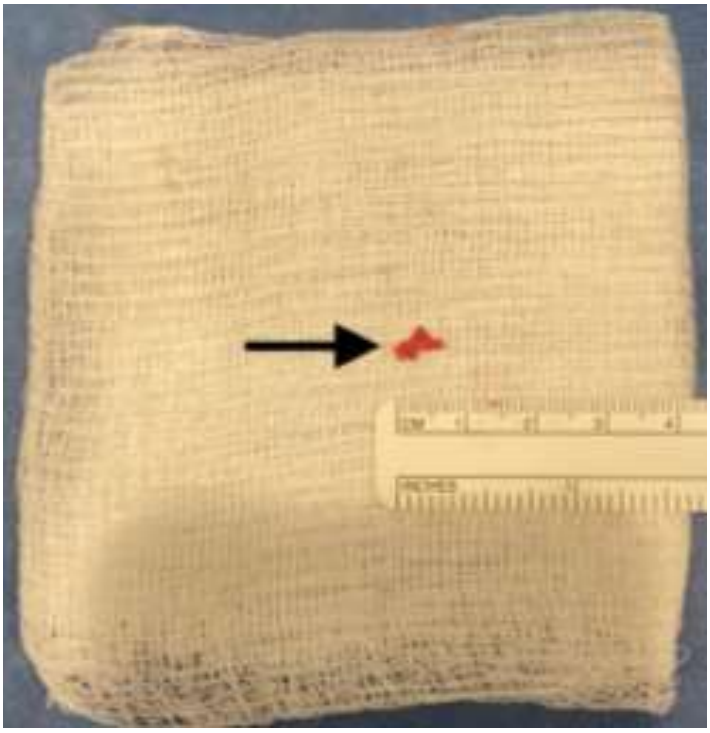


Image 3. Thrombotic atherosclerotic plaque removed during coronary angiography using a catheter.

DISCUSSION

This case highlights a rare but critical sequence of events: an elderly man with multiple cardiovascular risk factors who experienced an acute ischemic stroke with complete resolution following tenecteplase, only to develop an acute posterolateral STEMI shortly thereafter. This complex presentation underscores the challenges in diagnosing and managing concurrent or sequential cardiovascular and cerebrovascular events, particularly in the context of recent thrombolytic therapy.

The initial neurological presentation and rapid response to tenecteplase were textbook examples of successful thrombolysis for acute ischemic stroke. However, the subsequent development of STEMI presented a significant diagnostic dilemma. Given the patient's history of an aortic aneurysm, aortic dissection was a primary concern, necessitating a prompt aortic CTA to rule out this life-threatening condition. The immediate availability of a

comprehensive diagnostic pathway, including serial electrocardiograms (ECG), troponin levels, and imaging, was crucial in differentiating the cause of chest pain and activating the appropriate "Code STEMI" pathway.

Several mechanisms could explain the occurrence of STEMI in this patient. His extensive cardiovascular risk factors predisposed him to advanced atherosclerosis. The pathological finding of thrombus with associated calcified atherosclerotic plaque with normal coronary artery architecture strongly supports the hypothesis of embolic phenomenon as the primary event leading to left circumflex artery occlusion. The acute stress associated with the stroke, including sympathetic surge, inflammatory response, and hemodynamic fluctuations, could have further destabilized a vulnerable plaque, leading to its rupture.[6]

While thrombolytics like tenecteplase are designed to dissolve clots, there have been theoretical discussions and rare case reports suggesting a potential paradoxical prothrombotic effect or rebound hypercoagulable state following systemic thrombolysis.[5,7,8] This could occur through various mechanisms, such as increased thrombin generation, platelet activation, or depletion of natural anticoagulants. In a patient with preexisting unstable atherosclerotic plaque, such a state could potentially precipitate or exacerbate acute thrombus formation at another site.

While intracranial hemorrhage is the most feared bleeding complication of thrombolysis, new arterial thrombotic events distant from the original clot, such as myocardial infarction, are considerably rarer. A search of the medical literature revealed a few case reports rather than large-scale studies.[5,9] One review highlighted cases of acute limb ischemia or myocardial infarction occurring after systemic thrombolysis for pulmonary embolism or stroke, attributing some to paradoxical hypercoagulability but acknowledging the challenge in differentiating from coincidental events.[5] We identified only two other case reports that described a STEMI occurring after tenecteplase for stroke, where the underlying mechanism was also attributed to plaque rupture.[8,9] Our case adds to this limited body of evidence, providing the unique pathological confirmation of underlying atherosclerotic plaque alongside thrombus in the retrieved material without histological evidence of coronary disease on imaging. This suggests that while systemic hemostatic changes induced by tenecteplase cannot be entirely excluded, the primary event was likely related to the patient's preexisting and highly vulnerable aortic atherosclerosis leading to coronary embolism.

This case highlights several critical clinical implications. First, a high index of suspicion for new cardiovascular events must be maintained in post-thrombolysis stroke patients, even in the setting of successful neurological recovery. Patients with multiple cardiovascular risk factors are particularly vulnerable. Second, rapid and systematic evaluation, including serial ECGs, troponin measurements, and judicious use of imaging (ie, CTA to rule out aortic dissection), is paramount to distinguish new symptoms from other potential complications or coincidental events. Finally, the management of acute coronary syndromes in the context of recent thrombolysis requires careful consideration of bleeding risk versus ischemic risk, particularly when deciding on antithrombotic and antiplatelet regimens and the approach to revascularization. In our case, the decision to defer stent placement in favor of thrombectomy and balloon angioplasty reflected an attempt to minimize further potent antiplatelet requirements and bleeding risk in the immediate post-tenecteplase period.

LIMITATIONS

This is a single case report, limiting the generalizability of our findings. While the pathological findings provide strong evidence for coronary embolization, it is impossible to definitively determine the exact interplay between tenecteplase administration, the acute stroke, and the timing of the myocardial infarction without a larger cohort of similar cases.

CONCLUSION

This case report describes a unique and challenging clinical scenario of an acute STEMI succeeding successful tenecteplase administration for an acute ischemic stroke. The pathological and IVUS findings of thrombus with associated calcified plaque fragments in an otherwise non-obstructive vessel support an embolic mechanism, likely from an aortic source. This case underscores the need for vigilance regarding multi-territory embolic events in patients with a high burden of systemic atherosclerosis undergoing thrombolysis. Rapid diagnostic evaluation and multidisciplinary management are essential for optimizing outcomes in these complex patients.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Devin Howell, DO, Northwell Health, 2000 Marcus Avenue, Suite 300, New Hyde Park, NY 11042. Email: dhowell6@northwell.edu.

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From Frenulum Tear to Transfusion—An Unexpected Diagnosis of Hemophilia in a Toddler: A Case Report

Saxony Busta, DO
Mary Jane Piroutek, MD

Rady Children's Health Orange County, Department of Pediatric Emergency Medicine, Orange, California; University of California Irvine

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Introduction: Labial frenulum tears in infants are commonly associated with minor trauma but may raise concern for nonaccidental injury. These injuries are typically benign and self-limiting; however, persistent or excessive bleeding should prompt evaluation for an underlying bleeding disorder. Hemophilia A is an X-linked factor VIII deficiency with clinical symptoms that vary based on the level of severity.

Case Report: A previously healthy 14-month-old male presented to the pediatric emergency department three times within 10 days for persistent bleeding from a superior labial frenulum tear sustained after minor trauma. Initial examination was reassuring, and he was discharged without laboratory evaluation. On repeat presentation, he was found to have normocytic anemia and isolated prolonged activated partial thromboplastin time. Despite topical and oral antifibrinolytic therapy, he returned with worsening anemia, melena, hematemesis, pallor, and tachycardia. He required a packed red blood cell transfusion, intravenous aminocaproic acid, and hospitalization. Further hematologic evaluation revealed a low factor VIII activity level, confirming mild hemophilia A. There was no prior personal or family history of abnormal bleeding.

Conclusion: This case highlights that hemophilia A may initially present with disproportionate bleeding from a minor mucosal injury, even in the absence of a known bleeding history. Persistent bleeding after minor trauma warrants prompt coagulation evaluation to prevent diagnostic delay and serious complications. [Clin Pract Cases Emerg Med. 2026;10(4):589–592.]

Keywords: *hemophilia A; labial frenulum tear; pediatric bleeding; nonaccidental trauma differential; case report.*

INTRODUCTION

Labial frenulum tears in infants and toddlers are commonly associated with minor falls and accidental trauma. Such injuries warrant careful evaluation through detailed history-taking and thorough physical examination to exclude nonaccidental trauma. Isolated frenulum tears are typically benign and, like other minor mucosal injuries, often heal rapidly without intervention. However, persistent or excessive bleeding should prompt evaluation for an underlying bleeding disorder.

Hemophilia is an X-linked recessive, inherited coagulopathy that predominantly affects males and is characterized by factor VIII deficiency in hemophilia A and by factor IX deficiency in hemophilia B.[1] The incidence is approximately one in 5,000 male births, with hemophilia A accounting for about 80% of cases.[2] Clinical manifestations typically include spontaneous or trauma-induced prolonged bleeding, and an isolated prolonged activated partial thromboplastin time (aPTT) should raise clinical suspicion.[3] Diagnosis is confirmed by decreased plasma factor VIII or IX activity after exclusion of other coagulation abnormalities, including von Willebrand disease.[4] Disease severity correlates with factor activity levels and clinical phenotype.[5] Acute bleeding episodes require urgent treatment with factor replacement therapy.[4] Although hemophilia is primarily

inherited, de novo mutations account for 30–50% of cases.[3] We present a case of a 14-month-old male patient whose initial manifestation of hemophilia A was a bleeding superior labial frenulum tear, resulting in severe anemia from acute blood loss requiring transfusion and hospitalization.

CPC-EM Capsule

What do we already know about this clinical entity?
Hemophilia A causes prolonged bleeding, but mild disease may remain undiagnosed until minor injury and may mimic nonaccidental trauma.

What makes this presentation of disease reportable?
An accidental, isolated labial frenulum tear led to transfusion-requiring anemia and diagnosis of previously unrecognized mild hemophilia A in a healthy toddler.

What is the major learning point?
Persistent bleeding after minor trauma should prompt evaluation for an underlying bleeding disorder even with no family history.

How might this improve emergency medicine practice?
Early coagulation evaluation in children with persistent mucosal bleeding may prevent diagnostic delays and inappropriate concerm for nonaccidental trauma.

CASE REPORT

A previously healthy, fully vaccinated, ex-full term 14-month-old male with a history of prelingual bilateral sensorineural hearing loss presented to the pediatric emergency department (ED) three times within 10 days for persistent bleeding from a superior labial frenulum injury.

His initial visit was for a frenulum injury after biting a plastic baby gate. The patient's family called the nurse advice line and applied pressure to the area, but when the bleeding continued for approximately five hours despite pressure, the family presented to the pediatric ED. His physical exam on the first ED visit was notable for an upper lip frenulum laceration with minimal bleeding and no indication for repair. There were no other signs of trauma, bruising, or injury. Neurologic, abdominal, and musculoskeletal examinations were unremarkable. Vital signs were stable and appropriate for age. No laboratory testing or imaging was performed, bleeding stopped, and he was discharged with acetaminophen analgesia and anticipatory guidance.

Eight days later he returned to the pediatric ED after his mother found blood and clots on his bedding while he was sleeping. She reported intermittent bleeding from the upper gum since the initial injury but denied new trauma, hematuria, hematochezia, or personal or family history of bleeding disorders. His physical exam was notable for dried blood around the mouth with faint oozing from the upper lip frenulum with a small clot in place. Vital signs remained stable and appropriate for age. Laboratory evaluation revealed a normocytic anemia with hemoglobin 8.4 gm/dL (reference range, 8.0-21.0 gm/dL); hematocrit, 24.2% (33.0-39.0%); mean corpuscular volume (MCV), 80 fL (70-86 fL), prolonged aPTT 42.5 seconds (23.1-38.3 seconds); prothrombin time, 12.9 seconds (12.1-14.5 seconds); international normalized ratio (INR), 0.90 (0.86-1.14 INR); white blood count (WBC), 8.2 $\times 10^3/\mu L$ (5.0-15.5 $\times 10^3/\mu L$); platelet count 327 $\times 10^3/\mu L$ (250-600 $\times 10^3/\mu L$); fibrinogen, 237 (mg/dL) (212-466 mg/dL); D-dimer < 0.27 mcg/mL fibrinogen equivalent units (FEU) (0.09-0.53 mcg/mL FEU); and a normal complete metabolic panel (CMP). See table. Pediatric hematology recommended factor assays (VIII, IX, and XIII) and antifibrinolytic therapy. Topical thrombin was applied to the upper lip frenulum, but the patient spit it out and bleeding continued; he was then given oral aminocaproic acid. Two hours later the bleeding stopped, and the patient was discharged home with a five-day course of oral aminocaproic acid four times daily and pediatrician follow-up.

The patient re-presented to the pediatric ED 26 hours later for the third time with continued bleeding. Despite four doses of oral aminocaproic acid at home he continued to bleed from the frenulum injury. His mother also endorsed two episodes of black stools, one episode of bloody emesis, decreased activity level, and increased skin pallor. Physical exam was notable for pallor, dried blood on the face, and a blood clot at the upper lip frenulum with oozing around the clot. The patient was mildly tachycardic (146 beats per minute) with all other vital signs stable and appropriate for age. Repeat laboratory testing demonstrated worsening normocytic anemia (hemoglobin 5.2 gm/dL, hematocrit 15.1%, MCV 80 fL); normal WBC (8.4 $\times 10^3/\mu L$); mild thrombocytopenia platelet count (246 $\times 10^3/\mu L$) and a normal CMP. He received 10 mL/kg packed red blood cells over three hours and an intravenous (IV) dose of aminocaproic acid (50 mg/kg). The patient was admitted to the pediatric inpatient service with hematology consultation for severe anemia secondary to acute blood loss. After 24 hours of IV aminocaproic acid and one blood transfusion, his hemoglobin improved to 8.1 gm/dL. The patient returned to functional baseline without further evidence of bleeding. He remained clinically stable with vital signs appropriate for age and was discharged home after 24 hours with a five-day course of aminocaproic acid, outpatient hematology follow-up, and family education.

Further hematologic evaluation demonstrated a low factor VIII activity level (11% [reference range, 55-200%]), confirming the diagnosis of mild hemophilia A. The remainder

Table. All laboratory results for multiple emergency department visits and admission of toddler diagnosed with mild hemophilia A.

Laboratory Test	Second ED Visit (8 days postinjury)	Third ED Visit (9 days postinjury)	Factor Studies (Resulted after discharge)	Reference Range
WBC	8.2 $\times 10^3$/μL	8.4 $\times 10^3$/μL	—	5.0–15.5 $\times 10^3$/μL
Hemoglobin	**8.4 gm/dL**	**5.2 gm/dL**	—	8.0–21 gm/dL
Hematocrit	**24.2%**	**15.1%**	—	33.0–39.0%
MCV	80 fL	80 fL	—	70–86 fL
Platelets	327 $\times 10^3$/μL	246 $\times 10^3$/μL	—	250–600 $\times 10^3$/μL
aPTT	**42.5 seconds**	—	—	23.1–38.3 seconds
PT	12.9 seconds	—	—	12.1–14.5 seconds
INR	0.90	—	—	0.86–1.14
Fibrinogen	237 mg/dL	—	—	212–466 mg/dL
D-Dimer	<0.27 mcg/mL FEU	—	—	0.09–0.53 mcg/mL FEU
CMP	Normal	Normal	—	Normal
Factor VIII activity level	—	—	**11%**	55–200%
Factor VIII inhibitor screen	—	—	Negative	Negative
Factor IX activity level	—	—	91%	55–150%
Von Willebrand Factor Antigen	—	—	87%	55–200%
Von Willebrand Factor Activity	—	—	82%	55–200%

*Bolded values indicate significant values

ED, emergency department; *WBC*, white blood cel; μL, microliters; *gm/dL,* gram per deciliter; *MCV*, mean corpuscular volume; fL, femtoliters; *aPTT*, activated partial thrombin time; *PT*, prothrombin time; *INR*, international normalized ratio; *CMP*, comprehensive metabolic panel; *FEU*, fibrinogen equivalent units.

of his hematology workup was normal with a negative factor VIII inhibitor screen; normal factor IX activity level (91% [55-150%]); and normal von Willebrand factor (vWf) antigen and activity (vWF antigen 87% [55-200%]; vWF activity 82% [55-200%]). Of note, factor IX activity level and von Willebrand panel testing were sent post transfusion.

DISCUSSION

Severe hemophilia typically presents in the first months of life with spontaneous bleeding, while mild and moderate disease more commonly presents later in childhood and adolescence following trauma. Although the median age at diagnosis is approximately 36 months for mild hemophilia, this diagnosis should also be considered in younger children with bleeding that is prolonged or disproportionate to the reported mechanism of injury, even in the absence of a known family history.[6,7] While most patients with hemophilia A have a prolonged aPTT, a normal aPTT does not exclude mild disease. Measurement of factor VIII and IX levels should be considered in patients with a normal complete blood count and prothrombin time who have unexplained or disproportionate bleeding even when the aPTT is normal.

Our patient had a factor VIII level of 11%, consistent with mild hemophilia A. Despite undergoing circumcision and routine immunizations without significant bleeding complications, he presented with oral bleeding following a relatively minor trauma. This case illustrates the variable clinical presentation of mild hemophilia and highlights that the absence of significant bleeding during previous procedures does not exclude the diagnosis. The severity of hemophilia A and therefore the severity of hemorrhage is directly correlated to the scarcity of factor VIII. Normal factor VIII levels range between 50–150%. Severe hemophilia is defined as factor VIII activity less than 1%, moderate disease as 2–5%, and mild disease as 6–40%. Although spontaneous bleeding is uncommon in mild hemophilia, affected patients remain at risk for excessive bleeding following relatively insignificant trauma.[4,8] Treatment of acute bleeding in hemophilia A consists of prompt recombinant factor VIII replacement and should be initiated immediately, even before completion of patient assessment. Other adjuvant therapies include desmopressin, tranexamic acid, epsilon aminocaproic acid, and management of factor inhibitors.[3,4,7]

This case also highlights an important diagnostic consideration when evaluating young children with unexplained bleeding. Frequent falls and minor trauma during early ambulation can result in contusions and hemorrhage in children with hemophilia that may mimic nonaccidental trauma. Our patient presented with a labial frenulum tear, an injury that has been associated with several abusive mechanisms such as forceful feeding, stretching of the lip, or direct blows to the mouth.[9,10] However, isolated frenulum tears are not pathognomonic for abuse and have also been reported frequently after accidental falls in ambulatory children.[11] Comprehensive history-taking, thorough physical examination,

and consideration of underlying bleeding disorders are essential when evaluating pediatric patients with injuries concerning for nonaccidental trauma to ensure accurate diagnosis while maintaining appropriate vigilance for child abuse.

CONCLUSION

Our patient developed severe anemia from acute blood loss requiring transfusion and hospitalization following multiple emergency department visits for a minor mucosal injury. Prior to presentation, he had no history of abnormal bleeding despite a history of circumcision and no known family history of bleeding disorders. This case highlights the potential severity of bleeding disorders in pediatric patients and underscores that hemophilia may present atypically, even in the setting of a reassuring history. Minor trauma accompanied by excessive or persistent bleeding should prompt evaluation for an underlying bleeding disorder, because early recognition and intervention are critical to preventing morbidity and potentially life-threatening complications.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Rady Children's Health Orange County, Department of Pediatric Emergency Medicine, Orange, California; University of California Irvine, 1201 W La Veta Ave Orange, CA 92868. Email: saxony.busta@choc.org.

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Retrocaval Ureter Diagnosed Following Point-of-Care Ultrasound Detection of Hydronephrosis: A Case Report

Noah Marzook, MD*
Mostafa Alhabboubi, MD†
Ahmad Koshak, MD‡
Abdullah Basnawi, MD‡
Yasser Ajabnoor, MD†
Suha Albadr, MD§

*University of Jeddah, Faculty of Medicine, Department of Pediatrics, Jeddah, Saudi Arabia
†University of Jeddah, Faculty of Medicine, Department of Family Medicine, Jeddah, Saudi Arabia
‡University of Tabuk, Faculty of Medicine, Department of Internal Medicine, Division of Emergency Medicine, Tabuk, Saudi Arabia
§King Abdullah University of Science and Technology, International Medical Clinic, Department of Radiology, Thuwal, Saudi Arabia

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Introduction: Retrocaval ureter (RCU) is a rare congenital anomaly resulting from abnormal development of the inferior vena cava, leading to ureteral obstruction and hydronephrosis. Patients often present with nonspecific symptoms, making diagnosis challenging. Point-of-care ultrasound (POCUS) is frequently used in the emergency department (ED) to evaluate abdominal pain and may identify clinically important abnormalities requiring further investigation.

Case Report: A 30-year-old woman presented to the ED with a two-day history of nonspecific abdominal pain. Physical examination was notable only for mild epigastric tenderness. Abdominal POCUS performed by the emergency physician demonstrated moderate right-sided hydronephrosis. The patient had no urinary symptoms and normal renal function. Follow-up renal ultrasound confirmed hydronephrosis and proximal hydroureter without nephrolithiasis. Contrast-enhanced computed tomography subsequently revealed a RCU with the characteristic fish-hook configuration. Renography demonstrated preserved renal function with delayed drainage secondary to ureteral obstruction. The patient underwent surgical correction with ureteral reconstruction and stent placement.

Conclusion: This case highlights the value of POCUS in the evaluation of patients with nonspecific abdominal pain. Detection of hydronephrosis prompted further diagnostic evaluation and led to the diagnosis of a rare congenital urologic anomaly requiring surgical intervention. Emergency physicians should consider further investigation when hydronephrosis is identified on POCUS, even in the absence of urinary symptoms. [Clin Pract Cases Emerg Med. 2026;10(4):593–596.]

Keywords: *retrocaval ureter; hydronephrosis; point-of-care ultrasound; abdominal pain; case report.*

INTRODUCTION

Abdominal pain is one of the most frequent complaints of patients presenting to the emergency department (ED), accounting for 5-10% of ED visits.[1] Abdominal point-of-care ultrasound (POCUS) enhances diagnostic accuracy and streamlines the clinical workup for patients presentingwith abdominal pain.[2] In this case report, we describe a patient presenting to the ED with nonspecific abdominal pain, where abdominal POCUS was done as a part of the emergency physician's routine assessment. This led to the eventual diagnosis of a retrocaval ureter (RCU), a rare congenital urogenital abnormality for which surgical intervention is required. To the best of our knowledge, no previous cases of RCU have been reported in the literature in the context of using POCUS.

CASE REPORT

A 30-year-old woman presented to the ED with a two-day history of abdominal pain. She had no fever, vomiting, or diarrhea. On physical examination, her vitals were normal; the only notable finding was mild epigastric tenderness. Abdominal POCUS revealed right renal pelvis dilation. The patient did not report any urinary symptoms. Laboratory results showed a normal urinalysis; creatinine was 66 µmol/L (reference range, 45–90 µmol/L), and the calculated glomerular filtration rate was 110 mL/min/1.73 m^2 (90–120 mL/min/1.73 m^2). The physician considered the finding of hydronephrosis as potentially incidental and discharged the patient with a preliminary diagnosis of gastritis. A follow-up outpatient renal ultrasound was arranged to evaluate the hydronephrosis further and determine its underlying cause.

Two weeks later, the renal ultrasound showed moderate right-sided hydronephrosis with proximal hydroureter and no detectable renal stones (Image 1). The subsequent week, the patient was seen by a urologist, to whom she continued to complain of persistent abdominal pain. A contrast computed tomography (CT) (Image 2) showed persistent moderate hydronephrosis and proximal third hydroureter, with the renal pelvis reaching 2.2 cm in diameter. The proximal ureter deviated medially and posteriorly relative to the inferior vena cava (IVC), descending anterior to the aortocaval groove, displaying a positive "fish-hook sign." The patient was diagnosed with RCU.

Further investigation with a renogram showed preserved right kidney but delayed drainage due to ureteral compression of the right kidney. The patient underwent a right ureteral transposition, resection, and ureteroureterostomy over an indwelling double-J stent to correct the abnormality.

CPC-EM Capsule

What do we already know about this clinical entity?
Retrocaval ureter is a rare congenital anomaly in which the ureter passes behind the inferior vena cava, causing obstruction and hydronephrosis.

What makes this presentation of disease reportable?
The anomaly was found incidentally when point-of-care ultrasound (POCUS) showed hydronephrosis in a patient with nonspecific abdominal pain and no urinary symptoms.

What is the major learning point?
Hydronephrosis on POCUS warrants further imaging even without urinary symptoms; it can reveal surgically correctable anomalies such as retrocaval ureter.

How might this improve emergency medicine practice?
Using abdominal POCUS in undifferentiated abdominal pain can prompt earlier imaging, diagnosis, and timely referral for treatable urologic anomalies.

DISCUSSION

Retrocaval ureter is a rare congenital urogenital anomaly. The ureters typically descend laterally to the IVC. In patients with RCU, the ureter descends in a spiral path around the IVC. The proximal ureter passes posterior to the IVC at the L3 level to lie medially, while the distal ureter returns to its normal course, crossing from medial to lateral anterior to the lower IVC. It results from the abnormal persistence of the right subcardinal vein ventral to the ureter during IVC embryogenesis.[3] Even though it is a congenital defect, patients usually present in the third or fourth decade of life with a

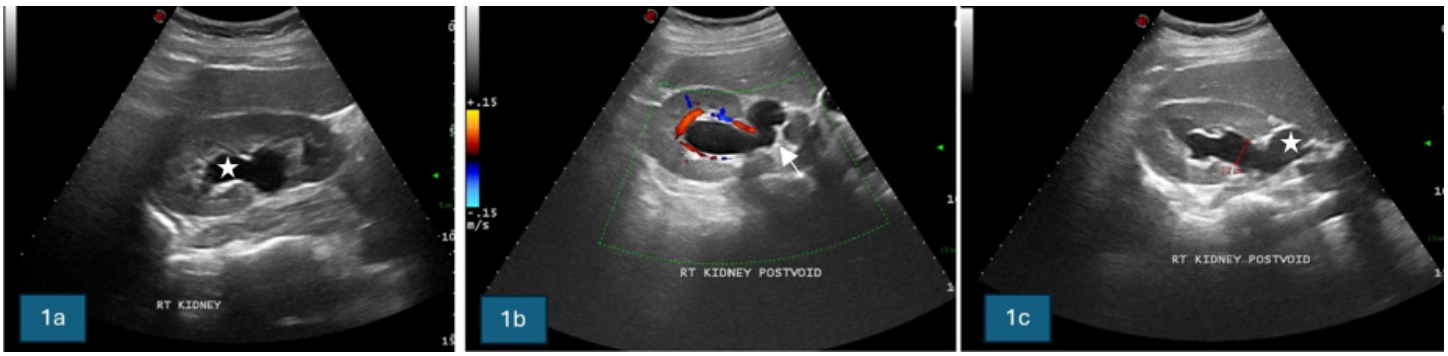


Image 1. Long-axis (**A**) and short-axis (**B,C**) ultrasound images of the right kidney demonstrating moderate hydronephrosis and proximal hydroureter (star). Image **B** reveals acute angulation at the ureteropelvic junction (arrow).

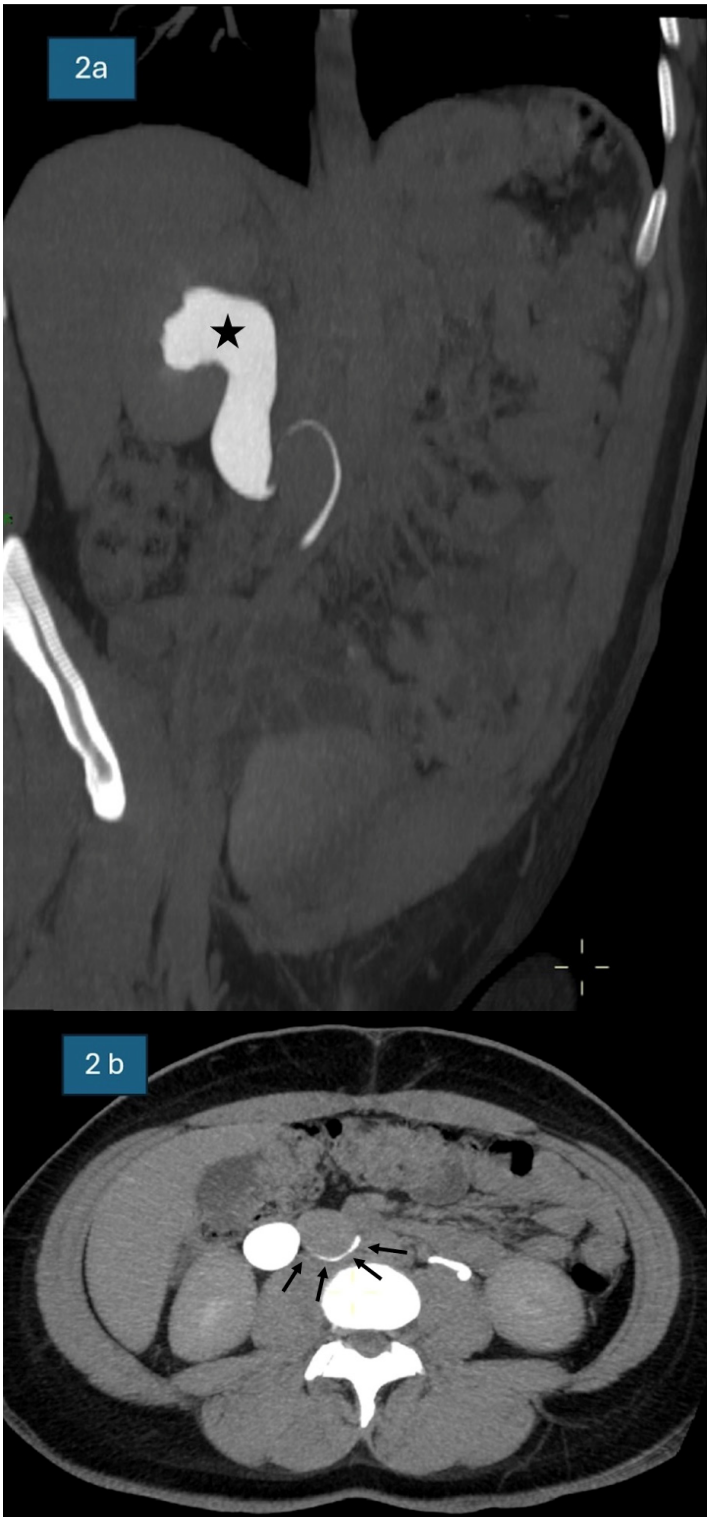


Image 2. Delayed contrast-enhanced computed tomography urogram with oblique multiplanar reformations. (**A**) Coronal image demonstrating moderate right hydronephrosis and proximal hydroureter (star) secondary to obstruction, with the proximal ureter following a retrocaval course medial and posterior to the inferior vena cava (IVC). (**B**) Axial image at the third and fourth lumbar level showing the right proximal ureter coursing posterior to the IVC, with focal narrowing behind the vein (arrows) consistent with a retrocaval ureter.

history of abdominal pain due to ureteric obstruction and associated hydronephrosis.[4] Prolonged ureteric obstruction can lead to obstructive uropathy, affecting renal function.[5] The clinical presentation of RCU is nonspecific, and its diagnosis relies primarily on radiological imaging, particularly contrast-enhanced CT.[6]

Hydronephrosis is often the first finding to prompt further investigation, but it is nonspecific and may lead to diagnostic uncertainty or misinterpretation.[4] Several imaging modalities can raise suspicion of an RCU. The hallmark feature commonly associated with RCU diagnosis is the fish-hook sign, also known as the reversed J-shaped ureter, which demonstrates a characteristic curvature of the proximal right ureter as it courses behind the IVC. This sign can be seen on intravenous pyelogram, retrograde pyelography, or CT urography.[5] The inability to confirm or exclude RCU on CT scan frequently results from inadequate opacification of the distal ureter. This limitation is usually due to obstruction or urinary stasis, which prevents clear visualization of the ureteral course, particularly in its distal segment. In such cases, placing a ureteral stent can improve diagnostic accuracy by opacifying the entire ureter, clarifying its course, and reducing unnecessary surgical interventions.[7]

In our case, the right proximal ureter is seen on CT coursing medial and posterior to the IVC at the L3–L4 (retrocaval course) level, with narrowing of the ureter as it passed behind the IVC. On ultrasound, RCU may be suggested when a transverse scan demonstrates a dilated ureter coursing posterior to the IVC. The use of color Doppler is valuable in this context because it helps differentiate the proximal ureter from adjacent vascular structures, specifically the right renal vein and the IVC.[6,8] Surgery is required in patients with persistent symptoms or worsening renal function. Repair usually includes the resection of the redundant RCU segment, interposition, and anastomosis.[7]

Abdominal POCUS can be used to identify various intraabdominal pathologies in patients presenting with abdominal pain, including hydronephrosis, as seen in our case.[8,9] The use of POCUS to identify hydronephrosis is one of the more commonly used applications. A meta-analysis of five studies (N = 1,773) comparing CT to hydronephrosis detected on POCUS showed a pooled sensitivity and specificity of 70.2% and 75.4%, respectively, with a calculated positive and negative likelihood ratio of 2.85 and 0.39.[10] While a negative test does not rule out the possible presence of hydronephrosis, a positive POCUS may aid in decision-making and further care of patients.

CONCLUSION

The early detection of hydronephrosis on POCUS triggered further imaging that identified a rare congenital anomaly, leading to timely surgical intervention. The use of POCUS in identifying hydronephrosis is reliable. While a negative result does not exclude hydronephrosis, a positive

finding may support clinical decision-making and guide further patient care. This case report identifies a rare entity that should be in the differential diagnosis for undifferentiated abdominal pain and for unexplained hydronephrosis.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Noah Marzook, FRCPC, University of Jeddah, Faculty of Medicine, Department of Pediatrics, Hamzah Ibn Al Qasim St, Al Sharafeyah, Jeddah 23218, Saudi Arabia. Email: nmarzook@uj.edu.sa.

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Inguinal Bladder Herniation in the Emergency Department: A Report of Two Cases

Bill Chan, MD
Skyler Sloane, BS
Colleen Campbell, MD
Binh Ly, MD
Rachna Subramony, MD

University of California, San Diego, Department of Emergency Medicine, San Diego, California

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Introduction: Inguinal bladder herniation is a rare subtype of inguinal hernia in which the urinary bladder protrudes into the inguinal canal. It is often asymptomatic but can present with a range of clinical severity in the emergency department (ED).

Case Report: We present a case report of two patients with inguinal bladder hernias. The first case describes a stable patient with bladder herniation without urinary obstruction, successfully managed with outpatient follow-up. The second case illustrates a more severe presentation with bladder involvement causing bilateral hydroureteronephrosis, requiring admission after failed reduction. This patient subsequently re-presented with urosepsis and developed multiple complications, including bacteremia and infective endocarditis.

Conclusion: Inguinal bladder herniation can present with a wide clinical spectrum in the ED. Early recognition, appropriate imaging, and thorough clinical assessment are critical in guiding management and preventing complications. [Clin Pract Cases Emerg Med. 2026;10(4):597–600.]

Keywords: *Inguinal bladder hernia; hydroureteronephrosis; urosepsis, emergency department, case report.*

INTRODUCTION

We discuss a case report of emergency department (ED) presentations of inguinal bladder hernias, an uncommon subtype of inguinal herniation in which a portion of the urinary bladder protrudes through the inguinal canal. Although inguinal hernias are common, bladder involvement occurs in fewer than 5% of cases, with incidence approaching 10% in older, obese men. Most cases are asymptomatic and discovered incidentally on computed tomography (CT) or intraoperatively. However, these benign presentations have the propensity to progress to significant obstructive uropathy and subsequent urosepsis requiring more aggressive management, making early recognition critical. These cases highlight the variable presentation and urgency of inguinal bladder herniation and emphasize the importance of appropriate escalation of care in the ED. Despite being described in surgical and urologic literature, inguinal bladder herniation remains underrepresented in emergency medicine, making recognition in the acute setting more challenging.

CASE REPORT

Case 1

A 72-year-old man with a history of right renal cell carcinoma status postnephrectomy, chronic hernia involving the right side of the bladder, prior hernia repair with mesh, and obesity presented from a skilled nursing facility with worsening abdominal and right flank pain. He stated that he was able to void but had to stand to do so due to the hernia's position. His vital signs were as follows: heart rate (HR), 87 beats per minute; blood pressure (BP) 126/73 mm Hg; respiratory rate

(RR), 18 breaths per minute; and temperature, 97.9 °F. Physical examination revealed a well-appearing patient with suprapubic tenderness without rebound or guarding, and a large nonreducible right inguinoscrotal hernia. Laboratory values revealed no leukocytosis or electrolyte abnormalities. Urinalysis did not show any evidence of infection. Computed tomography of the abdomen and pelvis with contrast demonstrated an inguinal hernia with a 3.9-cm neck containing a portion of the bladder as well as of the small bowel (a new development since the CT of the chronic hernia four months prior) consistent with interval progression of his chronic hernia.

Despite further protrusion of the bladder into the hernia sac, the patient was able to void in the ED, with documented 200-mL urine output. The subsequent postvoid residual measurement was 0 mL, as determined by bladder scan. The patient was provided analgesics and antiemetics in the ED and subsequently discharged back to the skilled nursing facility with anticipated outpatient follow-up with general surgery in three weeks. The patient completed his outpatient elective hernia repair surgery without complication.

CPC-EM Capsule

What do we already know about this clinical entity?
Inguinal bladder herniation is rare, often asymptomatic, and usually identified incidentally but can progress to significant obstructive uropathy.

What makes this presentation of disease reportable?
These two cases demonstrate the same pathology spanning stable outpatient herniation to obstruction, hydroureteronephrosis, and urosepsis.

What is the major learning point?
Inguinal bladder herniation has a broad clinical spectrum. Ultrasound and computed tomography (CT) findings of hydronephrosis or obstruction should prompt urgent evaluation.

How might this improve emergency medicine practice?
Greater awareness of this underrecognized entity may prompt earlier ultrasound/CT evaluation and closer attention to voiding symptoms, helping prevent missed obstruction.

Case 2

A 60-year-old man with no known past medical history presented with acute-onset lower abdominal and left testicular pain beginning the morning of presentation, associated with four episodes of nonbloody, nonbilious emesis. He reported a prior similar episode of inguinal pain and swelling that had self-resolved. He denied trauma, fever, chills, dysuria, hematuria, or genitourinary discharge but noted increasing difficulty with urination. Vital signs were as follows: HR, 62 beats per minute; BP, 149/92 mm Hg; RR, 13 breaths per minute; and temperature, 98 °F. Physical examination demonstrated a soft abdomen with suprapubic and left inguinal tenderness without peritoneal signs. The left inguinal region was markedly swollen with associated bilateral testicular swelling, without overlying erythema or skin changes. Laboratory studies were notable for leukocytosis to 13.4 × 10^9/L (reference range, 4.0–10.0 × 10^9/L); no significant electrolyte abnormalities; elevated lactate, 3.6 mmol/L (.5–2.0 mmol/L); and creatinine, 1.27 mg/dL (0.67–1.17 mg/dL).

Computed tomography abdomen and pelvis revealed a large left inguinal hernia containing a significant portion of the anterior bladder, with associated bladder wall thickening and surrounding inflammatory changes (Image 1). Extrinsic compression of the distal ureters had resulted in severe bilateral hydroureteronephrosis, as well as small volume ascites and findings suggestive of inflammatory enteritis (Image 2). Overall, these findings were concerning for clinically significant urinary obstruction secondary to bladder herniation. Throughout his ED course, the patient continued to endorse difficulty with voiding, with only minor dribbling occurring and no ability to create a stream.

Multiple attmpts by general surgery to manually reduce the hernia at bedside were unsuccessful despite manual pressure with Trendelenburg positioning and analgesia. A Foley catheter was successfully placed, yielding 800 mL of output, and the patient was admitted for further management. The urinalysis was negative for infection, and repeat lactate improved to 2.6 mmol/L. The patient remained hemodynamically stable and was discharged the next day after catheter removal, hernia reduction by catheter-induced bladder drainage and manual reduction, and a successful voiding trial.

Twelve days after his initial presentation, the patient returned to the ED with fevers, chills, and diarrhea for two days. He denied nausea, vomiting, respiratory symptoms, or sick contacts but experienced decreased urinary output without dysuria or pyuria. He had no pain associated with his groin or chronic left inguinal hernia. Vitals were notable for the following: HR, 124 beats per minute; BP, 140/93 mm Hg; RR, 16 breaths per minute; and temperature, 102.3 °F. Laboratory evaluation was notable for leukocytosis, 19.6 × 10^9/L (reference range, 4.0-10.0 x 10^9/L); hyponatremia,126 mmol/L (136–145 mmol/L); and elevated creatinine, 1.27 mg/dL, up from a baseline of 0.7 mg/dL. The initial lactate was 2.4 mmol/L. Urinalysis showed white blood cells, positive leukocyte esterase, and hematuria, concerning for urinary tract infection. The patient was then started on broad-spectrum antibiotics with



Image 1. Computed tomography demonstrating inguinal bladder herniation (arrow).

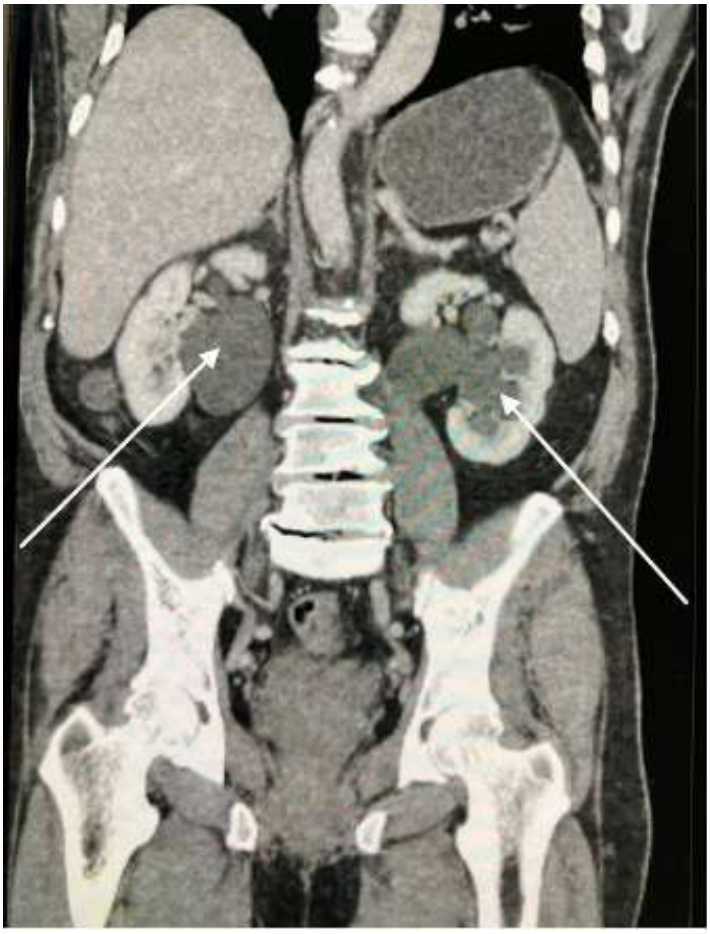


Image 2. Computed tomography showing bilateral severe hydronephrosis, as indicated by both arrows.

linezolid and piperacillin-tazobactam and fluid resuscitation.

Given his recent history of bladder-containing inguinal hernia with associated ureteral compression, the clinical picture was concerning for sepsis with a likely urinary source, possibly related to impaired urinary drainage and prior hydroureteronephrosis. Point-of-care ultrasound (POCUS) of the kidneys, bladder, and testicles revealed severe hydronephrosis secondary to the inguinal bladder herniation and persistent herniation of the bladder into the inguinal canal (Image 3). The general surgery service was consulted and confirmed that the hernia remained reducible without indication for acute operative intervention during this presentation. The urology serice recommended Foley catheter placement, which yielded approximately 500 mL of urine output. The patient was admitted to medicine for further management of suspected urosepsis and close monitoring. This progression suggests that even reducible hernias may predispose patients to delayed infectious complications.

The patient's hospital course was complicated by sepsis with *Staphylococcus epidermidis* both in urine and blood cultures, with a resultant small aortic valve vegetation consistent with infective endocarditis. Despite these complications, the patient improved clinically with antibiotics and supportive care. The leukocytosis and fever resolved, and renal function normalized. He was discharged after a 16-day admission with a Foley catheter in place with close outpatient follow-up, awaiting resolution of endocarditis before proceeding with surgical repair of the hernia.

DISCUSSION

Inguinal bladder hernias, while rare, are important to consider in patients presenting with flank pain and lower urinary tract symptoms, particularly in the setting of known

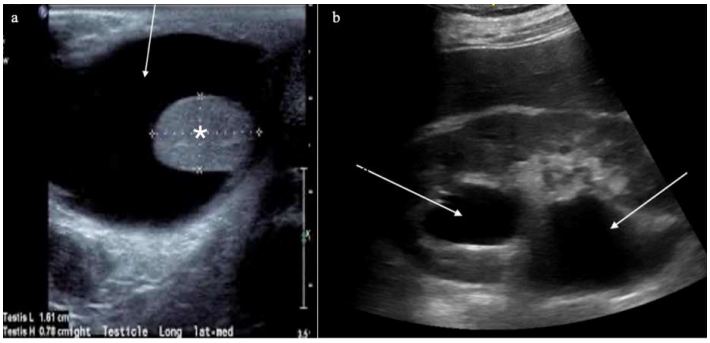


Image 3. ***A,*** Ultrasound of bladder herniation into the inguinal canal seen as the anechoic space behind the testicle (the testicle is annotated by the asterisk). The bladder is annotated by the arrow. **B,** Ultrasound of the right kidney demonstrating severe hydronephrosis (arrows).

inguinal hernias. Although many cases are asymptomatic and discovered incidentally, symptomatic presentations may include flank pain, two-stage voiding, urinary retention, and hematuria. When hydronephrosis is encountered on POCUS in a patient with inguinal hernia, further investigation is warranted to determine bladder involvement in herniation, and laboratory evaluation should be performed to assess renal function. More extensive involvement can result in complete bladder herniation, or scrotal cystocele, which is associated with complications such as obstructive uropathy, bowel involvement, and rarely, bladder ischemia.[1]

This case report of two cases highlights the broad clinical spectrum of inguinal bladder herniation. The first case demonstrates a stable presentation without urinary obstruction or infection, allowing for safe outpatient management. By contrast, the second case illustrates a more severe presentation with bladder involvement causing ureteral compression and bilateral hydroureteronephrosis, requiring hospital admission after failed bedside reduction. Together, these cases emphasize that similar anatomical pathology may present with markedly different clinical severity.

Computed tomography remains the diagnostic modality of choice, enabling identification of bladder involvement and associated complications such as hydronephrosis or bowel herniation. Point-of-care ultrasound serves as a valuable adjunct, allowing rapid bedside assessment of bladder volume, urinary retention, and hydronephrosis, and may suggest bladder displacement into the inguinal canal. Point-of-care ultrasound is also useful for identification of bowel obstruction within hernia contents, which we did not see in these cases. When findings on ultrasound are consistent with bladder obstruction, rapid treatment with Foley catheter drainage of the bladder should be considered. Integration of imaging with clinical assessment—including voiding ability and signs of infection or obstruction—guides management and disposition. While stable patients may be managed in the outpatient setting, those with obstruction, infection, or failed reduction require prompt surgical consultation. Early recognition of this condition can directly influence disposition and help prevent downstream complications.

CONCLUSION

Inguinal bladder herniation is a rare but important diagnosis in the ED, with presentations ranging from incidental findings to obstructive uropathy and sepsis. These cases highlight the importance of integrating imaging findings with clinical assessment, particularly in the case of evaluating urinary function and signs of infection or obstruction. Early recognition and appropriate disposition can prevent significant morbidity associated with delayed diagnosis.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Rachna Subramony, MD, University of California, San Diego, Department of Emergency Medicine, 200 West Arbor Drive, San Diego, CA 92103. Email: rsubramony@health.ucsd.edu.

Conflicts of Interest: By the *CPC-EM* article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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Delayed Revascularization in Rural Acute STEMI Patient on Direct Oral Anticoagulation: A Case Report

Seth Roetman, BS* *Idaho College of Osteopathic Medicine, Meridian, Idaho
Gary Brandecker, MD† †Idaho College of Osteopathic Medicine, Department of Emergency Medicine, Meridian, Idaho

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Introduction: Acute ST-elevated myocardial infarction (STEMI) is a medical emergency requiring timely revascularization to preserve myocardial injury. Rural populations often experience delays in treatment leading to increased risk of complications and mortality. Emergent treatment of STEMI in a patient on long-term direct oral anticoagulation facing delays to revascularization is a complex scenario for rural physicians.

Case Report: A 55-year-old male presented to the emergency department in rural Alaska and was diagnosed with acute inferior STEMI. The patient's history was notable for pulmonary embolism with long-term direct oral anticoagulation. Thrombolytic therapy was not recommended and prompt revascularization via percutaneous coronary intervention (PCI) was needed; however, the nearest PCI-capable hospital was hundreds of miles away, increasing the risk of ischemic cardiomyopathy and death. The patient achieved revascularization after transport and, in the following year, experienced multiple episodes of ventricular tachycardia leading to ablation and need for implantable cardioverter-defibrillator placement.

Conclusion: This case illustrates a complex but not uncommon scenario that rural physicians face. In rural patients requiring acute STEMI treatment but without timely access to a PCI-capable hospital, systemic thrombolysis and immediate transfer of care is often the only option for revascularization. There is a need for clear STEMI management guidelines in anticoagulated patients who present in rural settings. [Clin Pract Cases Emerg Med. 2026;10(4):601–604.]

Keywords: *ST-elevated myocardial infarction; rural; direct oral anticoagulation; percutaneous coronary intervention; case report.*

INTRODUCTION

Cardiovascular disease remains the most common cause of death in the United States and is expected to increase nationwide through 2050.[1] Cardiovascular mortality is higher in rural areas compared to urban settings, with this disparity increasing after the coronavirus disease 2019 pandemic.[2] In the case of ST-elevated myocardial infarction (STEMI), reperfusion therapy is recommended within 60 to 90 minutes of arrival at the hospital.[3] Reperfusion is effective up to, and possibly beyond, 12 hours; however, necrosis and cell death of the myocardium increases with delays beyond the recommended 90 minutes, leading to an increased risk of life-threatening complications.[4,5]

The two methods of achieving reperfusion (or revascularization) are systemic thrombolysis or percutaneous coronary intervention (PCI). Percutaneous coronary intervention provides better outcomes and is the gold-standard treatment for acute STEMI.[6] In rural patients requiring acute STEMI treatment but without timely access to a PCI-capable hospital, thrombolytics and immediate transfer is the next best option. The use of systemic thrombolysis in anticoagulated patients, however, carries a significant risk of bleeding and

intracranial hemorrhage.[3,7] In this case, we consider a man on long-term anticoagulation presenting with inferior STEMI in a rural area.

CASE REPORT

A 55-year-old man with a history of persistent lower extremity deep vein thrombosis, pulmonary embolism, and uncontrolled hypertension presented to his local emergency department (ED) via ambulance with severe sternal chest pain, diaphoresis, and mild nausea. The patient's chest pain began at approximately 7:45 AM while exercising on a stationary bicycle. As the pain worsened he was brought to the hospital by emergency medical services.

Prehospital electrocardiography (ECG) demonstrated STEMI, which was confirmed on ECG obtained in the ED (Image 1). The patient was compliant with his rivaroxaban medication, which he was taking for a venous thromboembolism diagnosed three years prior.

Physical examination revealed the following vital signs: blood pressure, 147/100 mm Hg; heart rate, 47 beats per minute; and temperature, 98.2 ºF. Cardiac examination was notable for bradycardia, which was his baseline, with regular rhythm and normal first and second heart sounds present. The patient appeared diaphoretic and was clutching his chest with severe, sternal chest pain without radiation. In collaboration with the on-call cardiologist at the nearest PCI-capable hospital, it was decided that immediate transport was required. Due to the patient's long-term oral anticoagulation, thrombolytic therapy was not recommended; thus, PCI was necessary for revascularization. An initial estimated time of arrival of 4.5 hours for medevac to the cardiac catheterization lab was given at 8:53 AM (expected arrival at 1:23 PM).

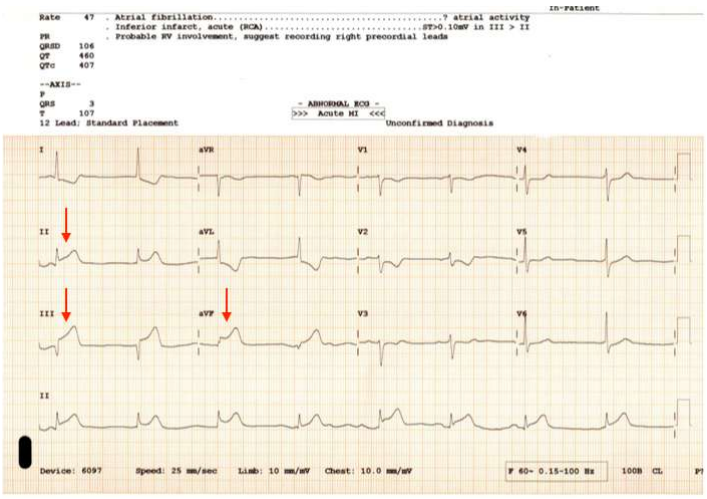


Image 1. Electrocardiogram recorded in the local emergency department at 8:35 AM demonstrating ST-elevation in leads II, III, and aVF, classic for an inferior myocardial infarction (arrows).

CPC-EM Capsule

What do we already know about this clinical entity?

Definitive treatment of ST-elevated myocardial infarction (STEMI) is percutaneous coronary intervention. When unavailable, systemic thrombolysis is the alternative, though it is often contraindicated.

What makes this presentation of disease reportable?

This case is notable for a rare combination of acute STEMI, lack of timely percutaneous coronary intervention access, transport delays, and ongoing anticoagulation complicating revascularization.

What is the major learning point?

Complex clinical decision-making is required in acute STEMI when transport is delayed and the patient is anticoagulated.

How might this improve emergency medicine practice?

In treating acute STEMI, "time is tissue." Understanding potential barriers to management can improve patient outcomes.

Patient management prior to transport included aspirin, acetaminophen, clopidogrel (600 mg), fentanyl, intravenous (IV) heparin (25,000 U), nitroglycerin, and intravenous fluids. The patient remained in the local hospital until transfer to medevac at 11:21 AM and arrived at the PCI-capable hospital at approximately 2 PM. More than six hours elapsed between arrival at the local ED and entry into the catheterization laboratory. The patient underwent a successful PCI and remained hospitalized for four days.

Follow-up over the next 18 months revealed an ejection fraction of 49.5% (50–70%), multiple near-syncopal episodes, and ventricular tachycardia (VT) during stress testing (Image 2). In a subsequent procedure, VT was easily induced in an area of inferoseptal scar resulting from the STEMI and ablated. As of this writing, the patient iwas scheduled to receive an implantable cardioverter-defibrillator.

DISCUSSION

Rural living is increasingly attractive to many for a variety of reasons. However, it does not come without risks. Rural populations experience higher rates of cardiovascular disease and increased mortality.[2] Rural communities face many barriers to healthcare including

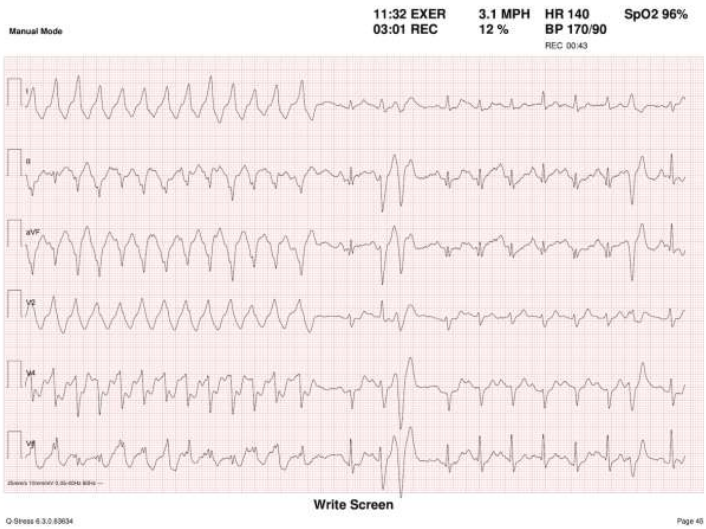


Image 2. Stress electrocardiogram taken while incline walking on treadmill revealing ventricular tachycardia across multiple leads.

distance, terrain, and weather conditions that may make receiving preventive and acute care challenging.[8,9] This frequently results in prolonged transport times during medical emergencies. This is especially worrisome in acute coronary syndromes, such as STEMI, because time to reperfusion is a significant determinant of patient outcomes.[3,10] According to the American Heart Association (AHA), reperfusion therapy is recommended within 60 to 90 minutes of arrival at the hospital.[3] Reperfusion is effective up to, and possibly beyond, 12 hours; however, cell death and necrosis of the myocardium increases with delays beyond the AHA-recommended time and leads to an increased risk of cardiovascular complications.[3,4,5] Percutaneous coronary intervention is the gold standard treatment, leading to decreases in mortality and reinfarction compared to thrombolytics, the only other method of revascularization.[6,11] However, many states have large rural populations without timely access to a PCI-capable hospital. This is true for over 40% of the population in 14 states including Alaska, Idaho, Iowa, Mississippi, New Mexico, and Oklahoma.[12] Although the number of PCI-capable hospitals has recently increased, access among the US population has increased only marginally.[12]

When PCI is not immediately available, thrombolytic therapy is typically recommended.[3] For rural patients requiring acute STEMI treatment, thrombolytics and immediate transfer of care to a PCI-capable hospital may be the only option. Thrombolytics are lifesaving, reducing mortality up to 24 hours from onset of symptoms.[3] However, there are many contraindications to thrombolytics including bleeding disorders, recent head trauma, advanced liver disease, and a host of other relative contraindications.[3] Current anticoagulant use is a relative contraindication according to the AHA guidelines for acute STEMI care.[3] Previous evidence has shown that long-term oral anticoagulation with concurrent use of thrombolytics leads to a significantly higher risk of bleeding and intracranial hemorrhage.[3,7] Recently, support has increased for concurrent use of direct oral anticoagulants and thrombolytics in other acute ischemic conditions.[13,14] Based on the current recommendation, however, thrombolytic use is not an option in many cases.

Anticoagulant use is common and increasing. Oral anticoagulant use increased significantly from 2011 to 2019, nearly doubling from under three million Medicare beneficiaries to over five million.[15] It is reasonable to assume these numbers have continued to increase in the last seven years as well; however, this does not account for those not covered by Medicare who require anticoagulation use.

CONCLUSION

This case presents a unique but not uncommon scenario of a patient with an acute STEMI in a rural area facing delays to transport. The case was complicated further by the patient's history of long-term direct oral anticoagulation. Acute STEMI is a medical emergency requiring immediate revascularization, typically achieved with PCI or thrombolytic therapy. Rural patients often lack timely access to PCI-capable hospitals and may be ineligible for thrombolytic therapy. This leaves the patient and physician in a complex and difficult situation. Proper management is imperative to minimize the risk of complications and death due to prolonged cardiac ischemia. Potentially broadening the use of thrombolytics in cases of delayed revascularization may be worth exploring. This case illustrates the need for clear management guidelines in acute STEMI care in a patient receiving long-term anticoagulation who faces substantial delays in transport.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Seth Roetman, BS, Idaho College of Osteopathic Medicine, 1401 E Central Dr, Meridian, ID, 83642. Email: sroetman@s.icom.edu.

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Spontaneous Cardiac Tamponade in a Patient with Severe Hemophilia A: A Case Report

Teenu Singh, MD, DM
Sachin Wali, MD, DNB, DM
Shreyas Gutte, MD, DM
Afzal Azim, MD, PDCC
Banani Poddar, MD, DNB

Sanjay Gandhi Postgraduate Institute of Medical Sciences, Department of Critical Care Medicine, Lucknow, India

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Introduction: Hemophilia A is an X-linked recessive bleeding disorder characterized by deficiency of factor VIII. The clinical features of this disease are hemorrhage and can be life-threatening when major organ systems are involved.

Case Report: We report the case of a 13-year-old boy with severe hemophilia A and factor VIII inhibitors who presented with fever and septic arthritis, later developing refractory shock with tachyarrhythmia. Despite initial suspicion of sepsis-related myocarditis, point-of-care ultrasound (POCUS) revealed massive hemorrhagic pericardial effusion with tamponade physiology. Recombinant factor VIIa was transfused as hemostatic rescue therapy; the patient was subsequently started on fresh frozen plasma and emicizumab. Ultrasound-guided pericardiocentesis resulted in rapid hemodynamic recovery.

Conclusion: This case highlights the importance of considering obstructive shock in unstable hemophilia patients and demonstrates that timely POCUS-based diagnosis and factor-supported pericardiocentesis can be lifesaving. [Clin Pract Cases Emerg Med. 2026;10(4):605–608.]

Keywords: *hemophilia A; cardiac tamponade; factor VIII deficiency; case report.*

INTRODUCTION

Hemophilia A is an X-linked recessive bleeding disorder caused by a quantitative or qualitative deficiency of factor VIII (FVIII) resulting from heterogeneous mutations in the FVIII gene located on chromosome Xq28.[1] The disease is characterized by spontaneous or trauma-related bleeding, with clinical manifestations largely determined by the severity of the deficiency. Bleeding most commonly involves joints (hemarthroses) and muscles (hematomas), although life-threatening hemorrhage can occur when critical organs such as the central nervous system or cardiovascular system are affected.

Hemorrhagic pericardial effusion leading to cardiac tamponade is a rare but potentially fatal complication of hemophilia. Early diagnosis and regular FVIII replacement therapy may help prevent such bleeding events. However, management becomes particularly challenging in patients who develop FVIII inhibitor antibodies because conventional high-dose FVIII replacement may be ineffective. Additional challenges include the bleeding risk associated with invasive procedures such as pericardiocentesis and the lack of standardized management guidelines for hemorrhagic pericardial effusion in patients with hemophilia and other bleeding disorders.[2,3]

Furthermore, hemophilia-related hemorrhagic pericardial effusion is exceedingly uncommon; a PubMed search identified only seven previously reported cases over the past several decades. We report a case of severe hemophilia A and concurrent FVIII inhibitor antibodies presenting with hemorrhagic pericardial effusion causing cardiac tamponade.

This case highlights the diagnostic and therapeutic challenges associated with this rare but life-threatening complication.

CASE REPORT

A 13-year-old boy diagnosed with hemophilia A and low-level FVIII inhibitors, regularly receiving FVIII replacement, presented to the hospital with a history of fever and swelling in the right knee with associated pain for four days and was admitted to the medical genetics ward (day 1). There was no history of trauma. On examination, vitals were as follows: heart rate (HR), 96 beats per minute; blood pressure (BP), 110/70 mm Hg; respiratory rate, 20 breaths per minute; and peripheral oxygen saturation (SpO_2), 99% on room air. His right knee was swollen with tenderness over the anterior aspect, with localized warmth and generalized restriction in range of movement. Dorsalis pedis artery and posterior tibial artery pulses of his right lower extremity were present. Laboratory investigations showed the following: hemoglobin, 7.4 g/dL (reference range, 11–14 g/dl); total leukocyte count (TLC), 17 × 10^9/L (4–10 × 10^9/L); platelets, 148 × 10^9/L (150–450 × 10^9/L); activated partial thromboplastin time (aPTT), 52 seconds (24–39 seconds); and international normalized ratio (INR), 1.25 (0.9–1.3). Ultrasonography of his right knee showed a fluid collection in the anterior aspect, which was hypoechoic with internal echoes suggestive of knee abscess.

The patient underwent incision and drainage in the operating room, and was managed along with empiric antibiotics, FVIII concentrate and monoclonal antibody emicizumab (day 5). There was reduction in pain and swelling, but intermittent fever for four hours a day up to 99.5 °F persisted; TLC count increased to 20 × 10^9/L, and antibiotics were modified as per culture reports (day 12).

On subsequent days, patient's fever intensified and became associated with dyspnea (day 15). On examination, his vitals were as follows: HR, 198 beats per minute; BP, 80/50 mm Hg; respiratory rate, 40 breaths per minute; and SpO2, 98% on room air. Electrocardiogram (ECG) was suggestive of narrow complex tachycardia (supraventricular tachyarrhythmia). The patient was started on oxygen supplementation, and two doses of intravenous (IV) adenosine were given over 3 seconds (total of 18 mg) with no reduction in HR. Following this, IV amiodarone (150 mg) followed by 1mg/kg/hr continuous infusion was started. He was also initiated on IV noradrenaline (norepinephrine) infusion at a rate of 0.6 µg/kg/min and noninvasive positive pressure ventilation. Possibility of myocarditis secondary to sepsis was considered, and he was transferred to intensive care unit-level of care. On examination, he had thready pulses with pulsus paradoxus, muffled heart sounds, an ECG showing electrical alternans (Image), and severe metabolic acidosis on arterial blood gas.

Suspecting obstructive shock, we performed point-of-care ultrasound (POCUS), which demonstrated a massive pericardial effusion (3.5 cm) between the visceral and parietal pericardium at end-diastole, with right ventricular diastolic collapse in the subcostal four-chamber view (Video). IIn consultation with a hematologist and a cardiologist, the decision was made to relieve the tamponade with pericardiocentesis. Laboratory results during this time showed the following: hemoglobin, 9.5 g/dL; TLC, 20.9 × 10^9/L; platelets, 10.9 × 10^9/L, INR, 4.0; and aPTT, 61 seconds. Liver function test showed the following: total bilirubin, 2.84 mg/dL (reference range, 0.3–1.3 mg/dl); direct bilirubin, 1.51 mg/dL (< 0.3 mg/dl); alanine aminotransferase, 34 u/L (5–55 u/L);

CPC-EM Capsule

What do we already know about this clinical entity?
Hemophilia A can rarely cause spontaneous hemopericardium and cardiac tamponade, a life-threatening complication requiring urgent recognition and treatment.

What makes this presentation of disease reportable?
Spontaneous hemopericardium caused tamponade in a child with severe hemophilia A, FVIII inhibitors, and sepsis-associated coagulopathy.

What is the major learning point?
Consider cardiac tamponade in unexplained shock in hemophilia; bedside point-of-care ultrasound (POCUS) enables rapid diagnosis, while hemostatic support permits lifesaving drainage.

How might this improve emergency medicine practice?
POCUS should be used early in unexplained shock in hemophilia, enabling rapid recognition of tamponade and timely, factor-supported pericardiocentesis.



Image. Patient monitor **A**) electrocardiogram strip showing electrical alternans, and **B**) arterial blood pressure waveform showing pulsus paradoxus.

aspartate aminotransferase, 225 u/L (15–60 u/L); and alkaline phosphatase, 170 u/L (142–335 u/L). The patient was transfused 250 µg/kg of recombinant FVII activated (Factor FVIIa), and subsequently started on 90 µg emicizumab and 15 ml/kg of fresh frozen plasma intravenously. After 30 minutes, ultrasound-guided percutaneous drainage of the tamponade through a subxiphoid approach was done, and a flexible 8-French catheter was left in the pericardial space as a pericardial drain.

An initial 300 mL of fluid was drained from the effusion, which was hemorrhagic in nature, suggestive of hemopericardium due to hemophilia A. There was improvement in hemodynamic parameters and signs of tamponade resolved on repeat ultrasound. Pericardial fluid sent for microbiological examination was negative for infectious etiology. On the first day, a total of 900 mL hemorrhagic fluid drained, with subsequent days decreasing in volume consistently (second day 250 mL, third day 180 mL, fourth day 30 mL). The catheter was removed on the seventh day following the pericardiocentesis. During this period the patient received seven units of packed red blood cells, seven units fresh frozen plasma, and six pooled cryoprecipitate units. The patient continued to be managed with FVIII (1,000 U twice a day) and emicizumab (90 µg daily). Repeat echocardiography showed no pericardial effusion, and the patient was transferred to ward on day 28.

DISCUSSION

This case illustrates a unique clinical scenario involving severe hemophilia A with low-titer FVIII inhibitors complicated by sepsis-associated coagulopathy and hemorrhagic pericardial effusion causing cardiac tamponade. The diagnosis was particularly challenging because refractory shock and tachyarrhythmia initially raised suspicion of myocarditis, while the underlying tamponade physiology was revealed by POCUS. The coexistence of FVIII inhibitors and sepsis-related coagulopathy substantially increased the risk of bleeding and complicated hemostatic management. Despite these challenges, timely diagnosis, hemostatic optimization and ultrasound-guided pericardiocentesis resulted in rapid hemodynamic recovery. This case highlights the importance of maintaining a high index of suspicion for cardiac tamponade in critically ill patients with hemophilia and demonstrates that factor-supported pericardiocentesis can be performed safely when appropriate hemostatic measures are instituted.

Hemorrhagic pericardial effusion involves collection of bloody fluid within the pericardial cavity and can evolve into cardiac tamponade. Common etiologies include thoracic trauma, complications of ischemic heart disease, infection, autoimmune conditions, and anticoagulants. Of the reported hemophilia cases with hemorrhagic pericardial effusion, there was a preceding illness such as rheumatic fever, viral fever, pr chest trauma that could have precipitated hemorrhagic pericardia effusion.[3-5] In our case, hemophilia A with FVIII inhibitors was the primary predisposing factor, whereas the coagulopathy secondary to sepsis and mild liver dysfunction could have exacerbated the bleeding. Hence, this underscores the importance of early source control in sepsis and prompt treatment of coagulopathy in patients with hemophilia A.

Cardiac tamponade results from accumulation of pericardial fluid causing impaired cardiac filling and reduced cardiac output.[6] In our patient, dyspnea, tachyarrhythmia, and refractory shock were initially attributed to sepsis and possible myocarditis, delaying consideration of an obstructive cause of shock. The diagnosis of tamponade is often challenging because classic findings such as Beck triad are frequently absent.[2,7] Also in critically ill patients, overlapping shock states may further obscure the diagnosis. Therefore, unexplained hemodynamic deterioration, escalating vasopressor requirements, or shock disproportionate to the apparent severity of sepsis should prompt evaluation for cardiac tamponade. In such situations, POCUS is invaluable because it can rapidly identify pericardial effusion and tamponade physiology at the bedside, allowing timely diagnosis and intervention. In this case, POCUS was instrumental in distinguishing obstructive shock from presumed septic shock and directly guided definitive management.

Primary management of cardiac tamponade is by drainage of the pericardial effusion; even a small amount of fluid drainage can help relieve intrapericardial pressure and tamponade effects. Although several relative contraindications exist, a retrospective review of 1,196 patients who underwent drainage by a cardiologist in the presence of thrombocytopenia (platelet count $< 50 \times 10^9$/L) and coagulopathy (INR > 1.5), did not show significant association with increased major bleeding.[7] Goz et al reported a case of an 11-year-old boy in which 1,000 U of factor VIII was given and pericardial window was created through a mini thoracotomy in the left fourth intercoastal space with drainage of 200 cc of fluid. There was no bleeding seen in cardiac structure or main vessels, and he was discharged on the fifth postoperative day.[8]

In our case, prior to pericardiocentesis, FVIIa and fresh frozen plasma were given for treatment of coagulopathy. The International Society on Thrombosis and Haemostasis clinical practical guidelines suggest that individuals with severe hemophilia A with FVIII inhibitors who are undergoing invasive procedures should be treated with bypassing agents, either FVIIa or activated prothrombin complex concentrate.[9] In the present case, given that emicizumab prophylaxis was ongoing, FVIIa was preferred because a combination of activated prothrombin complex concentrate and emicizumab could lead to thrombotic complications.[9] Importantly, in hemodynamically unstable patients with tamponade physiology, hemostatic optimization and procedural planning should occur simultaneously, and lifesaving pericardial drainage should not be delayed once hemostatic measures have been initiated.

Hemorrhage in patients with hemophilia can be effectively prevented with regular factor replacement therapy that

maintains adequate physiologic FVIII levels. In the case reported by Zayani et al, the FVIII dose was increased to 50 IU/kg every eight hours during an episode of hemorrhagic pericardial effusion. In contrast, in the 11-year-old male reported by Goz et al, bleeding was successfully managed with a lower-intensity regimen of 50 IU/kg every 12 hours.[6,8] The goal of prophylaxis in hemophilia A is to prevent spontaneous bleeding. Prophylactic dose usually ranges from 10–40 IU/kg three times weekly, which is achieved by maintaining trough FVIII levels around 1–3%, with higher target intensities ≥ 3–5% individualized for patients with higher bleeding severity. In patients with FVIII inhibitors, emicizumab provides an effective alternative with reduced treatment burden given its weekly schedule.[9,10] However, there are no standardized guidelines regarding optimal factor replacement during hemorrhagic pericardial effusion, and management should be individualized based on clinical severity and response to therapy. [9]

A practical approach to hemorrhagic cardiac tamponade in patients with hemophilia and FVIII inhibitors involves four key steps: 1) prompt recognition of tamponade should be considered in patients with hemodynamic instability by confirmation through POCUS; 2) hemostatic rescue should be initiated immediately using appropriate bypassing agents such as recombinant FVIIa or activated prothrombin complex concentrate and correction of associated coagulopathy with blood products should be undertaken as clinically indicated; 3) urgent pericardiocentesis should be performed once initial hemostatic support has been established; and 4) continued postprocedural hemostatic support and serial echocardiographic surveillance are essential to prevent recurrent bleeding and re-accumulation of pericardial fluid.

A limitation of this report is the inability to definitively determine the exact precipitating mechanism of the hemorrhagic pericardial effusion. Although severe hemophilia A with FVIII inhibitors was likely the primary contributor, concurrent sepsis-associated coagulopathy and mild hepatic dysfunction may have exacerbated the bleeding tendency and contributed to the development of hemorrhagic cardiac tamponade.

CONCLUSION

Acute hemorrhagic pericardial effusion and resulting cardiac tamponade occurring spontaneously in patients with hemophilia is a rare condition and no consensus exists for optimal management. This case gives insight regarding factor replacement in the presence of FVIII inhibitors and sepsis-induced coagulopathy with a consideration for need of urgent pericardiocentesis.

Video. Point-of-care ultrasound (subcostal four-chamber view) demonstrating a large circumferential pericardial effusion. Yellow arrow indicates the pericardial effusion, while a red arrow highlights diastolic collapse of the right ventricle, consistent with cardiac tamponade physiology.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Banani Poddar, MD, DNB, Dept of Critical Care Medicine, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Raebareli road, Lucknow -226014, India. Email: bananip@hotmail.com.

Conflicts of Interest: By the *CPC-EM* article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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Parainfectious Urticarial Rash in a Young Female with Epstein-Barr Virus Infection: A Case Report

Huang XinYao Oliver, MBBS* *Changi General Hospital, Department of Emergency Medicine, Singapore
Andrea Yap* †National University of Singapore, Yong Loo Lin School of Medicine, Singapore
Dawn Toh*
Lim Hoon Chin Steven, MBBS†

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Introduction: Urticarial rash is a common condition but can present atypically, making it difficult to clinically diagnose. Thorough assessment with awareness and exclusion of differentials is crucial for definitive treatment.

Case Report: This is a case of a 23-year-old woman who presented with signs and symptoms suggestive of streptococcal pharyngitis and was treated with clarithromycin by primary care. Two weeks later, the patient was referred to the emergency department for malaise and an acute, rapidly progressing pruritic rash. Further evaluation revealed an Epstein-Barr virus (EBV) infection.

Conclusion: This case highlights the differential diagnoses and diagnostic reasoning required for a patient with a rash and concurrent respiratory symptoms. Differential considerations include viral exanthem, delayed drug-induced reaction, EBV-associated antibiotic rash, scarlet fever, and urticarial vasculitis. Early identification of atypical urticarial lesions and the exclusion of clinical mimics are vital to guide appropriate therapy and optimize patient outcomes. [Clin Pract Cases Emerg Med. 2026;10(4):609–613.]

Keywords: *atypical urticaria; Epstein-Barr virus; urticarial vasculitis; case report.*

INTRODUCTION

Urticaria is a common dermatological presentation characterized by transient, pruritic wheals that typically resolve within 24 hours and follow a benign, self-limiting course. It arises from a wide range of triggers, including infections, medications, and immune-mediated processes. Given the varied etiologies of acute eruptions, refining the differential diagnosis can be challenging, particularly when the presentation includes atypical features. We report the case of a young woman who developed an urticarial eruption in the setting of Epstein-Barr virus (EBV) infection, characterized by purpuric, prolonged lesions and prominent systemic symptoms. This case illustrates the diagnostic reasoning required to distinguish atypical urticaria from its differential diagnoses and highlights the systematic exclusion process necessary to optimize patient outcomes.

CASE REPORT

A 23-year-old immunocompetent woman presented to the emergency department with prostration and a two-day history of rapidly progressing pruritic rash that started on her proximal thighs before becoming generalized, with the greatest distribution over her limbs and back.

She had a recent two-week history of fever, cough, runny nose, and sore throat that was treated by her family physician with a five-day course of clarithromycin (500 mg twice daily) due to presumed streptococcal pharyngitis (no specific testing). It was chosen due to her known allergy to amoxicillin-clavulanate potassium. The antibiotic course was completed three days before onset of her rash. She had also used two traditional Chinese medicines: watermelon powder for her oral ulcer three days prior to onset of rash; and pearl powder for sore throat for three days, which was discontinued on the same day that the rash appeared.

There was no history suggesting autoimmune disease, including recurrent ulcers, sicca symptoms, Raynaud. phenomenon or joint pain, and no family history of autoimmune disorders. There were no clinical features indicating rheumatic heart disease or glomerulonephritis as complications of scarlet fever. Previously, an allergic reaction to amoxicillin-clavulanate potassium resulted in an urticarial rash on both lower extremities.

On physical examination, there were erythematous urticated blanchable plaques over the inferior chin, thighs, shin, calves, and a few scattered on the abdomen, back, arms, hands, and soles. Purpuric plaques were noted on her calves (Image 1).

Oral cavity examination revealed bilateral erythematous tonsils without strawberry tongue or cervical lymphadenopathy. A small lower inner lip mucosal ulcer was noted. There were no target lesions, pustules, atypical targets, or mucocutaneous findings to suggest erythema multiforme, Stevens-Johnson syndrome, or other severe drug eruptions. Cardiovascular, renal, and joint examinations were unremarkable. Due to significant malaise, generalized weakness, and the purpuric feature of the rash, the patient was admitted for treatment and workup of systemic and autoimmune disorders.

Laboratory results revealed the following: microcytic hypochromic anemia with hemoglobin 9.9 g/dL (reference range, 12-16 g/dL); ferritin, 177 µg/L (7.6-179 µg/L), with further testing revealing alpha thalassemia; white blood cell count, 3.7 × 10^9/L) (4×10^9 -10×10^9/L); C-reactive protein, 11 mg/L (0.2-9.1 mg/L); and erythrocyte sedimentation rate, 52 mm/h (3-15 mm/h). An autoimmune panel was negative for antinuclear antibodies, anti–double-stranded DNA antibodies, and extractable nuclear antigens; complement levels were normal. An infectious panel was positive for EBV by polymerase chain reaction but negative for antistreptolysin O, herpes simplex virus, and dengue serologies.

CPC-EM Capsule

What do we already know about this clinical entity?
Urticaria is a common but usually self-limiting condition that arises from triggers such as infections, medications, and immune-mediated processes.

What makes this presentation of disease reportable?
Urticarial eruption occurred in a patient with Epstein-Barr virus infection who had atypical features of purpura, prolonged lesions, and prominent systemic symptoms.

What is the major learning point?
Urticaria with red flags, such as duration >24 hours, purpura and systemic symptoms, warrant a thorough workup.

How might this improve emergency medicine practice?
A structured differential for atypical urticaria helps emergency physicians exclude dangerous conditions, guiding workup and appropriate symptomatic care.



Image 1. A) Dorsum of hand. B) An urticarial and purpuric eruption on lower extremities in a patient with Epstein-Barr virus infection.

The overall clinical presentation was consistent with a parainfectious urticarial eruption in the setting of EBV infection. The patient was treated twice daily with oral fexofenadine (180 mg), mometasone cream, and aqueous cream. Following two days of observation, her lesions improved, and she was discharged in stable condition while skin biopsy reports were pending. A subsequent punch biopsy of the purpuric lesion on the right calf revealed spongiotic dermatitis with no evidence of vasculitis, dysplasia, or malignancy.

At a two-week dermatology clinic follow-up, the lesions had resolved with minimal postinflammatory hyperpigmentation. Direct immunofluorescence (DIF) results were pending at that time, but the tissue specimen subsequently demonstrated faint, fine granular deposits of IgM and bright granular deposits of C3 within the walls of the upper dermal vessels. Although these findings suggested vasculitis, clinical and histopathological correlation was required. During the eight-week follow-up, testing for antineutrophil cytoplasmic antibodies was proposed to the patient; however, she declined further investigation because

she remained systemically well without rash recurrence.

A young woman with a two-week history of upper respiratory tract infection symptoms treated with antibiotics presented with urticaria mixed with purpura on the limbs, associated with severe malaise and prostration. One differential diagnosis considered for the urticaria was a viral exanthem, given that the pruritic eruption followed an upper respiratory tract infection. Viral exanthems, however, generally present as maculopapular or morbilliform eruptions (Image 2). Urticarial eruptions involving transient, pruritic wheals are not a classic or common manifestation of a viral exanthem, although EBV has been uncommonly linked to urticarial presentations.[1,2]

Another differential diagnosis is a delayed drug eruption, supported by the patient's recent completion of an antibiotic course. Although the patient had a documented history of an allergic reaction (urticaria) to amoxicillin-clavulanate potassium, amoxicillin is a penicillin combined with a β-lactamase inhibitor, whereas clarithromycin is a macrolide. There is no known cross-reactivity between these drug classes. However, the possibility of an independent, unconfirmed drug hypersensitivity had to be considered, necessitating a thorough medication history.

Alternatively, this could have represented an EBV-associated antibiotic rash. Patients with suspected streptococcal pharyngitis who receive antibiotics and subsequently develop an eruption are classically considered to have underlying EBV infection. The most commonly proposed mechanism of antibiotic-induced skin reactions in the setting of infectious mononucleosis is that the underlying viral infection triggers antibiotic hypersensitivity secondary to immune alterations, rather than a true, persistent drug allergy. This virus-mediated immune alteration typically leads to a generalized, delayed hypersensitivity response involving the palms and soles, especially after exposure to aminopenicillins such as amoxicillin (Image 3). However, because this patient received clarithromycin—typically considered a safe alternative—this etiology was less probable. Furthermore, although this reaction was historically considered pathognomonic for EBV infection, with an incidence of 80% to100%, recent studies suggest a much lower incidence of 15% to 33%.[3]

The referring family physician had also suspected scarlet fever, which is characterized by a sandpaper-like exanthem and a strawberry tongue (Image 4). This can be supported diagnostically by detecting elevated antistreptolysin O titer indicating recent group A streptococcal infection. Untreated scarlet fever risks systemic complications such as rheumatic heart disease and glomerulonephritis.[4] In view of the severity of such complications, suspected scarlet fever should always be worked up and, if positive, treated with an appropriate antibiotic course. It was unlikely in this case because the patient's age was above the usual demographic (5–15 years old), and her symptoms were not pathognomonic of the

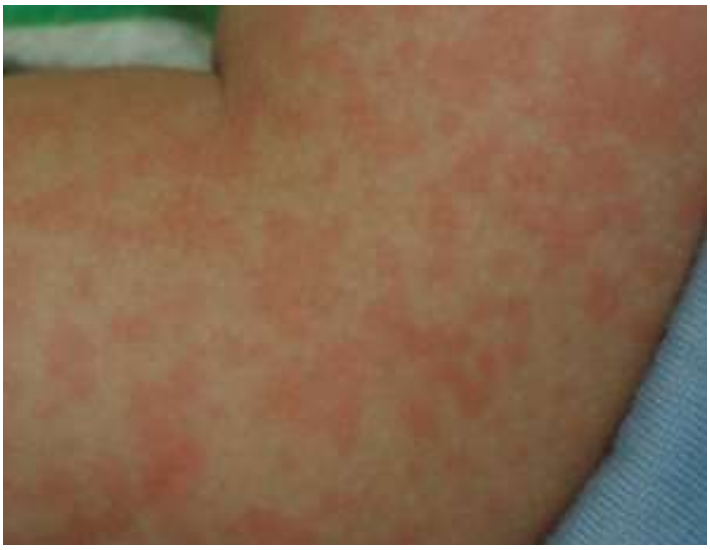


Image 2. Characteristically maculopapular or morbilliform viral exanthem rash. Reprinted from Rehmus et al[13] under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0) License.



Image 3. Erythematous, maculopapular eruption on the hand of a patient with an Epstein-Barr virus infection. Reprinted from Akiyama et al[14] under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0).

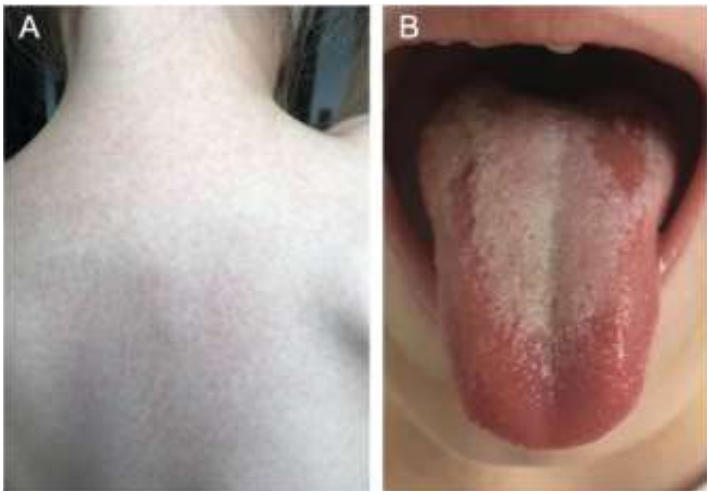


Image 4. (A) Sandpaper-like exanthem on the back and (B) strawberry tongue in a 4-year-old patient with scarlet fever. Source: Ślebioda et al.[15] This image is licensed under CC BY 3.0.

condition, with a different rash morphology and no scarlet tongue. Nevertheless, inpatient testing revealed normal antistreptolysin O titers.

While the above entities account for the common causes of such a rash, several atypical features in this patient's presentation—lasting greater than 24 hours, co-existing with purpura, absence of angioedema and presence of systemic symptoms (fever, malaise)—suggested an alternative pathology. Such features raise the suspicion of urticarial vasculitis, a rare entity where type 3 immune complex-mediated inflammation produces urticarial lesions with purpura rather than wheals. This disease is largely idiopathic, though it has been linked to connective tissue diseases, hematologic diseases, and autoinflammatory syndromes.[5] Our patient's purpuric-looking lesions raised the suspicion of vasculitis and demanded a more thorough evaluation.

Studies have shown that EBV can be associated with vasculitis although it is considered rare.[6-9] There have been case reports of EBV being implicated as a trigger of immune dysregulation in hypocomplementemic urticarial vasculitis (HUV). Tsai et al[10] described a case of acute respiratory distress syndrome in a patient with HUV, where EBV was detected, and Berggren et al reported persistent EBV-related antigens in a patient with HUV.[11] The proposed mechanism lies in molecular mimicry: anti-C1q auto-antibodies that cross-react with an EBV-derived nuclear antigen.[12]

The patient's purpuric lesions therefore prompted a concern for vasculitic processes. Recognition of such cases is essential because early identification of an underlying systemic or autoimmune trigger can alter management.

The punch biopsy of the purpuric right-calf lesion showed spongiotic dermatitis with no convincing features of vasculitis, while the DIF of biopsy tissue showed faint, fine granular deposits of IgM and bright granular deposits of C3 seen within the walls of the upper dermal vessels, suggestive of vasculitis. The incongruence between routine histology and DIF may reflect the timing of the biopsy, given that classic leukocytoclastic changes can resolve rapidly as the acute inflammatory change subsides, leaving only residual immune complex deposition detectable on DIF.

For our patient, the short duration of rash and spontaneous resolution suggests a subtle, self-limiting parainfectious vasculitis, consistent with a transient type III immune complex-mediated reaction that did not cause significant longstanding histological changes.

CONCLUSION

This report describes a patient with an EBV-associated parainfectious urticarial eruption who underwent detailed blood and histopathological evaluation. We systematically analyzed differential diagnoses, focusing on the exclusion of *Streptococcus* infection to guide rheumatic fever prevention measures. Prompt identification of atypical urticaria presentations and thorough diagnostic workups are vital to ensuring accurate clinical management and favorable patient outcomes.

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Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Huang XinYao Oliver, MBBS, Changi General Hospital, Department of Emergency Medicine, 2 Simei Street 3, Singapore 529889. Email: oliverhxy@yahoo.com.sg.

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Behavioral Trigger Identification in Recurrent Postpartum Chest Pain After Negative Cardiopulmonary Evaluation: A Case Report

Aditya S. Shrivastav, BSc CVT* *Datta Meghe Institute of Higher Education and Research, Department of Cardiology, Maharashtra, India
Suhas Tivaskar, MSc MRIT† †Datta Meghe Institute of Higher Education and Research, Department of Radiology and Imaging Technology, Maharashtra, India
Anurag Luharia, MD‡ ‡Datta Meghe Institute of Higher Education and Research, Department of Radiation Therapy, Maharashtra, India
Gaurav Mishra, MD§ §Datta Meghe Institute of Higher Education and Research, Jawaharlal Nehru Medical College, Department of Radiodiagnosis, Wardha, Maharashtra, India

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Introduction: Recurrent chest pain in postpartum patients frequently results in repeated emergency department (ED) visits despite negative cardiopulmonary investigations, creating diagnostic uncertainty and increasing healthcare resource utilization. We report a case highlighting the value of a structured postexclusion assessment to identify clinically relevant behavioral triggers after life-threatening causes have been excluded.

Case Report: A 33-year-old woman, six weeks postpartum, presented repeatedly with nocturnal chest pain, palpitations, and dyspnea. Serial electrocardiograms, cardiac biomarkers, chest imaging, and transthoracic echocardiography were consistently normal, excluding acute cardiopulmonary pathology. Careful reassessment of symptom chronology and behavioral history identified a reproducible temporal association between symptom onset and nocturnal binge-eating episodes. Following outpatient psychiatric and clinical psychological evaluation confirming Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, diagnostic criteria for binge-eating disorder, targeted behavioral counseling led to a marked reduction in symptom frequency and no further ED presentations during follow-up.

Conclusion: This case demonstrates that, after exclusion of life-threatening cardiopulmonary disease, a structured assessment emphasizing symptom temporality and behavioral triggers can uncover clinically actionable mechanisms underlying recurrent postpartum chest pain. Incorporating this approach into emergency department evaluation may facilitate appropriate management while reducing unnecessary repeat investigations and ED utilization. [Clin Pract Cases Emerg Med. 2026;10(4):614–618.]

Keywords: *chest pain; postpartum period; emergency service; noncardiac chest pain; eating disorders.*

INTRODUCTION

Chest pain accounts for approximately 5% to 8% of all emergency department (ED) visits worldwide and routinely prompts high-intensity investigation because of the need to rapidly exclude life-threatening cardiopulmonary disease.[1,2] Postpartum patients represent a subgroup with particularly

high ED utilization, especially during the first weeks following delivery, driven by physiological changes after childbirth, heightened symptom vigilance, and low thresholds for re-presentation when symptoms recur.[3,4] In this population, clinicians must remain alert for serious conditions including pulmonary embolism, peripartum cardiomyopathy, acute coronary syndromes, and other cardiopulmonary complications, which often justify comprehensive diagnostic evaluation.

Although most postpartum chest pain assessments ultimately exclude acute cardiac pathology, repeated presentations are common and contribute substantially to diagnostic workload, resource utilization, and patient distress.[1,3,5] For emergency physicians, recurrent chest pain with consistently negative investigations presents a practical challenge: Reassurance alone often fails to prevent return visits, yet clear guidance on next-step assessment once acute pathology has been excluded remains limited.[1]

The absence of structured postexclusion approaches may lead to repeated testing without meaningful progression in clinical understanding or management.[2,6] Such postexclusion assessment typically focuses on symptom temporality, behavioral triggers, and psychosocial context. This case is relevant to emergency clinicians because it illustrates a pattern frequently encountered in practice—recurrent postpartum chest pain, repeated negative cardiopulmonary investigations, and ongoing ED utilization—and demonstrates how a focused post-exclusion clinical assessment can yield clinically actionable insight beyond repeated diagnostic exclusion.[6] To our knowledge, few published reports have specifically described a structured postexclusion clinical assessment integrating symptom temporality and behavioral triggers in recurrent postpartum chest pain after exclusion of life-threatening cardiopulmonary disease. This case therefore offers a practical diagnostic approach for emergency clinicians managing recurrent postpartum chest pain after negative investigations.

CPC-EM Capsule

What do we already know about this clinical entity?
Recurrent postpartum chest pain often leads to repeated emergency department (ED) visits despite negative cardiopulmonary investigations.

What makes this presentation of disease reportable?
A structured post-exclusion assessment identified binge-eating disorder as the reproducible behavioral trigger after repeated negative evaluations.

What is the major learning point?
Assessment of symptom temporality and behavioral triggers may reveal clinically actionable diagnoses after serious cardiopulmonary disease has been excluded.

How might this improve emergency medicine practice?
Structured postexclusion assessment may facilitate appropriate referral and decrease recurrent ED utilization.

CASE REPORT

A 33-year-old woman presented repeatedly to the ED over a 6-week period with recurrent chest pain during the early postpartum phase, beginning approximately 6 weeks after an uncomplicated vaginal delivery. Her pregnancy and delivery were uneventful. She had no history of cardiovascular disease, metabolic disorders, or prior similar symptoms.

Chest pain episodes were sudden in onset, predominantly nocturnal, and associated with palpitations and dyspnea. Pain severity was reported as high (up to 8/10 on a numerical rating scale). On several occasions, transient left-sided chest heaviness prompted evaluation for acute coronary syndrome. There was no clear exertional trigger, radiation of pain, or positional or pleuritic component. Despite reassurance following prior evaluations, symptom recurrence frequently prompted repeat ED attendance. Across multiple ED presentations, cardiopulmonary pathology was systematically excluded. Serial electrocardiograms demonstrated normal sinus rhythm without ischemic changes (Image).

Cardiac biomarkers, including high-sensitivity cardiac troponin I, remained below the institutional diagnostic threshold (reference range, < 26.2 ng/L). Routine laboratory investigations including hemoglobin, white blood cell count, and C-reactive protein were all within normal reference ranges. Transthoracic echocardiography demonstrated normal cardiac structure with preserved left ventricular ejection fraction (approximately 60–65%), normal chamber dimensions, and no regional wall motion abnormalities or pericardial effusion. There was no evidence of postpartum cardiomyopathy or sustained arrhythmia. Pulmonary embolism was considered during evaluation and excluded based on low clinical probability and absence of supportive findings on clinical evaluation and imaging.

During acute assessments, clinicians considered several differential diagnoses including gastroesophageal reflux disease, panic-related symptoms, musculoskeletal chest pain, pulmonary embolism, and cardiomyopathy. These possibilities

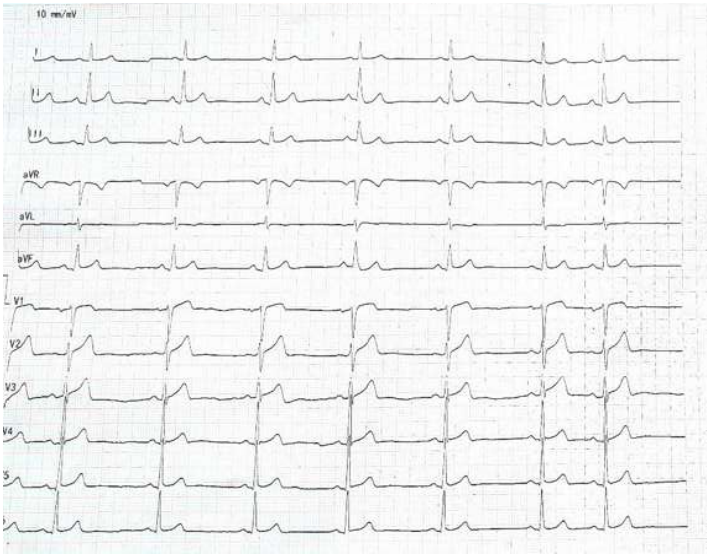


Image. Normal electrocardiogram recorded during chest pain episode. Representative 12-lead electrocardiogram obtained during one of several emergency department presentations.

were evaluated using clinical assessment and appropriate investigations and were considered unlikely based on repeatedly normal findings and the absence of supportive clinical features (Table).

Physical examination was unremarkable, with stable vital signs and a body mass index of 22.4 kg/m². During symptomatic presentations, vital signs remained stable; blood pressure, approximately 118/72 mm Hg; heart rate, approximately 100–110 beats per minute (bpm); respiratory rate, 20 breaths per minute, and SpO_2, 98% on room air. There were no signs of malnutrition, purging behavior, or restrictive intake.

After repeated negative investigations, we obtained a more detailed history focusing on symptom timing and behavioral patterns. This assessment suggested recurrent nocturnal binge-eating episodes beginning during the postpartum period. The patient was subsequently referred for outpatient psychiatric and clinical psychological evaluation, where a comprehensive behavioral assessment was performed. The diagnosis of binge-eating disorder was established according to Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), diagnostic criteria by the treating psychiatrist and clinical psychologist. The assessment included a detailed clinical interview focusing on eating patterns, episode frequency, loss of control during binge episodes, associated distress, and symptom chronology. Reconstruction of a symptom–behavior timeline using clinical interview and patient diary review demonstrated that chest pain episodes consistently occurred 30–90 minutes after binge-eating episodes (Figure). Although no validated eating-disorder screening questionnaire was administered, the diagnosis was established through specialist DSM-5–based clinical assessment.

No chest pain was reported on nights without binge eating.

Transient sinus tachycardia (100–110 bpm) was documented during episodes. Episodes were also associated with epigastric fullness, belching, and heightened awareness of cardiac sensations.

Over 6 weeks, repeated ED visits led to multiple negative cardiopulmonary evaluations. Following confirmation of the diagnosis, the patient received behavioral counseling from the multidisciplinary team, including psychoeducation regarding binge-eating disorder, regular meal scheduling, avoidance of

Table. Diagnostic evaluation and clinical course in recurrent postpartum chest pain.

Domain	Key Findings	Assessment/Intervention	Outcome
Acute coronary syndrome	Chest heaviness, palpitations	Serial ECGs, troponin I	Normal ECGs and biomarkers
Postpartum cardiomyopathy	Dyspnea, chest discomfort	Transthoracic echocardiography	Normal ventricular function
Pulmonary embolism	Dyspnea, tachycardia	Clinical probability assessment ± imaging	No supporting findings
Gastroesophageal reflux disease	Postprandial discomfort	Clinical correlation	Not consistent; no response to antacids
Panic-related symptoms	Chest pain, palpitations	Clinical assessment	Temporal pattern favored behavioral etiology
Musculoskeletal pain	Localized tenderness	Physical examination	No reproducible tenderness
Arrhythmia	Palpitations	ECG monitoring	No arrhythmia detected
Behavioral trigger (binge eating)	Nocturnal episodes, postprandial timing	Symptom–behavior correlation, outpatient referral	Reduced binge frequency and chest pain
Follow-up course	Recurrent ED visits initially	Behavioral counseling	No further ED visits

ECG, electrocardiogram; *ED*, emergency department.

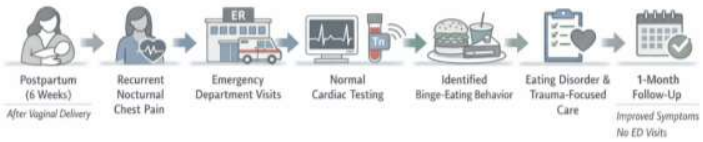


Figure. Clinical course timeline across the postpartum period. *ED*, emergency department.

nocturnal binge-eating episodes, recognition of symptom triggers, reassurance regarding the absence of underlying cardiopulmonary disease, and continued psychiatric and nutritional follow-up. She was referred for outpatient behavioral and nutritional follow-up to reinforce these strategies.

At 1-month follow-up, binge-eating frequency had decreased substantially (from near-nightly episodes to approximately once weekly), accompanied by a parallel reduction in chest pain episodes (Table). No further ED presentations were reported during the follow-up period.

Patient Perspective

The patient reported that understanding the relationship between nocturnal binge-eating episodes and chest pain reduced anxiety about a cardiac cause and improved confidence in managing recurrent symptoms.

DISCUSSION

Recurrent chest pain is a frequent and resource-intensive presentation in the ED, particularly in postpartum patients, requiring exclusion of life-threatening conditions such as pulmonary embolism, peripartum cardiomyopathy, and acute coronary syndromes. Consequently, comprehensive diagnostic evaluation is routinely performed. Despite this, a substantial proportion of patients are ultimately diagnosed with noncardiac chest pain after negative investigations,[6,7] with emerging evidence highlighting the contribution of psychiatric factors in recurrent presentations.[8] After exclusion of cardiopulmonary pathology, persistent diagnostic uncertainty often remains. In many cases, reassurance alone does not prevent repeat ED visits, resulting in recurrent low-yield investigations, increased healthcare utilization, and ongoing patient distress, a pattern well described in patients with recurrent noncardiac chest pain presenting to ED.[9] The absence of structured approaches for evaluating symptoms after diagnostic exclusion may contribute to this cycle of repeated testing without meaningful progression in clinical understanding.

The key observation in this case was the consistent temporal association between nocturnal binge-eating episodes and subsequent chest pain. Large-volume postprandial intake may produce chest discomfort through gastric distension, transient gastroesophageal reflux, and autonomic activation, all of which can mimic cardiac chest pain.[6,10] In this case, chest pain reliably occurred within a defined 30- to 90-minute postprandial window and was absent on nights without binge eating, making temporality the most informative clinical observation.

Alternative explanations were also considered. Gastroesophageal reflux disease, esophageal spasm, anxiety-related autonomic symptoms, and panic-related chest pain can all produce similar clinical presentations. However, the reproducible association between binge-eating episodes and symptom onset, combined with improvement following behavioral counseling and reduction in binge episodes, was consistent with a behavioral and postprandial physiological mechanism. In this case, the eating behavior was not formally diagnosed during the ED visit itself but was subsequently evaluated and confirmed during outpatient follow-up after referral. In this case, the diagnosis of binge-eating disorder was established by a psychiatrist and clinical psychologist using DSM-5 diagnostic criteria during outpatient follow-up. Although no validated screening questionnaire was administered, DSM-5–based specialist clinical assessment represents the diagnostic standard for binge-eating disorder in routine clinical practice.

From an emergency medicine perspective, structured postexclusion assessment is essential in recurrent chest pain. A focused evaluation of symptom temporality, nocturnal patterns, recent eating behavior, and associated autonomic symptoms may help identify reproducible symptom–behavior relationships and guide appropriate outpatient referral, reducing repeat low-yield investigations. This case should also be interpreted in light of several limitations. As a single-patient observation, the findings cannot establish a causal relationship between binge-eating behavior and chest pain, and the proposed mechanism remains inferential. Although the diagnosis of binge-eating disorder was established by a psychiatrist and clinical psychologist using DSM-5 diagnostic criteria, no validated eating-disorder screening questionnaire was administered. This may limit direct comparison with studies using standardized screening instruments.

CONCLUSION

Recurrent postpartum chest pain with negative investigations may reflect reproducible behavioral triggers not identified through routine testing. Structured postexclusion assessment focusing on symptom temporality and behavior can provide actionable clinical insight and reduce repeat ED utilization.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Aditya S. Shrivastav, BSc CVT, Datta Meghe Institute of Higher Education and Research, Department of Cardiology, Maharashtra, India. Email: aaditya.6361@gmail.com.

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Partial Segmental Thrombosis of the Corpus Cavernosum Following Valsalva Maneuver: A Case Report

Sitha Konopack, MD, MPH
Jessica Diaz, MD
University of Florida College of Medicine, Department of Emergency Medicine, Gainesville, Florida

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Introduction: Partial segmental thrombosis of the corpus cavernosum is a rare vascular condition that may present with nonspecific groin or pelvic pain and mimic more common surgical or urologic emergencies.

Case Report: A 33-year-old man presented to the emergency department with acute left-sided groin and pelvic pain after straining during defecation. Initial history and evaluation raised concern for incarcerated inguinal hernia, epididymo-orchitis, renal colic, lymphadenopathy, and soft tissue infection. Computed tomography demonstrated asymmetric enlargement of the left corpus cavernosum, while penile ultrasound showed diminished cavernosal flow. Magnetic resonance imaging confirmed segmental thrombosis of the left corpus cavernosum. The patient was treated conservatively with rivaroxaban and analgesia, resulting in symptomatic improvement.

Conclusion: Partial segmental thrombosis of the corpus cavernosum should be considered in patients presenting with unexplained groin or perineal pain when standard abdominal, penile, testicular, and hernia evaluations are unrevealing. Magnetic resonance imaging may be necessary for definitive diagnosis, and conservative management is frequently successful. [Clin Pract Cases Emerg Med. 2026;10(4):619–622.]

Keywords: *emergency department; pelvic pain; corpus cavernosum thrombosis; groin pain; case report.*

INTRODUCTION

Groin and lower pelvic pain are common emergency department (ED) presentations with a broad differential diagnosis, including inguinal hernia, urologic infection, renal colic, testicular torsion, musculoskeletal injury, paraphimosis, priapism, and soft tissue infection. Vascular causes involving penile structures are often overlooked during initial evaluation.

Partial segmental thrombosis of the corpus cavernosum (PSTCC) is an uncommon vascular disorder characterized by localized thrombosis within the erectile tissue of the penis and has rarely been described in the literature.[1–5] Unlike ischemic priapism, PSTCC often presents without sustained erection, contributing to delayed diagnosis or misdiagnosis.[1–3]

We describe a case of PSTCC presenting as acute groin and pelvic pain following a Valsalva maneuver during defecation, highlighting the diagnostic challenges and imaging findings relevant to emergency medicine practice.

CASE REPORT

A 33-year-old male with a history of prior left inguinal hernia repair presented to the ED with 3 days of progressive left groin and pelvic pain that began immediately after straining during defecation. He reported difficulty passing stool and transient nonbloody emesis but denied fever, urinary symptoms, hematuria, penile discharge, trauma, erectile dysfunction, or sustained erection. He denied a personal or family history of thromboembolic disease.

On presentation, his vital signs were as follows: blood pressure, 116/82 mm Hg; heart rate, 67 beats per minute; respiratory rate, 15 breaths per minute; temperature, 36.7 °C; and SpO_2, 99% on room air. Physical examination demonstrated a well-appearing male in no acute distress. The abdomen was

soft, nontender, and nondistended without guarding or rebound tenderness. Examination of the external genitalia revealed a normal, flaccid penis without swelling, tenderness, erythema, discoloration, or priapism. A small palpable nodule with minimal tenderness was identified in the left perineum. Bilateral testes were descended and equal in size, with mild tenderness to palpation of the left testicle. There was no scrotal swelling, crepitus, erythema, skin discoloration, or other evidence of soft tissue infection involving the perineum. The initial emergency department differential diagnosis included incarcerated inguinal hernia, epididymo-orchitis, renal colic, lymphadenopathy, and soft tissue infection.

Computed tomography (CT) of the abdomen and pelvis demonstrated no intra-abdominal pathology or recurrent hernia but did reveal asymmetric enlargement of the left corpus cavernosum (Image 1).

Subsequent penile ultrasound demonstrated a heterogeneous avascular lesion within the left corpus cavernosum, raising concern for cavernosal vascular pathology (Image 2).

Given the patient's persistent pain and indeterminate imaging findings, urology consultation was obtained, and further evaluation with magnetic resonance imaging (MRI) and cavernosal blood gas analysis was recommended. The cavernosal blood gas demonstrated a markedly hypoxic sample with low oxygen tension and oxygen saturation, consistent with localized cavernosal ischemia and supportive of the diagnosis of partial segmental thrombosis of the corpus cavernosum (Table).

Magnetic resonance imaging of the pelvis subsequently confirmed a 7-cm segment of thrombosis involving the left corpus cavernosum with absent postcontrast enhancement consistent with thrombus (Image 3).

Cavernosal blood gas analysis obtained in the ED demonstrated hypoxic venous blood, supporting localized ischemia. The final diagnosis was partial segmental thrombosis of the corpus cavernosum. The patient was started on an intravenous heparin infusion and admitted to the family medicine service with ongoing urology and hematology consultation.

The patient was managed collaboratively with urology and hematology consultation. Initial treatment consisted of intravenous heparin infusion for anticoagulation along with ibuprofen for analgesia. Following clinical stabilization,

CPC-EM Capsule

What do we already know about this clinical entity?
Partial segmental thrombosis of the corpus cavernosum is a rare penile vascular disorder that often presents without classic priapism.

What makes this presentation of disease reportable?
We report a case of isolated groin and pelvic pain following a Valsalva maneuver, mimicking more common surgical and urologic conditions.

What is the major learning point?
Partial segmental thrombosis of the corpus cavernosum should be considered in patients with unexplained groin or perineal pain and unrevealing initial evaluation.

How might this improve emergency medicine practice?
Recognition of this uncommon diagnosis may facilitate appropriate imaging, specialty consultation, and timely conservative management.



Image 1. Computed tomography of the abdomen and pelvis with intravenous contrast. (**A**) Axial view demonstrates asymmetric enlargement of the left corpus cavernosum, highlighting changes in cross-sectional diameter of the cavernous body (white arrow). (**B**) Coronal view demonstrates longitudinal asymmetry of the left corpus cavernosum, demonstrating the cranial-to-caudal extent of the enlargement (black arrow).

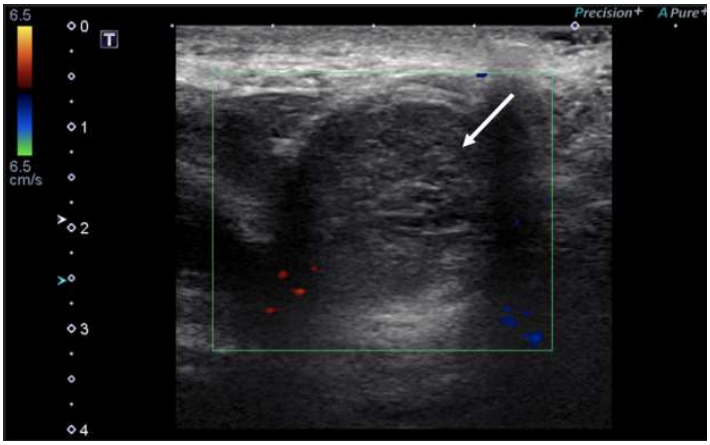


Image 2. Penile ultrasound. Grayscale ultrasound demonstrates heterogeneous avascular lesion within the left corpus cavernosum (arrow).

Table. Cavernosal blood gas analysis.

Parameter	Result
pH	7.46
pCO_2	37 mmHg
pO_2	35 mmHg
HCO_3^-	26 mmol/L
Base Excess	+2 mmol/L
SpO_2	70%
Total CO_2	27 mmol/L

CO_2, carbon dioxide*; HCO_3*, bicarbonate; *mmHg*, millimeters of mercury; *mmol/L*, millimoles per liter; *pCO_2*, partial pressure of carbon dioxide; *pO_2*, partial pressure of oxygen; SpO_2, oxygen saturation.

the patient was transitioned to rivaroxaban 20 mg daily with a planned treatment duration of 3 months. No surgical intervention was required. The patient reported symptomatic improvement prior to discharge and was discharged with outpatient urology and hematology follow-up. No adverse events occurred during hospitalization. Outpatient evaluation for hypercoagulability revealed no provoking factors, and hypercoagulable testing was negative. The patient completed a three-month course of rivaroxaban with continued symptomatic improvement but was subsequently lost to follow-up.

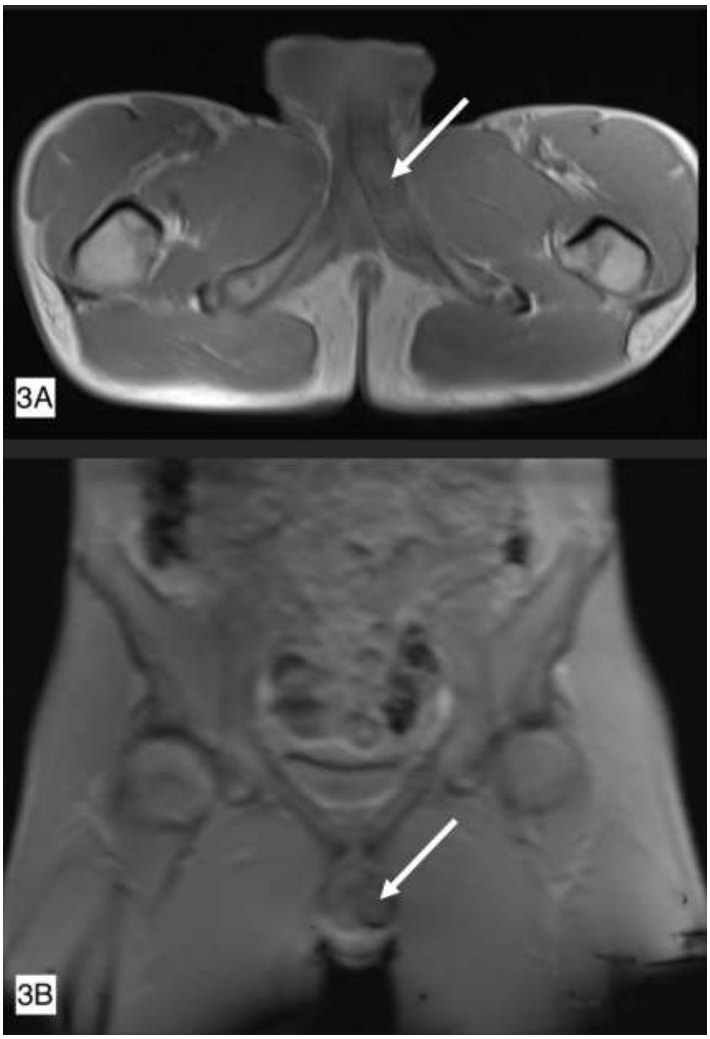


Image 3. Magnetic resonance imaging of the pelvis. **(A)** Axial view demonstrates asymmetry between the left and right corpora cavernosa, with localized, segmental expansion of the left corpus cavernosum. The white arrow highlights widening and an absence of typical blood flow or internal contrast enhancement within the affected tissue. **(B)** Coronal view demonstrates asymmetric, segmental enlargement of the left corpus cavernosum (white arrow), with a distinct T2-hypointense signal in the proximal portion and mass effect on the adjacent right (contralateral) corpus cavernosum.

DISCUSSION

From an emergency medicine perspective, PSTCC is an uncommon diagnosis that may not be initially considered in patients presenting with isolated groin or perineal pain. Patients frequently lack the prolonged erection or obvious penile findings associated with ischemic priapism, and the diagnosis may be overlooked during the initial ED evaluation. Initial assessment appropriately prioritizes more common and time-sensitive conditions, including incarcerated inguinal hernia, urologic infection, renal colic, testicular torsion, musculoskeletal injury, paraphimosis, priapism, and soft tissue infection. The palpable inguinal abnormality in this case may have represented referred tenderness or extension along proximal corporal structures rather than discrete superficial pathology, although the precise correlation between the physical examination finding and imaging abnormality remained uncertain.

Partial segmental thrombosis of the corpus cavernosum is a rare vascular condition that characteristically presents with localized penile, groin, or perineal pain without the prolonged erection typical of an ischemic priapism.[1–3] The condition is thought to result from venous outflow obstruction within a septated segment of the corpus cavernosum, leading to localized thrombosis.[2] Valsalva maneuvers, including straining during defecation, are proposed precipitating factors because transient increases in intracavernosal venous pressure may promote venous stasis and thrombosis.[1–3] Superficial dorsal penile vein thrombosis, also known as Mondor disease, may present with similar penile or groin pain but typically involves the superficial dorsal venous system rather than cavernosal tissue.[2]

Unlike ischemic priapism, PSTCC often involves only a segment of the corpus cavernosum due to the presence of a fibrous septum, thereby preventing complete corporal involvement and sustained erection. Diagnosis is primarily imaging-based. Ultrasound may demonstrate an avascular heterogeneous lesion, while MRI provides superior characterization of thrombus extent and exclusion of alternative penile pathology.[2] Although CT demonstrated asymmetric cavernosal enlargement, the finding was nonspecific, whereas MRI provided definitive characterization

of the thrombus and exclusion of alternative pathology. Cavernosal blood gas analysis may demonstrate hypoxia consistent with localized ischemia. Management is typically conservative and consists of anticoagulation and nonsteroidal anti-inflammatory medications, with most patients experiencing favorable outcomes.[1–4]

For emergency physicians, this case highlights the importance of including penile vascular pathology in the differential diagnosis of patients presenting with unexplained groin or perineal pain. Unrevealing abdominal imaging should not exclude consideration of deep pelvic or penile vascular pathology, particularly when symptoms persist despite an otherwise reassuring evaluation. Correlation of focal inguinal or perineal examination findings with advanced imaging may facilitate earlier diagnosis and appropriate conservative management.

CONCLUSION

Partial segmental thrombosis of the corpus cavernosum is an uncommon but important vascular cause of groin and perineal pain that may mimic more common surgical or urologic conditions. Emergency physicians should consider PSTCC when standard abdominal, testicular, and hernia evaluations are unrevealing. Magnetic resonance imaging may be required for definitive diagnosis, and conservative management with anticoagulation is often successful.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Sitha Konopack, MD, MPH, University of Florida College of Medicine, Department of Emergency Medicine, 1329 SW 16th Street, Suite 5270, Gainesville, FL 32610. Email: seetha@ufl.edu.

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Emergency Department Use of Genicular Nerve Block for Sickle Cell Pain Crisis: A Case Report

Rosemary Beranek, DO* *Hennepin County Medical Center, Department of Emergency Medicine, Minneapolis, Minnesota
Hayley Musial, MD† †Essentia Health, Department of Emergency Medicine, Duluth, Minnesota
Daniel Parsons-Moss, MD*
Andrea Dreyfuss, MD, MPH*

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Introduction: Acute pain crises are a common emergency department presentation in patients with sickle cell disease. Pain management typically relies on multimodal analgesia, with opioids often playing a central role. We propose incorporating regional anesthesia techniques into the pain management regimen.

Case Report: We present a case of a young patient with sickle cell disease experiencing an acute pain crisis with severe knee pain. A genicular nerve block was performed, resulting in significant pain reduction in the affected knee.

Conclusion: Regional anesthesia, including isolated nerve blocks, may be safe and effective as part of a multimodal pain approach for patients suffering from acute pain crises due to sickle cell disease. This may help reduce the need for opioid or other sedating pain medications, as well as the inherent risks associated with increasing doses, such as respiratory depression. [Clin Pract Cases Emerg Med. 2026;10(4):623–627.]

Keywords: *genicular nerve block; regional anesthesia; sickle cell disease; acute pain crisis; case report.*

INTRODUCTION

One of the most common presentations of sickle cell disease (SCD) to the emergency department (ED) is sickle cell crisis or acute pain crises resulting from vaso-occlusion of vessels.[1,2] The mainstay of treatment for these patients in the acute setting often involves the use of opioid medications.[3] These medication regimens may limit the opioid doses patients can receive in the ED, frequently necessitating inpatient admission if pain control remains inadequate.

Although few data are available regarding the use of regional anesthesia in the ED for controlling chronic pain, the available data shows improvement in pain scores and decreased opiate use.[4] To date, there are no cases in the emergency medicine literature documenting the use of regional anesthesia in the ED as part of multimodal pain control for SCD patients in acute pain crises. Most of the published research describing the use of regional anesthesia for SCD patients focuses on epidural anesthesia.[5,6] To our knowledge, no case reports to date have described the use of genicular blocks for sickle cell-related pain. These blocks have been described in anesthesia literature following knee replacements or knee trauma but have not yet been adopted into widespread use in the ED.[7–10]

CASE REPORT

We present the case of a 27-year-old male with SCD who presented to the ED with acute sickle cell pain, mainly in his right knee and wrist. The patient had a history of sickle cell beta thalassemia, osteonecrosis of the lumbar spine, and splenomegaly, with multiple prior presentations for vaso-occlusive crises managed with hydroxyurea, glutamine, and crizanlizumab infusions.

The patient called emergency medical services when his pain was unrelieved with his prescribed oxycodone.

Paramedics administered 0.5 mg of intravenous (IV) hydromorphone. In the ED, he initially received 15 mg IV ketorolac, 975 mg oral acetaminophen, and 1 mg IV hydromorphone. Over the next several hours, the patient required several more doses of 0.5 to 1 mg IV hydromorphone boluses before he was eventually started on a patient-controlled analgesia pump of hydromorphone. He developed intermittent mild to moderate hypoxia due to decreased respiratory drive, requiring 3–4 liters of supplemental oxygen via nasal cannula. The patient was still uncomfortable, complaining of persistent knee pain despite the patient-controlled analgesia. The team proposed a nerve block to reduce this pain. He consented to a genicular block, which was performed at the bedside. The patient reported an improvement in pain from a "10/10 to a 2/10" approximately 30 minutes after the procedure. He was admitted to the hospital for ongoing management of his sickle cell crisis.

Sensory innervation of the knee is complex and involves terminal sensory branches of the femoral, obturator, and sciatic nerves. The anterior knee is divided into four quadrants, each with a respective genicular nerve branch: the superomedial genicular nerve, the superolateral genicular nerve, the inferomedial genicular nerve, and the inferolateral (ILGN) genicular nerve.

While the American Society of Regional Anesthesia and Pain Medicine recommends blocking three of these nerves but omitting the ILGN to prevent accidentally blocking the adjacent common peroneal nerve (which could result in foot drop),[5] the physician may opt to target this nerve as well to provide greater pain coverage. Anesthesia guidelines also suggest blocking the femoral nerve branch innervating the vastus intermedius, which lies anteriorly along the femur, to provide anesthesia over the patellar region.

Our block targeted all four genicular nerves. The patient did not receive a block to the femoral nerve due to physician discretion and patient preference to forgo a fifth injection. The procedure took approximately 35 minutes. Ropivacaine 0.5% was used, which generally takes 10–20 minutes to take effect. The block may last up to 15 hours.

The sonographic landmarks for these nerves are relatively simple to find. The landmarks are shallow and require visualization of the bony cortex of the femur and tibia. With little overlying soft tissue, these landmarks are superficial; therefore, a linear probe is used (Image 1). The objective of the procedure is not to visualize the nerves themselves but rather the bony cortex at the slope between the shaft and epicondyles of the long bones. Color Doppler is an additional tool used to visualize the four genicular arteries, which run in the genicular neurovascular bundle. For the inferior landmarks, the tibial metaphysis is the target landmark (Images 2 and 3), whereas for the superior nerves it is the femoral epicondyle.

CPC-EM Capsule

What do we already know about this clinical entity?
Nerve blocks are safe and effective for many causes of musculoskeletal pain or injuries as an adjunct to standard pain regimens.

What makes this presentation of disease reportable?
A genicular block relieved acute knee pain in a patient with sickle cell disease.

What is the major learning point?
Specific pain sites, such as joints, can be targeted for pain reduction using regional anesthesia.

How might this improve emergency medicine practice?
Incorporating regional anesthesia into a multimodal regimen offers targeted pain control and minimizes total analgesic use.

DISCUSSION

This case represents the first documented use of a genicular block for sickle cell-related pain in the emergency setting. For patients with acute pain crisis, management often requires repeated doses of opioids or patient-controlled anesthesia pumps. These large or stacked doses can cause significant respiratory depression, which greatly increases morbidity risk.[3] Using multimodal analgesia to improve pain, while reducing opioid consumption, has become increasingly recognized as best practice in pain management.

Regional anesthesia has emerged as a valuable component of multimodal analgesia in emergency medicine. While few data exist specifically for regional anesthesia in sickle cell disease, growing evidence supports its use for acute pain management in the ED. A systematic review by Ritcey et al of the use of regional analgesia for hip and femoral neck fractures in the ED found that nerve blocks significantly reduced pain scores and opioid consumption compared to standard analgesia alone.[4] While the review focused on traumatic injuries rather than chronic pain, it demonstrates the feasibility and efficacy of implementing regional anesthesia in the ED.

Beyond hip fractures, emergency physicians have successfully employed various peripheral nerve blocks for acute pain control. Burl et al demonstrated successful use of posterior tibial nerve blocks for plantar frostbite pain with improved pain

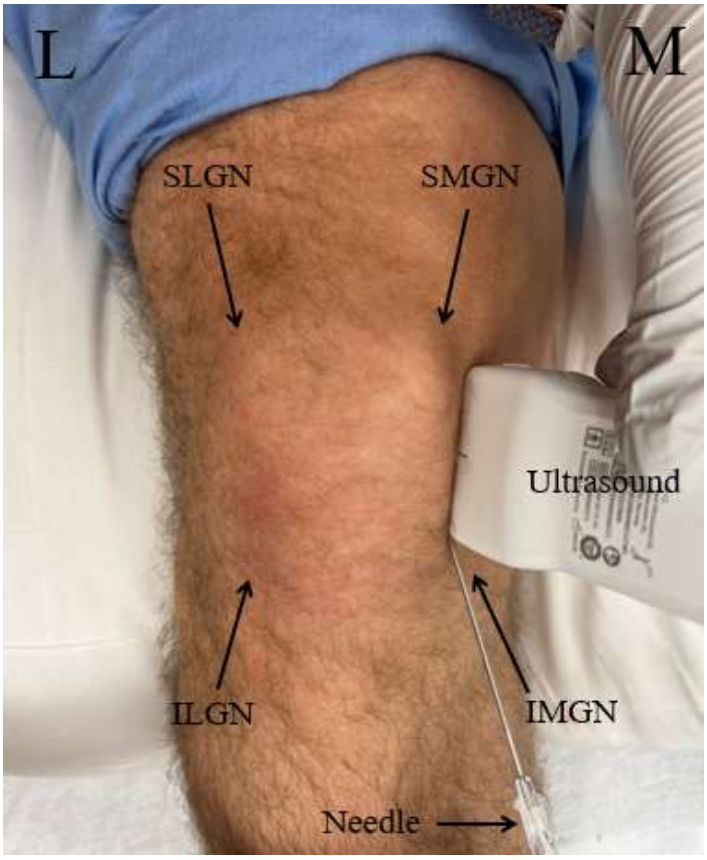


Image 1. Four injection sites for genicular nerve block, with linear ultrasound probe and needle at the inferomedial genicular nerve site. In clockwise order from the probe are the inferolateral, superolateral, and superomedial nervie sites.
ILGN, inferolateral genicular nerve; *IMGN,* inferomedial genicular nerve; *L*, lateral; *M,* medial; *SLGN,* superolateral genicular nerve

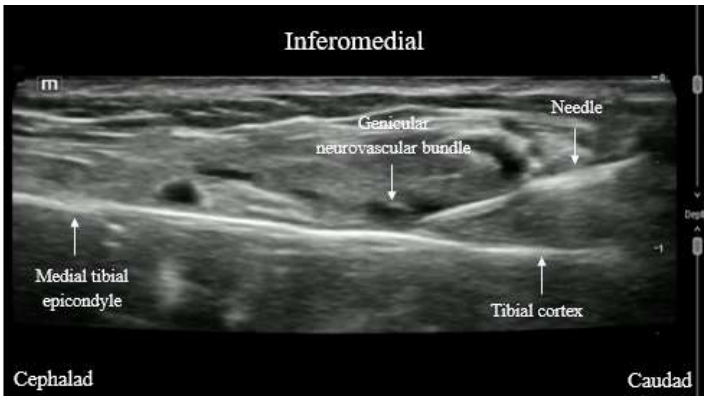


Image 2. Ultrasound image with relevant anatomy labeled for the inferomedial genicular nerve and the needle visualized in-plane with bony target of the tibial cortex.

scores.[11] Additionally, Nagel et al described femoral and fascia iliaca blocks to manage proximal traumatic femur fractures. They also compared the safety of these peripheral nerve blocks to a case where use of opioids caused oversedation of a patient, which led to her being intubated.[12] These studies collectively support the principle that targeted nerve blocks can provide effective analgesia while minimizing systemic medication side effects and complications.

Genicular nerve blocks have primarily been described in the chronic pain and anesthesia literature for managing knee osteoarthritis. Randomized controlled trials by Shanahan et al and Kim et al demonstrated the efficacy of ultrasound-guided genicular nerve blocks in decreasing pain scores in patients with chronic knee osteoarthritis. These studies established genicular blocks as viable options for chronic knee pain management in outpatient settings.[7] This was also assessed by Kim et al in a randomized controlled trial to assess efficacy of genicular nerve blocks with anesthetic alone or in combination with a corticosteroid.[8] While their study goal was to compare the differences in medication type, they documented success in pain reduction with the peripheral nerve block, regardless of whether corticosteroids were used.

More recently, genicular nerve blocks have been successfully applied in the ED for acute and subacute knee pain. Sobel and Oswald reported the first case of a 3-point genicular nerve block for acute traumatic knee pain in the ED, demonstrating rapid pain relief and improved mobility.[10] Kong et al subsequently described successful management of subacute osteoarthritic knee pain in the ED using genicular blocks.[9]

The ultrasound-guided technique offers several advantages for emergency physicians. The anatomic landmarks—the bony cortex at the femoral epicondyles and tibial metaphysis—are superficial and relatively consistent across patients. Unlike many nerve blocks that require direct visualization of neural structures, the genicular block targets the nerves based on their consistent anatomic relationship to bony landmarks and associated vessels. This makes the learning curve more accessible for physicians developing their regional anesthesia skills.

The application of regional anesthesia to chronic pain populations, particularly patients with sickle cell disease, represents an underexplored frontier. Given that vaso-occlusive crises commonly affect specific joints and anatomic regions, targeted peripheral nerve blocks offer a logical approach to pain control.

LIMITATIONS

There were multiple challenges that we faced. The patient reported pain in multiple joints, which made complete pain relief impossible. He had been placed on a patient-controlled hydromorphone pump prior to the genicular block. After the block, he began complaining of more pain in his wrist, requesting an additional block at that site. While his pain was mostly relieved in his knee, he did require ongoing IV

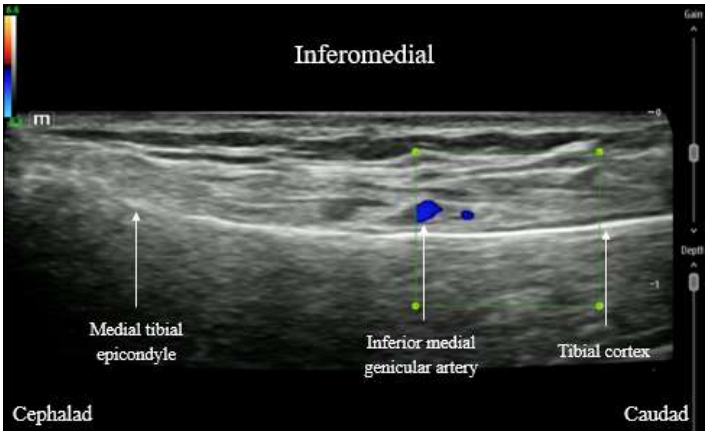


Image 3. Ultrasound image with relevant anatomy labeled for the inferomedial genicular nerve, needle visualized in-plane with bony target of tibial cortex. Color Doppler identifies the artery in the genicular neurovascular bundle.

parenteral medications for remaining pain in other joints. This is an expected limitation of regional anesthesia in SCD patients. It was also difficult to measure reduction in the patient's use of patient-controlled anesthesia postblock for the same reasons. To learn more about the benefits of this block for pain crises in patients who do not yet require patient-controlled anesthesia, in.future we aim to offer the block to SCD patients earlier in their presentation.

There were also challenges to performing the procedure itself. Because this block requires five injection sites to successfully block the entire knee, multiple injection sites can be time-consuming for the physician and uncomfortable for the patient. However, with extensive practice and greater ease in finding the landmarks, this block has the potential to be a useful adjunct for pain relief, given the superficiality and relative anatomic consistency of the landmarks.

CONCLUSION

This case report demonstrates a novel application of genicular nerve blocks as an adjunct in managing localized joint pain for sickle cell patients presenting to the ED in acute pain crisis. Although regional anesthesia has become more established in trauma settings, its use in chronic pain populations—particularly those with sickle cell disease—remains underexplored. This case supports the feasibility, safety, and efficacy of genicular nerve blocks in the ED, especially when standard opioid-based regimens are limited by side effects or insufficient relief. Further investigation is warranted to assess its broader utility, optimal timing, and impact on outcomes such as opioid consumption, hospital length of stay, and patient satisfaction. Using regional anesthesia may serve as a meaningful step toward multimodal pain control and improved care for a historically undertreated and vulnerable population.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Rosemary Beranek, DO, Hennepin County Medical Center, Department of Emergency Medicine, 730 S 8 St., Minneapolis, MN 55460. Email: rose.beranek@hcmed.org.

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Focal Hepatic Hot Spot Sign Mimicking Active Extravasation on CT Angiography in Superior Vena Cava Syndrome: A Case Report

R. Gentry Wilkerson, MD* *University of Maryland School of Medicine, Department of Emergency Medicine, Baltimore, Maryland
Vanesa Brecher, BS† †Philadelphia College of Osteopathic Medicine, Philadelphia, Pennsylvania

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Introduction: Caused by portosystemic venous shunting, the focal hepatic hot spot sign (or hot quadrate sign) is a radiologic finding most commonly associated with superior vena cava (SVC) obstruction. On contrast-enhanced imaging, this finding may mimic active hepatic extravasation or focal hepatic pathology, posing a diagnostic challenge in the emergency department (ED).

Case Report: We report a case of a 70-year-old man with progressive cognitive decline and hypoxemic respiratory failure requiring intubation. Computed tomography (CT) angiography of the chest demonstrated a focal area of intense contrast enhancement in the left hepatic lobe, initially concerning for active extravasation. Further review of the imaging revealed a right upper lobe lung mass and a large mediastinal mass causing high-grade SVC stenosis. The hepatic finding was subsequently identified by the reading radiologist as a focal hepatic hot spot sign representing a vascular pseudolesion due to collateral venous flow. The patient was diagnosed with SVC syndrome secondary to malignancy and underwent chemotherapy before transitioning to comfort care.

Conclusion: The focal hepatic hot spot sign represents a hepatic pseudolesion resulting from altered venous return and collateral circulation, most commonly in the setting of SVC obstruction. In the ED, this finding may be misinterpreted as hemorrhage or focal hepatic pathology, potentially leading to unnecessary diagnostic or therapeutic interventions. Recognition of the focal hepatic hot spot sign is essential for emergency physicians to avoid misdiagnosis and identify underlying central venous obstruction. Awareness of this imaging pitfall can improve diagnostic accuracy and guide appropriate management in patients undergoing contrast-enhanced CT imaging. [Clin Pract Cases Emerg Med. 2026;10(4):628–631.]

Keywords: *focal hepatic hot spot sign; hot quadrate sign; superior vena cava syndrome; hepatic pseudolesion; case report.*

INTRODUCTION

The focal hepatic hot spot sign (or hot quadrate sign) is a radiologic finding characterized by focal enhancement in segment IV of the liver due to portosystemic venous shunting.[1] Although most commonly associated with superior vena cava (SVC) obstruction, it may be misinterpreted on contrast-enhanced imaging as active hepatic extravasation or focal hepatic pathology.[2] Given the widespread use of computed tomography (CT) angiography in the emergency department (ED) for evaluation of undifferentiated patients, emergency physicians are increasingly likely to encounter incidental hepatic findings, making familiarity with this imaging finding essential to avoid diagnostic error. We present a case in which this finding initially raised concern for intra-abdominal hemorrhage but ultimately led to the diagnosis of SVC syndrome from an underlying malignancy.

CASE REPORT

A 70-year-old man with a past medical history of

hypertension, coronary artery disease with previous coronary artery stent placement, ischemic cardiomyopathy with a previously documented ejection fraction of 25% to 30%, chronic obstructive pulmonary disease, and continued heavy cigarette smoking was brought in by family members due to worsening cognitive decline and frequent falls over the previous month. The family reported a fall 3 weeks prior to ED presentation with persistent difficulty with moving his left arm since the fall.

At triage, his vital signs were as follows: blood pressure, 127/84 mm Hg; heart rate, 82 beats per minute; respiratory rate, 16 breaths per minute; temperature, 36.3 °C; and oxygen saturation level, 97% on room air. He was somnolent with snoring respirations. The rest of his cardiopulmonary exam was unremarkable. There was 2+ pitting edema of the bilateral lower extremities. He would waken briefly to strong tactile stimuli and was able to follow brief, simple commands, but he was not able to speak. There was no obvious focal neurologic deficit. During the patient's ED visit, he became increasingly hypoxic requiring oxygen supplementation by nasal cannula and then high-flow nasal cannula, and eventually intubation with mechanical ventilation.

A CT angiogram of the chest demonstrated a focal area of intense contrast enhancement within the medial aspect of the left hepatic lobe (Image 1), initially raising concern by the clinical team for active contrast extravasation. Additional review of the imaging revealed a 3.6 × 2.8 cm right upper lobe lung mass in the setting of fibrocystic and emphysematous lung changes, as well as a large 6.8 × 6.9 × 6.9 cm mediastinal mass causing significant mass effect on the right main pulmonary artery and SVC (Image 2), resulting in high-grade stenosis and findings consistent with SVC syndrome. The focal hepatic enhancement was subsequently identified by the radiologist as a vascular pseudolesion representing collateral venous flow to segment IV of the liver, consistent with the focal hepatic hot spot sign. The patient underwent a 3-day induction of chemotherapy with atezolizumab/carboplatin/etoposide. Due to persistence of acute hypoxemic respiratory failure, the patient's family elected to change the patient's code status to "do not resuscitate" and made him comfort care measures only. The patient was transferred to an inpatient hospice unit on hospital day 14.

CPC-EM Capsule

What do we already know about this clinical entity?
The focal hepatic hot spot sign is a vascular pseudolesion due to portosystemic shunting, most commonly associated with superior vena cava (SVC) obstruction.

What makes this presentation of disease reportable?
The focal hepatic hot spot sign mimicked active extravasation on computed tomography angiography and contributed to recognition of malignant SVC obstruction.

What is the major learning point?
Intense enhancement of hepatic segment IV can reflect collateral venous flow from SVC obstruction rather than hepatic hemorrhage or a true liver lesion.

How might this improve emergency medicine practice?
Recognizing this sign can prevent unnecessary intervention and prompt evaluation for potentially life-threatening central venous obstruction.



Image 1. Axial contrast-enhanced computed tomography angiography demonstrates a focal area of intense hyperenhancement within segment IV of the liver (white arrow), initially concerning for active contrast extravasation.

DISCUSSION

The focal hepatic hot spot sign (or hot quadrate sign) is a radiologic finding characterized by focal hyperenhancement of segment IV of the liver (the quadrate lobe) during arterial and early portal venous phases of contrast-enhanced imaging.[1] This finding represents a hepatic pseudolesion caused by altered venous return and portosystemic shunting, most commonly in the setting of SVC obstruction.[3] This finding has also been described on technetium-99m sulfur colloid liver scans, further supporting its underlying mechanism of altered hepatic perfusion due to portosystemic shunting.[2]

In cases of SVC obstruction, contrast injected into the upper extremity is diverted through collateral venous

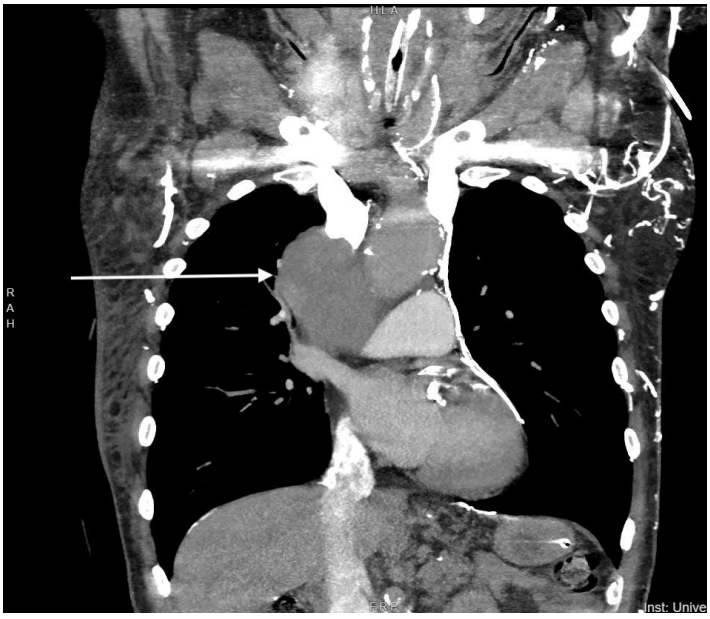


Image 2. Coronal contrast-enhanced computed tomography demonstrates a large mediastinal mass (arrow) causing significant compression of the superior vena cava (SVC), resulting in SVC syndrome and the development of collateral venous pathways responsible for the focal hepatic hot spot sign.

pathways, including the internal thoracic and paraumbilical veins, ultimately reaching the liver via the veins of Sappey and preferentially perfusing the left hepatic lobe.[3] The resulting localized increase in contrast delivery produces the characteristic focal enhancement seen on CT. Recognition of this phenomenon is essential because it reflects underlying central venous pathology rather than intrinsic hepatic disease. Hepatic pseudolesions related to altered venous drainage may be present in up to 20% of contrast-enhanced CT studies, although the focal hepatic hot spot sign represents a specific and clinically meaningful subtype.[4]

In the ED, the focal hepatic hot spot sign is particularly important because it may be misinterpreted as active contrast extravasation or focal hepatic pathology on contrast-enhanced CT, especially when identified incidentally during evaluation for other acute conditions.[2] Such misinterpretation may prompt unnecessary diagnostic workup, invasive procedures, or inappropriate clinical management. Recognition of this pseudolesion should instead prompt careful evaluation for central venous obstruction, particularly SVC syndrome, which may present with subtle or nonspecific findings early in its course. Classic clinical findings of SVC syndrome include facial and upper extremity swelling, dyspnea, and the presence of the Pemberton sign, in which elevation of the arms results in facial plethora and venous distention; however, these findings may be absent early in the disease process.[5] In this case, the initial concern for hepatic extravasation was redirected after identification of mediastinal pathology causing significant SVC compression. Recognition of this imaging sign allows emergency physicians to appropriately redirect diagnostic evaluation, avoid unnecessary interventions, and expedite identification of the underlying etiology.

The differential diagnosis for focal hepatic hyperenhancement on contrast-enhanced CT includes both true hepatic lesions and pseudolesions. Entities such as hemangioma, focal nodular hyperplasia, and hepatocellular carcinoma may demonstrate hyperattenuation and can mimic this appearance, particularly when imaging is obtained during arterial phase acquisition.[6] However, unlike true lesions, the focal hepatic hot spot sign does not produce mass effect and is typically localized to segment IV near the falciform ligament. Correlation with clinical findings and careful evaluation for associated features of central venous obstruction, such as mediastinal masses, venous collateralization, or SVC narrowing, can aid in distinguishing this pseudolesion from true pathology. Recognition of these distinguishing features is critical in the emergency setting, where rapid interpretation of imaging findings directly impacts diagnostic accuracy and subsequent management decisions.

CONCLUSION

The focal hepatic hot spot sign is an important imaging finding that may be encountered incidentally on contrast-enhanced CT in the ED. Recognition of this pseudolesion is critical to avoid misdiagnosis as hepatic hemorrhage or focal liver pathology. In the appropriate clinical context, this finding should prompt evaluation for central venous obstruction, including SVC syndrome. For emergency physicians, recognition of this sign can prevent misdiagnosis, avoid unnecessary intervention, and serve as an early imaging clue to life-threatening central venous obstruction.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: R. Gentry Wilkerson, MD, University of Maryland School of Medicine, Department of Emergency Medicine, University of Maryland School of Medicine, 110 South Paca Street, 6th Floor, Suite 200, Baltimore, MD 21201. Email: gwilkerson@som.umaryland.edu.

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Drug-Induced Immune Thrombocytopenia Caused by Sulfamethoxazole-Trimethoprim: A Case Report

Alec Powell, DO
Louisa Weindruch, DO
Jonathan Thornton, MD

Baylor University Medical Center, Department of Emergency Medicine, Dallas, Texas

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Introduction: Drug-induced immune thrombocytopenia (DITP) is a rare process in which specific drugs induce antibodies against platelet antigens, resulting in the destruction, consumption, or impaired production of platelets.[1,3] Sulfamethoxazole-trimethoprim can result in DITP.[2]

Case Report: This is a case of a 64-year-old man who developed severe thrombocytopenia after sulfamethoxazole-trimethoprim use. He presented to the emergency department with oral hemorrhagic lesions, mucositis, and petechiae. Laboratory evaluation revealed a low platelet count. Drug-induced immune thrombocytopenia was suspected, and the sulfamethoxazole-trimethoprim was discontinued. The patient's platelet count responded rapidly after discontinuation of the offending drug and administration of dexamethasone.

Conclusion: This case highlights the importance of early recognition of DITP and the avoidance of re-exposure to the causative agent. Prompt drug withdrawal and adjunctive corticosteroid therapy can facilitate complete clinical recovery. [Clin Pract Cases Emerg Med. 2026;10(4):632–634.]

Keywords: *drug-induced immune thrombocytopenia; thrombocytopenia; sulfamethoxazole-trimethoprim; mucosal bleeding; case report.*

INTRODUCTION

Drug-induced immune thrombocytopenia (DITP) is caused by drug-specific antibodies that develop against platelet antigens, most commonly the glycoprotein IIb/IIIa complex (GPIIb/IIIa) and the glycoprotein Ib/IX complex (GPIb/IX),[1] which can result in suppression of hematopoiesis, megakaryocytopoiesis inhibition, platelet destruction, and platelet consumption.[1,3] This potentially life-threatening hematologic condition can be caused by drugs such as including quinine, carbamazepine, vancomycin, eptifibatide, sulfamethoxazole, and trimethoprim.[2] We present a case of sulfamethoxazole-trimethoprim–induced thrombocytopenia presenting with oral mucosal bleeding, mucositis, petechiae, and a critically low platelet count in a patient who had a similar presentation with a prior exposure. This case highlights a rare complication of a commonly prescribed medication and demonstrates the importance of prompt recognition in the emergency department (ED) setting.

CASE REPORT

A 64-year-old man with type 2 diabetes mellitus, hypertension, hyperlipidemia, and chronic urinary retention presented to the ED with several hemorrhagic oral lesions and a rash on his bilateral lower extremities. Four days prior to presentation, his chronic indwelling Foley catheter had been removed at an outside hospital. The patient, believing he had developed a urinary tract infection, started taking leftover sulfamethoxazole-trimethoprim belonging to his wife. Within three days, he developed hemorrhagic lesions on his tongue and a petechial rash involving his lower extremities. He reported dark stools two days prior to presentation but denied hematemesis, melena, and hematuria at presentation. He recalled a prior reaction to sulfa drugs that resulted in an

episode of "low blood counts."

The patient was well-appearing, in no acute distress, afebrile, and hemodynamically stable. On physical exam, he had hemorrhagic blisters on the tongue and lips (Image 1), mucositis, and petechiae involving the bilateral lower extremities (Image 2). No scleral icterus, lymphadenopathy, or hepatosplenomegaly was observed.

Laboratory evaluation was most notable for significant thrombocytopenia, with a platelet count of less than 1×10^3 platelets/μL (reference range, 150–400 × 10^3/μL). Hemoglobin and hematocrit were within normal limits.

Dermatology consult found the presentation not to be consistent with Stevens-Johnson syndrome or toxic epidermal necrolysis. The hematology team suspected drug-induced immune thrombocytopenia and recommended dexamethasone 40 mg intravenously (IV) in the ED, followed by daily doses while inpatient. Platelet transfusion and IV immunoglobulin were deferred due to absence of severe symptoms and active bleeding.

During his hospital stay, the patient's platelet count and clinical findings improved rapidly after discontinuation of sulfamethoxazole-trimethoprim (Table). His hospital course was complicated by mild steroid-induced hyperglycemia, which was managed conservatively. The patient's platelet count was adequately responding; therefore, hematology agreed without the decision to withhold the last dose of dexamethasone given the hyperglycemia. The patient was discharged on hospital day five after complete platelet recovery with instructions for repeat labs outpatient one week after discharge.

CPC-EM Capsule

What do we already know about this clinical entity?

Drug-induced immune thrombocytopenia (DITP) is a rare process by which exposure to certain drugs results in production of antibodies against platelet antigens.

What makes this presentation of disease reportable?

We present a case of sulfamethoxazole-trimethoprim–induced thrombocytopenia presenting with oral mucosal bleeding and petechiae.

What is the major learning point?

Several medications can cause DITP and lead to significant thrombocytopenia. Early recognition, treatment, and avoidance of re-exposure is crucial.

How might this improve emergency medicine practice?

This case highlights a rare complication of a commonly prescribed medication and demonstrates the importance of prompt recognition in the emergency care setting.

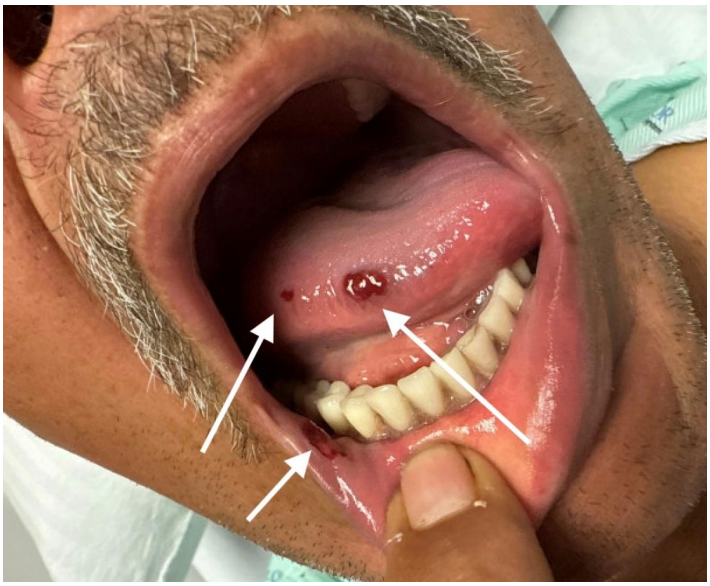


Image 1. Hemorrhagic blisters seen on patient's buccal mucosa and lateral tongue (arrows).

DISCUSSION

Sulfamethoxazole–trimethoprim-induced immune thrombocytopenia is a well-known cause of DITP; use of other medications such as quinine, quinidine, vancomycin, penicillin, rifampin, ibuprofen, mirtazapine, oxaliplatin, suramin, carbamazepine, and ceftriaxone can cause this condition.[4] A study by Kaufman et al reported 38 cases of DITP per million users of sulfamethoxazole-trimethoprim.[5] It is most commonly caused by the production of immunoglobulin G antibodies that bind to platelet antigens, specifically GPIIb/IIIa and GPIb/IX.[1]

Thrombocytopenia is defined as a platelet count of less than 150 × 10^3/μL[2]; however, in DITP, the platelet nadir is frequently less than 20 ×10^3/μL.[1] Onset typically occurs 5 to 10 days following drug exposure, resulting in the clinical manifestations of thrombocytopenia.[1] Classic clinical signs include purpuric lesions on the skin and mucous membranes, hematuria, and gastrointestinal hemorrhage.[3] Diagnostic confirmation is aided by platelet recovery after discontinuation and recurrence upon accidental re-exposure. In this patient, acute platelet reduction manifested shortly after initiating sulfamethoxazole-trimethoprim, consistent with a documented

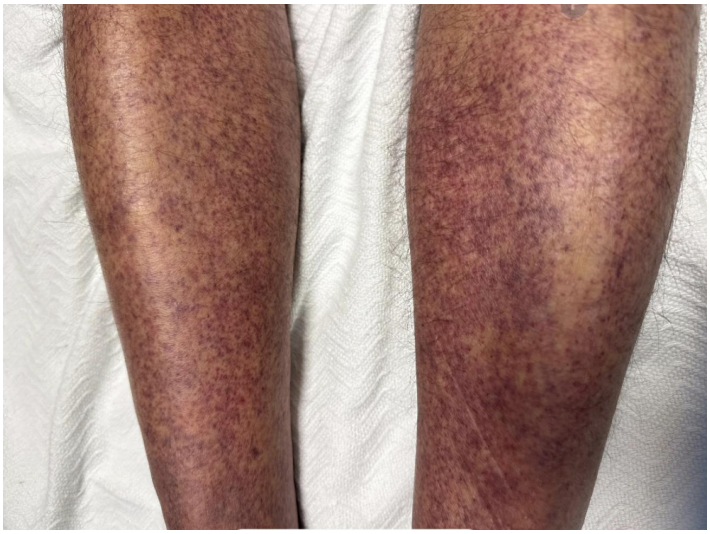


Image 2. Extensive petechial rash on the patient's lower extremities.

historical episode following exposure to the same agent.

When the specific causative agent cannot be definitively identified, discontinuation of all nonessential medications is warranted.[1] As platelet count improves, the symptoms of thrombocytopenia will begin to subside, usually within 1 to 2 days (4 to 5 half-lives) after stopping the medication.[3] Steroids can be used as an adjunct to increase the rate of platelet recovery. Intravenous immunoglobulin (IVIG) and platelet transfusions are typically reserved for severe bleeding, such as intracranial or intrapulmonary hemorrhage, or for persistent thrombocytopenia.[1,3] Platelet transfusion may not show any benefit if the drug antibodies are still in the patient's system.[1]

Table. Platelet count and hospital course.

Hospital Day	Platelet Count ($\times 10^3/\mu L$)	Clinical Status
1	Less than 1	Oral mucosal hemorrhagic lesions, petechiae
2	11	Oral lesions improving
3	43	Petechiae resolving
4	85	Continued improvement
5	163	Complete recovery

μL, microliters.

CONCLUSION

This case demonstrates the clinical presentation of drug-induced immune thrombocytopenia and the recovery process, focusing on the rate of recovery and treatments that may accelerate it. It is important for clinicians to recognize drug-induced immune thrombocytopenia and consider new medications as possible causes. Immediate cessation of the offending agent is the primary therapeutic intervention for DITP. Steroids, IVIG, and platelets may serve as beneficial adjuncts depending on the clinical picture.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Louisa Weindruch, DO, Baylor University Medical Center, Department of Emergency Medicine, 3500 Gaston Ave, Dallas, TX 75246. Email: Louisa.Weindruch@bswhealth.org.

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Nitrous Oxide "Whippet" Induced Subacute Combined Degeneration of the Spinal Cord: A Case Report

Joshua Kern, MD
Carolyn Shanks, MD
University of Texas-Southwestern, Department of Emergency Medicine, Division of Medical Toxicology, Dallas, Texas

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Introduction: Subacute combined degeneration (SCD) is an uncommon neurologic complication of vitamin B12 deficiency characterized by demyelination of the dorsal and lateral columns of the spinal cord, resulting in weakness, paresthesias, sensory deficits, and gait ataxia. Recreational nitrous oxide inhalation has emerged as an important cause of functional vitamin B12 deficiency and may precipitate SCD, particularly in otherwise healthy individuals.

Case Report: We report a case of nitrous oxide-associated SCD in a patient who presented to the emergency department with progressive neurologic symptoms. This case illustrates a rare but increasingly recognized complication of recreational nitrous oxide use.

Conclusion: Nitrous oxide-induced SCD should be considered in patients presenting with subacute weakness, paresthesia, and ataxia, particularly when there is a history of recreational inhalant use. Early recognition and treatment with intramuscular vitamin B12 replacement, together with cessation of nitrous oxide exposure, are essential to optimize neurologic recovery. [Clin Pract Cases Emerg Med. 2026;10(4):635–639.]

Key Words: *subacute combined degeneration; vitamin B12 deficiency; nitrous oxide; myelopathy; emergency medicine.*

INTRODUCTION

Inhaled nitrous oxide (N_2O) has emerged as an increasingly prevalent substance for recreational use. Historically, it was used in medical settings (including dentistry) for its quick anesthetic effects. Commonly known as "laughing gas," N_2O produces a brief period (approximately 20 seconds) of euphoria, contributing to both its therapeutic appeal and its misuse.[1] Beyond healthcare, N_2O is also a popular food additive, functioning as a propellant for certain foods, including whipped cream.[1] Recreational use of N_2O is not a recent phenomenon. "Nitrous oxide frolics" date back to the early 1800s, just years after its initial discovery. Abuse was particularly common among medical professionals who used N_2O in their practices.[2]

Since 2000, recreational N_2O use has become more widespread.[3] It is commonly obtained from steel cartridges filled with compressed N_2O used for preparation of whipped cream. When used in this manner, it is colloquially referred to as whippets, nitro, nangs, or balloons. These N_2O canisters are easy and legal to obtain, which could explain the popularity and growing use, especially among the younger population.[3] The 2019 Global Drug Survey, which gathers data from self-selected drug users from over 30 countries, found that 91% of those surveyed (n = 123,814) had used N_2O at least once in their lifetime.[4]

When used in a controlled hospital setting, N_2O is remarkably safe.[5] Vitamin B12 deficiency is rare in healthy individuals, as hepatic stores of vitamin B12 are typically sufficient to last years. However, certain populations are at increased risk, including those with decreased dietary intake of vitamin B12 (such as those following vegan diets) and those with malabsorptive issues such as pernicious anemia, atrophic gastritis, or ileal resection.[5]

Nitrous oxide can cause vitamin B12 (cyanocobalamin)

deficiency secondary to oxidation and inactivation of the vitamin B12 enzyme. Nitrous oxide oxidizes a cobalt ion on the vitamin B12 coenzyme, which converts it from an active monovalent form (CO^{+}) to an inactive divalent form (CO^{2+}); this irreversibly inhibits its enzymatic activity. Furthermore, the cobalt moiety of vitamin B12 functions as an essential methyl carrier in the formation of methionine from homocysteine and 5-methyltetrahydrofolate. Inactivation of this pathway impairs methionine synthesis, leading to downstream inhibition of DNA synthesis and myelin production, which underlies the hematologic and neurologic manifestations of N_2O toxicity[6] (Figure).

Subacute combined degeneration (SCD) of the spinal cord often presents with progressively worsening neurologic deficits, including impaired vibratory sense and proprioception, ataxia, sensory loss, weakness, and spastic quadriparesis. These manifestations are likely secondary to demyelinating effects on the lateral corticospinal tracts and dorsal column of the spinal cord.[8] Cognitive decline can also be appreciated, which is often reversible with timely recognition and treatment.[8]

Recreational use of N_2O can also cause hematologic effects and bone marrow suppression, secondary to the effects of vitamin B12 deficiency and impaired methionine synthesis.[6] Laboratory studies often reveal a macrocytic anemia with elevated homocysteine and methylmalonic acid (MMA) levels.[8] Magnetic resonance imaging (MRI) of the entire spine is recommended when SCD is suspected, which can show characteristic T2 hyperintensity of the posterior (dorsal) columns in up to 68% of affected patients.[9] Despite common clinical involvement of the lateral spinal cord tracts, corresponding radiographic abnormalities in this region are uncommon.[8]

The use of N_2O has been linked to severe neurologic injury and death. However, these outcomes are thought to result predominantly from hypoxic and asphyxiation during inhalation, rather than secondary to direct neurotoxic effects of N_2O.[10]

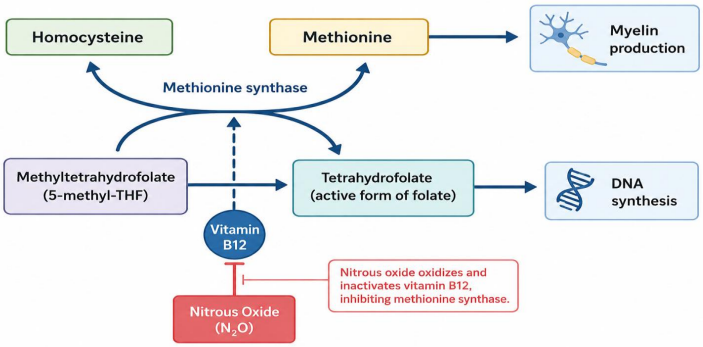


Figure. Depiction of vitamin B12 biochemistry.

CPC-EM Capsule

What do we already know about this clinical entity?
Recreational nitrous oxide use can inactivate vitamin B12, causing subacute combined degeneration with progressive weakness, sensory loss, and ataxia.

What makes this presentation of disease reportable?
Severe neurologic dysfunction with unrevealing spinal magnetic resonance imaging (MRI), absent macrocytosis, and bilateral pulmonary emboli highlights an easily missed diagnosis.

What is the major learning point?
Nitrous oxide-induced myelopathy can occur without characteristic MRI findings or macrocytosis; exposure history and biochemical testing are key to diagnosis.

How might this improve emergency medicine practice?
Targeted questioning about nitrous oxide use and early B12 testing can expedite treatment and cessation counseling, potentially reducing lasting disability.

CASE REPORT

A 27-year-old man with a history of prior substance use disorder, including marijuana and N_2O ("whippets"), presented to the emergency department (ED) after his father requested a welfare check by emergency medical services. According to his father, the patient had experienced 3 days of progressively worsening mentation and had become bedridden. On presentation, he was unable to provide a meaningful history secondary to his altered mentation, and he reported only left thigh pain. Additional history obtained from the patient's father revealed that his son had recently been admitted to an outside hospital due to a subacute inability to walk. During that admission, he had an extensive workup including MRI and lumbar puncture, but no definitive diagnosis was made.

Initial vital signs were notable for the following: tachycardia (heart rate, 108 beats per minute); hyperthermia (37.8 °C); hypotension (78/50 mm Hg); and hypoxemia with SpO_2 in the upper 80% range on room air. Examination was notable for an ill-appearing patient, covered in feces and urine. He was oriented only to self and year but was otherwise

unable to answer questions. Neurologic examination demonstrated marked bilateral lower extremity weakness; sensation was difficult to assess secondary to his neurologic status. No clonus or rigidity was appreciated on exam. Lower extremity reflexes were brisk. His abdomen was distended and tender in the lower region. He had 2+ pitting edema in the bilateral lower extremities.

A broad workup was initiated. Point-of-care bladder ultrasonography revealed significant urinary retention with greater than 2 L of fluid in his bladder, for which a Foley catheter was placed. Imaging studies were obtained including computed tomography (CT) of the head, CT of the thoracic spine, CT of the lumbar spine, CT of the abdomen and pelvis, and CT angiography (CTA) of the chest. Findings on the CTA of the chest revealed acute pulmonary emboli in the bilateral lungs, as well as a consolidation in the left lower lung lobe. Computed tomography of the head revealed mild generalized volume loss, advanced for the patient's age. An MRI lumbar spine demonstrated no acute findings.

Laboratory workup revealed a mild normocytic anemia with a hemoglobin of 12.3g/dL (reference range, 13.2–16.9 g/dL) and a mean corpuscular volume of 88.9 fL (80–100 fL). There was no leukocytosis or thrombocytopenia. Serum creatinine was elevated to 1.73 mg/dL (0.67–1.17 mg/dL), with a creatine kinase elevation to 1,888 U/L (39–308 U/L), which eventually peaked at 2,827 U/L. Thyroid-stimulating hormone was within normal limits. Urine toxicology was positive for cannabinoid use, while the serum ethanol level was undetectable. Further evaluation revealed an undetectable vitamin B12 level. A subsequent homocysteine and methylmalonic acid were obtained; homocysteine and MMA levels were markedly elevated at 60.6 µmol/L (5–15 µmol/L) and 15.93 µmol/L (< 0.04 µmol/L), respectively (Table).

Resuscitative measures were initiated, including intravenous fluids and empiric antibiotic therapy. A heparin drip was started. The patient was subsequently admitted to the hospital. The neurology team agreed that this case was suspicious for SCD secondary to whippet use. Additional MRI studies of the brain, cervical spine, and thoracic spine were obtained; however, no definitive radiographic abnormalities were identified (Image 1 and 2).

Table. Initial laboratory studies in a patient presenting with altered mental status.

Laboratory value	Value on admission	Reference value
Hemoglobin	12.3 (g/dL)	13.2–16.9 (g/dL)
Mean corpuscular volume	88.9 (fL)	80–100 (fL)
Creatinine	1.73 (mg/dL)	0.67–1.17 (mg/dL)
Creatinine kinase	1,888 (U/L)	39–308 (U/L)
Vitamin B12	< 150 (pg/mL)	180–914 (pg/mL)
Homocysteine	60.6 (µmol/L)	5–15 (µmol/L)
Methylmalonic acid	15.93 (µmol/L)	0–0.04 (µmol/L)

dL, deciliter; *fL*, ; *g*, grams; *L*, liter; *mg*, milligram; *mL*, mililiter; *µmol*, micromoles; *pg*, picogams; *U*, units.

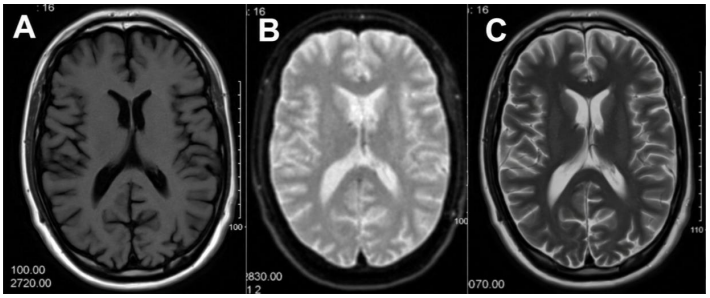


Image 1. Magnetic resonance imaging of the brain: (**A**) T1 flair; (**B**) diffusion-weighted imaging; (**C**) T2 blade. No acute abnormalities appreciated.

The patient was started on aggressive vitamin B12 repletion, starting with intramuscular (IM) vitamin B12 1,000 mcg daily for 7 days, followed by IM vitamin B12 1,000 mcg weekly for 4 weeks. Subsequently, he was transitioned to oral vitamin B12 1,000 mcg daily for ongoing maintenance therapy. Over the course of his hospitalization, the patient demonstrated gradual neurologic improvement, and he was discharged to an inpatient rehabilitation facility for continued recovery.

DISCUSSION

Recreational N_2O use and its neurologic complications are increasingly recognized.[4] Many of the neurologic and hematologic complications arise from deficiencies in vitamin B12, which are induced from N_2O use. Diagnosing use in the ED can be challenging, as there are no readily available screening tests to confirm exposure. Additionally, patients may

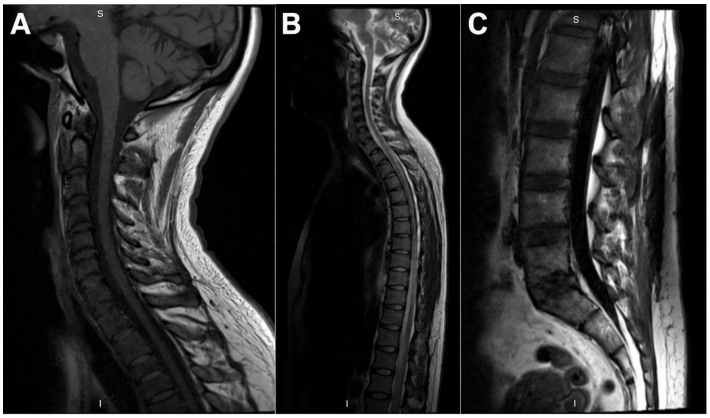


Image 2. Magnetic resonance imaging: (**A**) cervical, (B) thoracic, and (**C**) lumbar. No acute abnormalities appreciated. Magnetic resonance imaging did not reveal the characteristic T2 hyperintensity of the posterior (dorsal) columns usually seen in patients with subacute combined degeneration.

not disclose their use, or they may be unable to provide an accurate history due to altered mental status.

Neuroimaging, particularly MRI, can support the diagnosis of N_2O-induced SCD. Characteristic findings include T2-weighted hyperintensities in the posterior and lateral columns of the spinal cord, reflecting involvement of the dorsal columns and corticospinal tracts.[7,11] These abnormalities are most commonly seen in the cervical region, followed by the thoracic region.[7,11] Brain MRI is often unrevealing, with no abnormalities identified in most cases.[11] Importantly, the absence of radiographic findings does not exclude the diagnosis; characteristic imaging findings are only seen in approximately 68% to 78% of diagnosed cases.[9]

Certain laboratory studies can further support the diagnosis. Patients will often have a macrocytic anemia, although this finding is not universally present. Patients will also likely have low serum vitamin B12 levels, as well as an elevated homocysteine and methylmalonic level.[8] Importantly, vitamin B12 levels may be falsely normal or even elevated in some SCD patients. Obtaining homocysteine and methylmalonic levels can be helpful for diagnosis when clinical suspicion remains high.[8]

Our patient also presented with a concurrent diagnosis of acute pulmonary embolism. Although this may initially appear to have been unrelated, a potential mechanistic link exists through the prothrombotic effects of elevated homocysteine levels. It is well-established that elevated homocysteine levels are associated with thrombotic diseases through a variety of mechanisms including endothelial dysfunction, oxidative stress, impaired anticoagulant pathways, and promotion of coagulation activation.[15]

The primary treatment for SCD secondary to N_2O use is aggressive vitamin B12 replacement. Although recommendations vary, it is reasonable to start with 1,000–2,000 mcg of daily IM vitamin B12 for 1 week, followed by 1,000 mcg IM vitamin B12 weekly for 4 to 9 weeks.[6,12] After this initial repletion, patients should continue maintenance therapy with daily oral administration of 1,000–2,000 mcg of B12 supplementation until resolution of symptoms.[6,12] Prognosis is generally favorable with prompt recognition and treatment, although the degree of neurologic recovery is variable. A 2022 review of 96 patients found that approximately 75% experienced significant improvement in their neurologic symptoms after appropriate vitamin B12 supplementation.[13] Another review found that 86% of patients had significant improvement, but only 14% achieved complete resolution of their symptoms.[14]

Several factors have been associated with improved neurologic outcomes, including younger age at presentation (< 50 years), shorter duration of symptoms, absence of sensory deficits, absence of a positive Romberg sign, absence of a positive Babinksi sign, involvement of fewer spinal segments on MRI (fewer than 7), presence of spinal cord edema, and absence of spinal cord atrophy.[14]

Additional supplementation with methionine may be beneficial, as methionine is a direct substrate for methionine synthase.[14] In addition to medical therapy, physical therapy and rehabilitation plays an essential role in recovery.[12] Finally, sustained abstinence from N_2O exposure is also critical to prevent recurrence of neurologic injury.

CONCLUSION

This case report details the diagnosis, management, and prognosis of subacute combined degeneration secondary to recreational use of nitrous oxide. As recreational N_2O becomes more prevalent, clinicians must recognize this physiology and maintain a high index of suspicion in patients with unexplained neurologic symptoms. Early recognition and intervention are essential to preventing long-term neurologic sequelae.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Joshua Kern, MD, Universtiy of Texas-Southwestern, Department of Emergency Medicine, 5323 Harry Hines Blvd. E4.300, Dallas, TX 75390. Email: Joshua.kern@UTSouthwestern.edu.

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Carotid Artery Dissection After Cervical Traction Device Use: A Case Report

Kian D. Samadian, MD[*†‡††]
Maureen K. Nothnagle, PA-C, MPH[*‡]
Sina M. Mostaghimi, MD[*‡§]
Sandra Alhoyek, MD[*‡¶]
Rehmat Babar, MD[‡‖**]
Shuhan He, MD[*‡§]

*Massachusetts General Hospital, Department of Emergency Medicine, Boston, Massachusetts
†Brigham and Women's Hospital, Department of Emergency Medicine, Boston, Massachusetts
‡Harvard Medical School, Boston, Massachusetts
§Massachusetts Eye and Ear, Department of Emergency Medicine, Boston, Massachusetts
¶Massachusetts Eye and Ear, Department of Ophthalmology, Boston, Massachusetts
‖Massachusetts General Hospital, Department of Neurology, Boston, Massachusetts
**Brigham and Women's Hospital, Department of Neurology, Boston, Massachusetts
††Present affiliation: University of Pennsylvania, Department of Emergency Medicine, Philadelphia, Pennsylvania

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Introduction: Internal carotid artery (ICA) dissection is a time-sensitive vascular emergency that may present with subtle findings, most notably painful Horner syndrome. Mechanical stress to the cervical vasculature is a recognized trigger for this condition.

Case Report: We report a 56-year-old man who developed ipsilateral ptosis, miosis, and head and neck pain after using a home cervical traction device. Computed tomography angiography showed focal critical stenosis of the distal cervical left ICA consistent with dissection, along with a contralateral pseudoaneurysm suggesting prior injury. He was treated with dual antiplatelet therapy.

Conclusion: This case highlights a potential association between cervical traction device use and carotid dissection and underscores the importance of recognizing painful Horner syndrome and bilateral carotid pathology, while prompting clinicians to specifically inquire about traction device use. [Clin Pract Cases Emerg Med. 2026;10(4):640–643.]

Keywords: *internal carotid artery dissection; Horner syndrome; cervical traction device; carotid artery injury; case report.*

INTRODUCTION

Carotid artery dissection is an important cause of stroke in adults younger than 60 years and frequently presents initially to the emergency department (ED).[1-6] Internal carotid artery (ICA) dissection may manifest with cerebral ischemia, but a substantial subset of patients presents with isolated local symptoms, most commonly unilateral head or neck pain and partial Horner syndrome. Horner syndrome results from disruption of the oculosympathetic pathway. In the setting of ICA dissection, the lesion typically affects sympathetic nerve pathways traveling along the carotid artery, producing ipsilateral ptosis and miosis without facial anhidrosis. Painful Horner syndrome is classically associated with carotid dissection and should prompt urgent vascular imaging. Mechanical forces applied to the cervical spine, including manipulation and traction, can generate shear stress along the carotid artery.

Home cervical traction devices are widely marketed for spinal alignment and neck pain relief and apply sustained axial distraction forces. The vascular safety profile of these devices

remains poorly characterized. This report aims to describe a case of ICA dissection presenting as painful Horner syndrome following use of a cervical traction device and to highlight key diagnostic considerations for emergency clinicians. This case report was prepared in accordance with the CARE (CAse REport) guidelines.[7] Written informed consent was obtained from the patient for publication of this case report and accompanying images.

CPC-EM Capsule

What do we already know about this clinical entity?
Carotid artery dissection is a cause of painful Horner syndrome and ischemic stroke, often following trauma or cervical manipulation.

What makes this presentation of disease reportable?
This case links home cervical traction device use with carotid artery dissection, an uncommon and underrecognized mechanism of vascular injury.

What is the major learning point?
Painful Horner syndrome after cervical traction should prompt urgent vascular imaging to evaluate for carotid artery dissection.

How might this improve emergency medicine practice?
Recognizing cervical traction as a risk factor may expedite diagnosis, treatment, and stroke prevention in patients with subtle neurologic findings.

CASE REPORT

A 56-year-old man with a history of benign prostatic hyperplasia, gastroesophageal reflux disease, asthma, allergic rhinitis, and cataracts was transferred from an ophthalmologic ED after computed tomography angiography (CTA) of the head and neck demonstrated a left ICA dissection during evaluation of a headache and drooping of the left eyelid. Approximately two weeks before presentation, the patient used an adjustable cervical neck traction device intended for spinal alignment and neck pain relief. Immediately after performing a cervical traction stretch, he developed a new left-sided headache. Over the ensuing week, he noted intermittent left posterior head and neck pain and progressive swelling and drooping of his left eyelid. He denied focal weakness, numbness, dysarthria, dysphagia, aphasia, or visual loss.

Three days before transfer, he was evaluated at an ophthalmologic ED, where ptosis was not documented. He returned three days later because of persistent left eyelid drooping. He denied high-velocity chiropractic manipulation or major trauma. On arrival to the ED, vital signs were notable for the following: blood pressure, 161/95 mm Hg; heart rate, 67 beats per minute; and peripheral oxygen saturation, 100% on room air. He was afebrile. On presentation, the patient was well-appearing and in no acute distress. Head, ears, nose, and throat examination was unremarkable, with an atraumatic head, a patent oropharynx, and normal tympanic membranes bilaterally. Ophthalmologic examination demonstrated anisocoria, with the left pupil smaller than the right, although both pupils were round and reactive to light. Administration of 0.5% apraclonidine ophthalmic drops resulted in reversal of the anisocoria with dilation of the left pupil and improvement in ptosis, confirming sympathetic denervation consistent with Horner syndrome. Cardiovascular examination revealed a regular rate and rhythm, and the lungs were clear to auscultation bilaterally. The abdomen was soft, nondistended, and nontender.

Neurologic examination demonstrated that the patient was alert and oriented with fluent speech and no evidence of dysarthria or aphasia. Cranial nerves II through XII were intact aside from the left-sided ptosis and miosis noted on ophthalmologic examination. Motor strength was full throughout, sensation was intact to light touch, finger-to-nose testing was normal bilaterally, and there was no pronator drift. Gait was normal.

Computed tomography angiography of the head and neck demonstrated short-segment luminal irregularity with focal critical stenosis in the distal cervical left ICA at the skull base, consistent with dissection, and a 0.9-cm pseudoaneurysm in the distal cervical right ICA, likely reflecting prior arterial injury (Images 1 and 2).

The patient reported surprise that a home neck traction device could be associated with a vascular injury and expressed appreciation for the timely diagnosis and management. He stated that he would avoid similar devices in the future. Neurology recommended initiation of dual antiplatelet therapy with aspirin and clopidogrel. At outpatient neurology follow-up, the patient remained neurologically intact without evidence of ischemic complications. His symptoms were stable, and he continued dual antiplatelet therapy. Further vascular imaging and longitudinal follow-up were planned.

DISCUSSION

This case describes ICA dissection presenting as painful Horner syndrome following use of a home cervical traction device. The differential diagnosis of painful Horner syndrome includes pathology involving any portion of the three-neuron oculosympathetic pathway. Central etiologies include lateral

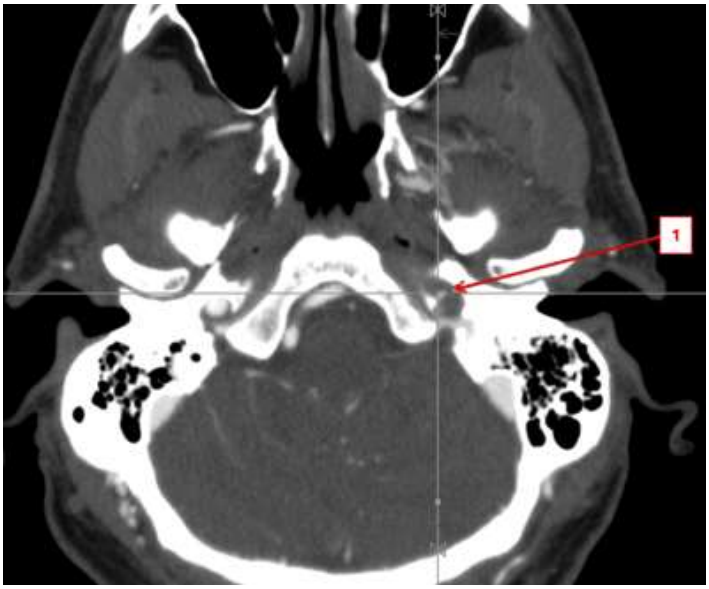


Image 1. Computed tomography angiography of the head and neck demonstrating short-segment luminal irregularity with focal critical stenosis in the distal left internal carotid artery at the skull base (arrow), consistent with dissection.



Image 2. Computed tomography angiography of the head and neck demonstrating a 0.9 centimeter pseudoaneurysm in the distal cervical right internal carotid artery (arrow), likely reflecting prior arterial injury.

medullary infarction, demyelinating disease, and brainstem tumors. Preganglionic lesions include apical lung tumors, mediastinal masses, cervical trauma, and postoperative injury. Postganglionic causes include ICA dissection, carotid artery aneurysm or pseudoaneurysm, cavernous sinus pathology, cluster headache, and orbital tumors. Additional considerations for unilateral ptosis with anisocoria include third cranial nerve palsy, pharmacologic pupillary abnormalities, and physiologic anisocoria. Ipsilateral neck pain and reversal of anisocoria with apraclonidine supported Horner syndrome. The temporal relationship between cervical traction, headache onset, and subsequent ptosis and miosis suggests a mechanically induced vascular injury.

Recognition of Horner syndrome in the ED carries significant diagnostic weight. In carotid dissection, disruption of postganglionic sympathetic fibers along the ICA produces ipsilateral ptosis and miosis without facial anhidrosis. Unilateral head or neck pain with new-onset ptosis or anisocoria should prompt evaluation for carotid dissection.[8-11] Many patients lack focal deficits, and stroke may develop later due to thromboembolism. Early diagnosis allows timely antithrombotic therapy and reduces ischemic risk. Imaging revealed both an acute left ICA dissection and a contralateral 0.9-cm pseudoaneurysm. Bilateral carotid pathology is clinically significant, suggesting either sequential dissections from mechanical stress or underlying arterial vulnerability. The distal cervical ICA near the skull base is particularly susceptible to mechanical stress due to its mobility and tethering. Axial traction may increase shear forces along the arterial wall, leading to intimal injury, intramural hematoma, and pseudoaneurysm formation.[12-13] The lesion location in this case corresponds to a region vulnerable during traction.

Our report complements two recent published reports describing cervical arterial injury after handheld massage gun use and bilateral carotid artery dissection following blunt trauma.[14-15] Unlike these reports, our patient developed ICA dissection after use of a commercially available home cervical traction device without preceding high-energy trauma or chiropractic manipulation. As these devices become increasingly accessible to the public, emergency physicians should recognize home cervical traction as another potential mechanism of cervical vascular injury.

This report has limitations. As a single case, causation cannot be definitively established. However, the temporal sequence, focal dissection at a mechanically susceptible site, and absence of major trauma support a device-related mechanism. The contralateral pseudoaneurysm also raises concern for prior subclinical injury.

CONCLUSION

This case reinforces a key emergency medicine principle: painful Horner syndrome should prompt immediate evaluation for carotid artery dissection. It also highlights a potential association between cervical traction devices and vascular injury. As these devices become more accessible, clinicians

should maintain a high index of suspicion and obtain a targeted history to identify possible mechanical triggers.

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During the preparation of this work the authors used ChatGPT by OpenAI to refine the text of the manuscript and improve clarity. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

The authors attest that their institution does not require Institutional Review Board approval. Written informed consent for publication of this case report and accompanying images was obtained from the patient. Documentation on file.

Address for Correspondence: Kian D. Samadian MD, University of Pennsylvania, Department of Emergency Medicine, 3400 Spruce St, Philadelphia, PA 19104. Email: kian.samadian@pennmedicine.upenn.edu.

Conflicts of Interest: By the *CPC-EM* article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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Use of Point-of-Care Ultrasound to Confirm and Manage a Morel-Lavallée Lesion in the Emergency Department: A Case Report

Siddharth Shanbhag, BS
Ami Kurzweil, MD
Eisenhower Health, Department of Emergency Medicine, Rancho Mirage, California

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Introduction: Morel-Lavallée lesions are closed, soft tissue degloving injuries that occur when shearing forces separate the subcutaneous tissue from the underlying fascia. This creates a potential space for hemolymphatic fluid accumulation. Morel-Lavallée lesions are frequently under-reported and often misdiagnosed as hematomas or abscesses, leading to potential mismanagement. While magnetic resonance imaging (MRI) is considered the gold standard for diagnosis, point-of-care ultrasound (POCUS) has been shown to be an effective adjunct that can rapidly confirm sonographic features consistent with Morel-Lavallée lesions, support MRI-established findings, and guide procedural intervention in the emergency department (ED) setting.

Case Report: We describe a 20-year-old woman presenting to the ED with persistent right-sided hip pain two months after a pedestrian–motor vehicle collision. Initially misdiagnosed as a hematoma, an outpatient follow-up MRI identified a large Morel-Lavallée lesion, with POCUS confirming subcutaneous anechoic fluid collection in the ED. Ultrasound-guided aspiration yielded 150 mL of serosanguinous fluid, with real-time imaging confirming decompression. Laboratory analysis demonstrated elevated red blood cell and nucleated cell counts consistent with Morel-Lavallée lesion.

Conclusion: This case highlights the integrated diagnostic and therapeutic role of point-of-care ultrasound as an adjunct to MRI, confirming sonographic features of a Morel-Lavallée lesion, guiding aspiration, and providing real-time postprocedural assessment within a single ED encounter in a patient presenting two months after injury. [Clin Pract Cases Emerg Med. 2026;10(4):644–647.]

Keywords: *Morel-Lavalle lesion; point-of-care ultrasound; emergency medicine; ultrasound-guided aspiration; case report.*

INTRODUCTION

A Morel-Lavallée lesion is a closed soft tissue degloving injury that occurs when a shearing force separates the subcutaneous tissue from the underlying fascia.[6] This leads to the potential formation of a space between the two layers, typically filled with hemolymphatic fluid.[6] Morel-Lavallée lesions can present acutely or be delayed in onset, and they can often be misdiagnosed due to their nonspecific external signs, such as ecchymosis and swelling resembling hematoma or abscesses.[5,6] As a result, they are frequently underdiagnosed or mismanaged in the acute trauma setting.[5]

Early recognition is critical to guide management and prevent further complications such as skin necrosis and capsule formation.[6] The gold standard for diagnosing Morel-Lavallée lesions is magnetic resonance imaging (MRI) due to its superior contrast and multiplanar capabilities, allowing for detection of capsule formation and lesion chronicity. However, point-of-care ultrasound (POCUS) has emerged as a rapid, cost-effective, radiation-free imaging modality within the emergency department (ED) that can serve as an effective adjunct to MRI, providing sonographic confirmation of characteristic features and guiding procedural intervention.[1-4,7] In orthopedic and radiology practice, percutaneous ultrasound-guided aspiration has similarly been employed as a first-line management strategy for Morel-Lavallée lesions, with favorable outcomes reported across multiple patient series.[8,9]

The following case report demonstrates the utility of POCUS in the ED as an adjunct to confirm an MRI-established diagnosis of a Morel-Lavallée lesion in a patient who was initially misdiagnosed with a hematoma, and the subsequent use of POCUS for guided aspiration. While prior emergency medicine and orthopedic literature documents both POCUS-guided aspiration and ultrasound-guided drainage of these lesions,[1-4,7-9] our report specifically illustrates POCUS used to confirm MRI findings, guide complete aspiration with real-time decompression monitoring, and facilitate definitive ED management.

CPC-EM Capsule

What do we already know about this clinical entity?
Morel-Lavallée lesions are closed degloving injuries that may mimic hematomas or abscesses and are frequently underdiagnosed.

What makes this presentation of disease reportable?
Point-of-care ultrasound confirmed a magnetic resonance imaging-diagnosed Morel-Lavallée lesion and enabled ultrasound-guided aspiration and real-time confirmation of decompression.

What is the major learning point?
Point-of-care ultrasound can confirm characteristic features of a Morel-Lavallée lesion, guide aspiration, and immediately assess procedural success.

How might this improve emergency medicine practice?
Point-of-care ultrasound can integrate bedside assessment and image-guided treatment of Morel-Lavallée lesions during a single emergency department encounter.

CASE REPORT

A 20-year-old woman presented to the ED with persistent right-sided hip and thigh pain. Two months prior to admission the patient was involved in a pedestrian–motor vehicle collision and was evaluated at a trauma center. Workup at that facility showed no fracture. Swelling over the hip was initially diagnosed as a hematoma. An MRI performed outpatient revealed evidence of a fluid collection suspicious for a Morel-Lavallée lesion measuring approximately 10 × 12 cm (Image 1). The patient was then lost to follow-up for 11 days. She was called by her primary physician and instructed to come into the ED.

Physical examination revealed swelling localized to the lateral aspect of the right hip, indurated skin, and boggy to palpation, with the affected leg being warm to touch with normal sensation. The patient's vital signs were within normal limits, and sepsis screening did not indicate suspected infection. Point-of-care ultrasound of the right hip was performed by an attending emergency physician, revealing a large subcutaneous anechoic fluid collection measuring approximately 10 × 5 cm (Image 2). The collection contained internal echogenic debris but no sonographic evidence of abscess formation or swirl sign. These findings were consistent with the initial MRI diagnosis of Morel-Lavallée lesion, revealing a closed soft tissue degloving injury and supporting the clinical suspicion.

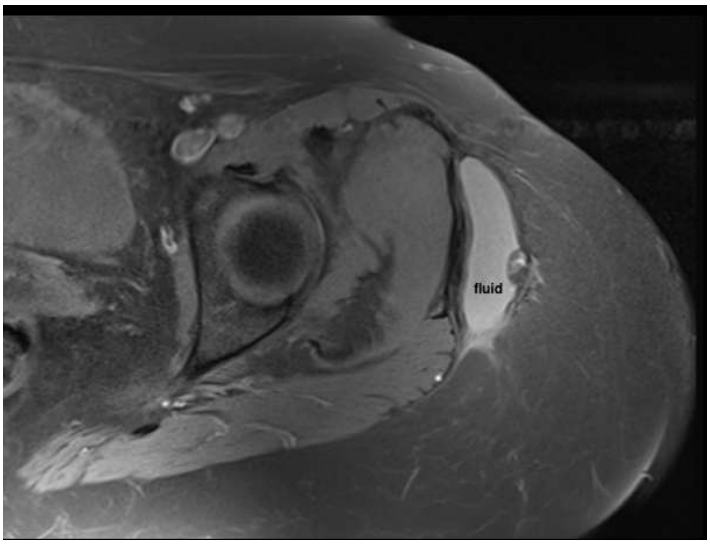


Image 1. Representative case of an axial T2-weighted magnetic resonance imaging of a Morel-Lavallée lesion, demonstrating circumscribed thin-walled fluid collection at the interface between the subcutaneous fat and deep fascia, with fluid-fluid level and small globular focus of fat signal intensity.
Image courtesy of Dr. J. Linklater, Radiopaedia.org, rID: 26390. Licensed under CC BY-NC-SA.[10]

Following ultrasound imaging, the patient underwent in-plane ultrasound-guided needle aspiration and drainage of the fluid collection over the right hip. Approximately 150 mL of serosanguinous fluid was aspirated. Subsequent real-time ultrasound imaging was performed to confirm complete aspiration and drainage of fluid that occupied the space between subcutaneous tissue and underlying fascia; imaging demonstrated a significant reduction of the aforementioned space (Image 3). The patient was provided a conservative management plan of using compression tape over the affected area, and she was instructed to follow up with general surgery for any reaccumulation or further management.

The fluid was sent for laboratory testing, specifically a body fluid culture, body fluid cell count, and body fluid

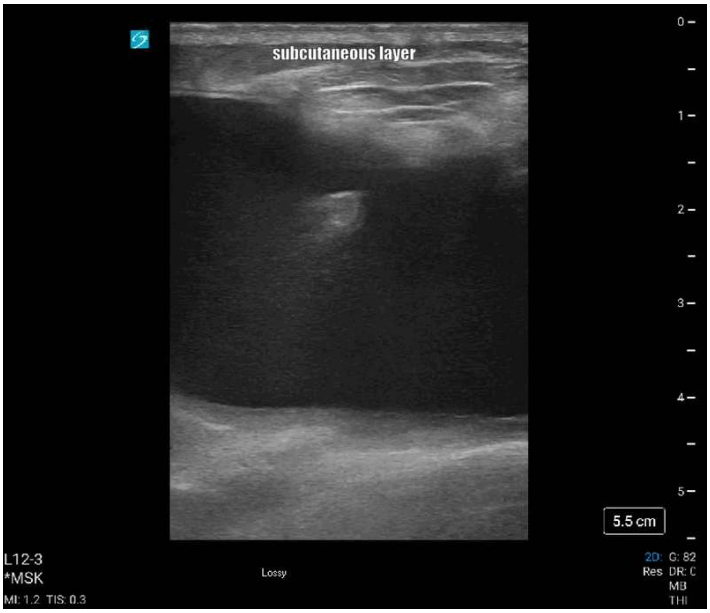


Image 2. Point-of-care ultrasound revealing subcutaneous anechoic fluid collection measuring approximately 10 × 5 centimeters with evidence of hyperechoic debris.

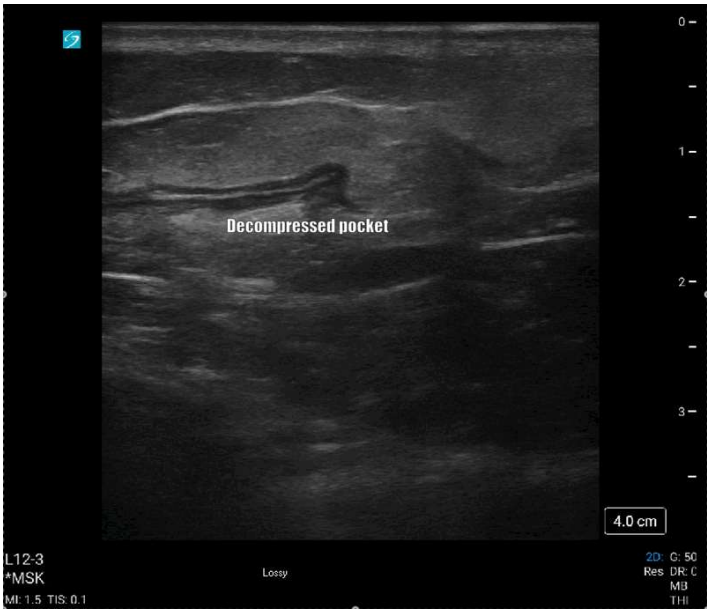


Image 3. Postaspiration of the fluid collection over the right hip, demonstrating significant decompression between subcutaneous tissue and underlying fascia.

differential count. The aspirated fluid appeared serosanguinous and amber in color. Laboratory analysis demonstrated a total fluid red blood cell count of 29,000/cm^2 (reference range, ~1,000/ cm^2) and a total nucleated cell count of 171/cm^2 (0–5/ cm^2). These results were abnormal and consistent with degloving injury with a collection of hemolymphatic fluid, providing strong evidence of a Morel-Lavallée lesion.

DISCUSSION

Morel-Lavallée lesions are closed soft tissue degloving injuries that result from shearing forces separating the hypodermis from the underlying fascia, creating a space for potential hemolymphatic fluid accumulation. These lesions remain underdiagnosed, in part because their clinical presentation frequently mimics more common conditions such as hematomas, seromas, or abscesses. The variable timing of symptom onset, which can range from weeks to even months and years, adds to diagnostic difficulty.[5] Without recognition and proper management, Morel-Lavallée lesions can progress to chronic encapsulation, recurrent fluid collections, infection, impaired wound healing, and devascularization of surrounding tissue that could lead to necrosis.[6]

Magnetic resonance imaging is often considered the imaging gold standard for confirming and characterizing these lesions because it can evaluate capsule formation and lesion chronicity.[6] However, MRI may not be readily available in the acute care setting due to cost, resource limitations, and scheduling delays. Ultrasound offers a rapid and cost-effective modality that can be performed at the bedside in the ED.[6,7] Sonographic features, such as fusiform hypoechoic or anechoic fluid collections between the subcutaneous tissue and fascia sometimes with internal septations or echogenic debris, are highly suggestive of Morel-Lavallée lesions.[6]

Our case joins an existing body of literature that discusses the use of POCUS in the context of the treatment and management of Morel-Lavallée lesions. This case underscores POCUS as a cost-effective modality that is used in conjunction with MRI to confirm diagnosis, guide aspiration, and confirm near-complete decompression of hemolymphatic fluid-filled space.[1-4, 7]

This case is unique in that POCUS serves as an effective adjunct to MRI diagnosis, providing fast bedside imaging to confirm the presence of characteristic subcutaneous fluid collection consistent with a Morel-Lavallée lesion. In addition, POCUS aided in ruling out signs of abscess formation and other potential pathologies, which was critical in determining appropriate management. It also served a therapeutic role by guiding real-time aspiration of serosanguinous fluid and verifying complete decompression of the fluid collection, providing real-time confirmation of procedural success.[4] Although POCUS has been demonstrated in this case to be an expedient option for the identification of a Morel-Lavallée lesion, its use is highly dependent on the sonographer's proficiency.[6]

In addition, this case has several limitations inherent to a single case report. Most notably, the original MRI images are inaccessible from outside hospital records, precluding direct side-by-side comparison of MRI and POCUS findings; the MRI diagnosis is corroborated through documented physician notes. Additionally, long-term follow-up data beyond the presented ED encounter are limited.

CONCLUSION

This case highlights the use of POCUS as an effective adjunct for the diagnosis of a Morel-Lavallée lesion. Point-of-care ultrasound provided rapid confirmation, excluded abscess formation, and guided successful aspiration with immediate postprocedural assessment. It confirmed, in real time, significant evacuation of the previously fluid-filled pocket occupying the space between subcutaneous tissue and underlying fascia thereby reducing long-term complications. The ability to integrate diagnosis and treatment in a single encounter underscores ultrasound's value as a practical, cost-effective tool that can expedite care in the ED, prevent complications, and improve outcomes in patients presenting with posttraumatic soft tissue swelling with underlying Morel-Lavallée lesions. In this case, POCUS helped to raise suspicion by supporting the initial MRI diagnosis and facilitated ultrasound-guided aspiration and immediate postprocedural assessment. Magnetic resonance imaging remains the gold standard for definitive diagnosis and characterization of Morel-Lavallée lesions, while POCUS is a valuable early imaging adjunct in the ED.

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Ami Kurzweil, MD, Eisenhower Health, Emergency Department, 39000 Bob Hope Dr, Rancho Mirage, CA 92270. Email:akurzweil@eisenhowerhealth.org.

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Diagnosing a Fractured Pedunculated Osteochondroma Using Point-of-Care Ultrasound in the Emergency Department: A Case Report

Jared Wilson, DO
Martin Tremwel, MD
Madeline Query, BS

University of Illinois College of Medicine–Peoria, Department of Emergency Medicine, Peoria, Illinois

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Introduction: This case report discusses the use of point-of-care ultrasound (POCUS) in the diagnosis of a fractured pedunculated osteochondroma in an 8-year-old boy after minor trauma. Although osteochondromas are the most common benign bone tumors, especially in this age, historically only radiographs have been obtained in the emergency department to diagnose these benign bone tumors.

Case Report: An 8-year-old boy reported ongoing pain for 2 months but had an acute onset worsening of his pain after minor trauma to the medial distal right femur. Point-of-care ultrasound was used to diagnose a fractured pedunculated osteochondroma prior to radiographic imaging.

Conclusion: We demonstrated the practicality and ease of POCUS in assisting with the diagnosis of an osteochondroma and the subtle fracture of the base. Both POCUS and radiographic imaging may be beneficial when used in conjunction for the diagnosis of benign bone tumors in this age group. [Clin Pract Cases Emerg Med. 2026;10(4):648–650.]

Keywords: *osteochondroma; point-of-care ultrasound; pediatric; musculoskeletal ultrasound; case report.*

INTRODUCTION

Osteochondroma is the most common benign bone tumor, most commonly diagnosed in children younger than 20 years of age.[1] They account for 20% to 50% of benign bone tumors and 9% of all bone tumors.[1-4] There are two specific features of osteochondromas: the presence of cortical and medullary continuity; and the presence of a cartilage cap.[1] While mostly painless, causes of pain include fracture, nerve impingement, or vascular compromise.[1] Initial diagnostic study of choice is a radiograph while ultrasound can aid in the diagnosis and evaluation, particularly in measuring cartilage cap thickness.[1] A cap thickness less than 2.0 cm in children and less than 1.5 cm in adults is reassuring and less likely to have malignant transformation.[2] This presentation was typical, but we demonstrated use of point-of-care ultrasound (POCUS) in the initial diagnosis and were able to diagnose fracture and cortical irregularity which have historically been done solely with radiographs.

CASE REPORT

History

An 8-year-old boy presented to our emergency department (ED) for acute on chronic right knee pain. He initially reported two months of right knee pain with acute worsened symptoms the night before after bumping his medial knee with his elbow. He now reported a palpable bump. He was initially evaluated by his primary care clinician several months earlier and was diagnosed with growing pains.

The patient had no significant past medical or social history. His only allergy was to cephalexin. On physical

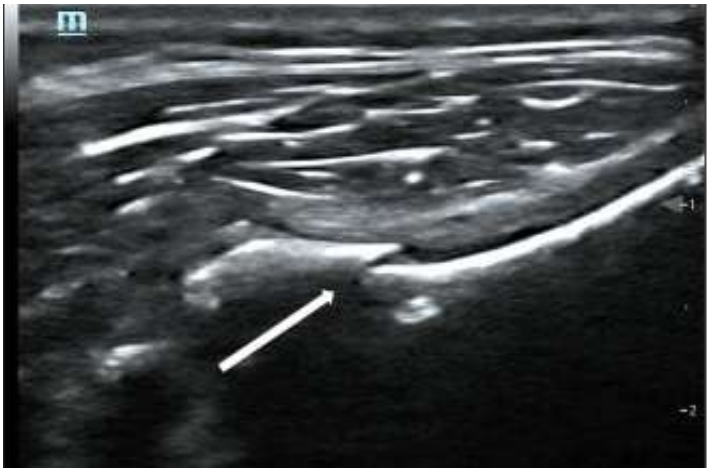


Image 1. Point-of-care musculoskeletal ultrasound using a linear probe in long axis demonstrates a hyperechoic step-off (white arrow) consistent with distal medial femur fracture.

CPC-EM Capsule

What do we already know about this clinical entity?
Osteochondromas are common benign bone tumors in children; fracture is a recognized complication that can cause acute pain.

What makes this presentation of disease reportable?
This case demonstrates a novel use of point-of-care ultrasound (POCUS) to diagnose a fractured distal femoral osteochondroma after minor trauma in a child.

What is the major learning point?
POCUS can identify cortical irregularity and fracture of an osteochondroma and may complement radiographs in the emergency department.

How might this improve emergency medicine practice?
POCUS may help emergency physicians rapidly identify bony lesions and suspected fracture, guiding radiography and specialty follow-up.

exam, he was noted to have a palpable swollen/firm area on the right distal medial femur without evidence of overlying erythema, fluctuance or induration. This area was significantly tender to palpation. He maintained full range of motion of the knee.

Diagnostic Imaging

Initial diagnostic imaging in this case was POCUS (Image 1). This demonstrated a hyperechoic density visualized on the right distal medial femur with likely step-off or defect consistent with fracture. Given the concern for cortical irregularity and possible bone lesion, further imaging was obtained, which included radiographs of the right knee in four views as well as a consultative ultrasound performed in the radiology suite.

Radiographs demonstrated a circumscribed exophytic bone lesion arising from the medial aspect of the distal femur measuring approximately 20 × 10 mm (Image 2). This was contiguous with adjacent cortex and trabeculae and characteristic for an osteochondroma. Seen best on the anteroposterior view, there appeared to be a fracture through the base of the osteochondroma with approximately 1 mm cortical step-off inferiorly. Consultative ultrasound demonstrated an exophytic osseous lesion arising from the distal medial femur, consistent with an osteochondroma (Image 3).

With the above findings of osteochondroma with fracture as seen on both plain radiographs and ultrasound, the pediatric orthopedics team recommended outpatient follow-up for further discussion of management. He was discharged from the ED in stable condition, able to bear weight as tolerated. He was seen in an orthopedic clinic two days later where they discussed management options including surgical excision. The patient and his mother elected to undergo surgical excision and underwent successful surgical removal of the osteochondroma two months later.

DISCUSSION

This case demonstrates the ability of POCUS to assist in the diagnosis of fractured osteochondromas in the ED. Although plain radiographs are still the standard imaging modality for initial diagnosis, ultrasound has been shown to be an effective supplemental tool. In osteochondroma, ultrasound is typically used to evaluate the cartilaginous cap rather than radiographs as initial diagnostic imaging.[1] This case was a typical presentation of classic osteochondroma but had an atypical fracture history with only very minor trauma to the area. Ultrasound enabled visualization of the cortical irregularity concerning for fracture pathology, which was able to be fully visualized on plain radiographs.

No similar case reports were identified in our literature review. Existing literature discusses the utility of POCUS for fractures as a reasonable option when radiographs are unavailable or to limit radiation exposure.[5] However, despite the benefits of ultrasound, its use can be limited because it is operator dependent.[5] Specific to osteochondroma, POCUS



Image 2. Right knee radiograph demonstratesa bone lesion arising from the medial aspect of the distal femoral diametaphysis; appearance is characteristic of an osteochondroma (white arrow).



Image 3. Radiologist-performed ultrasound of an exophytic osseous lesion arising from the distal right medial femur, consistent with an osteochondroma (white arrow).

does not allow visualization of the cartilage cap when it is inwardly oriented or deep in soft tissue.[5]

CONCLUSION

An 8-year-old boy presented with a typical presentation of osteochondroma of the distal right femur. His acute onset of worsened pain after minor trauma was likely related to fracture through the stalk of the lesion, which was able to be visualized on POCUS as well as confirmed on radiographic imaging. He followed up with an orthopedic specialist and underwent successful excision two months after initial diagnosis in the ED. The case demonstrates that POCUS can be an effective tool in facilitating the diagnosis of benign bone tumors.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Jared Wilson, DO, University of Illinois College of Medicine–Peoria, Department of Emergency Medicine, 530 NE Glen Oak Ave, Peoria, Illinois, 61605. Email: jnwilson23@osfhealthcare.org.

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Soft Palate Perforation with Endotracheal Tube During Video Laryngoscopy

Samuel J. Thomas, BS*
David I. Zimmer, MD, DVM†
Jack H. Langford, BS*
Toby J. Brenner, BS*
Kate M. Kelly, BSN‡
Donald F. Zimmer, MD*§

*Indiana University School of Medicine, Department of Emergency Medicine, South Bend, Indiana
†University of Louisville Hospital, Department of Otolaryngology, Louisville, Kentucky
‡George Washington University School of Medicine and Health Sciences, Department of Clinical Research and Leadership, Washington, District of Columbia
§Memorial Hospital, Department of Emergency Medicine, South Bend, Indiana

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Case presentation: A 57-year-old man with a history of obesity-hypoventilation syndrome, diabetes mellitus, and chronic renal insufficiency was intubated with an 8.0-mm endotracheal tube under video laryngoscopy in the emergency department due to respiratory failure. After successful intubation, bloody secretions were noted in the patient's mouth. Subsequent examination revealed that the endotracheal tube had caused a through-and-through perforation at the junction of the soft palate and the anterior tonsillar pillar. Because the intubation had been successful, the endotracheal tube was not removed, and the soft palate was transected to free the tube. The patient made an uneventful recovery.

Discussion: Soft palate perforation is an uncommon injury that has been reported more frequently with video laryngoscopy than direct laryngoscopy. To prevent this injury and ensure its rapid recognition, it is recommended that the clinician sequentially look at the "5 M's" during intubation: Mouth–Monitor–Mouth–Monitor–Mouth. [Clin Pract Cases Emerg Med. 2026;10(4):651–653.]

Keywords: *endotracheal intubation; laryngoscopy; soft palate; oropharynx; soft tissue injuries.*

CASE PRESENTATION

A 57-year-old man with a past medical history significant for obesity-hypoventilation syndrome, diabetes mellitus, and chronic renal insufficiency presented to the emergency department (ED) in respiratory failure. The vital signs were as follows: respiratory rate, 32 breaths per minute; blood pressure, 90/60 mm Hg; pulse, 130 beats per minute; temperature, 98.6 °F; and oxygen saturation was 83% with diminished bilateral breath sounds, which led to the patient being placed on bilevel positive airway pressure. After 30 minutes, arterial blood gases revealed the following: pH 7.06 (reference range, 7.35–7.45); oxygen pressure, 47 mm Hg (75–105 mm Hg); and carbon dioxide pressure, 116 mm Hg (33–45 mm Hg). The patient therefore required endotracheal intubation, which was performed using a video laryngoscope with a Macintosh 4 blade. The first attempt failed because of gastric secretions that required suctioning. The second attempt was successful; however, the secretions were noted to be bloody (Image).

Immediate end-tidal carbon dioxide detection was normal when the patient was placed on synchronized intermittent mandatory ventilation. He was transferred to the intensive care unit. Continued bleeding from the oropharynx resulted in an otolaryngology consultation. Evaluation of the oropharynx revealed a right soft palate perforation through which the endotracheal tube (8.0-mm inner diameter, 11.8-mm outer diameter) passed directly into the trachea (Video). Because the patient was ventilated satisfactorily, the endotracheal tube was repositioned anteriorly and medially, allowing for transection and repair of the soft palate. He made an uneventful recovery following five days of mechanical ventilation, during which he responded to diuresis, pulmonary toilet, bronchodilators, and corticosteroids.

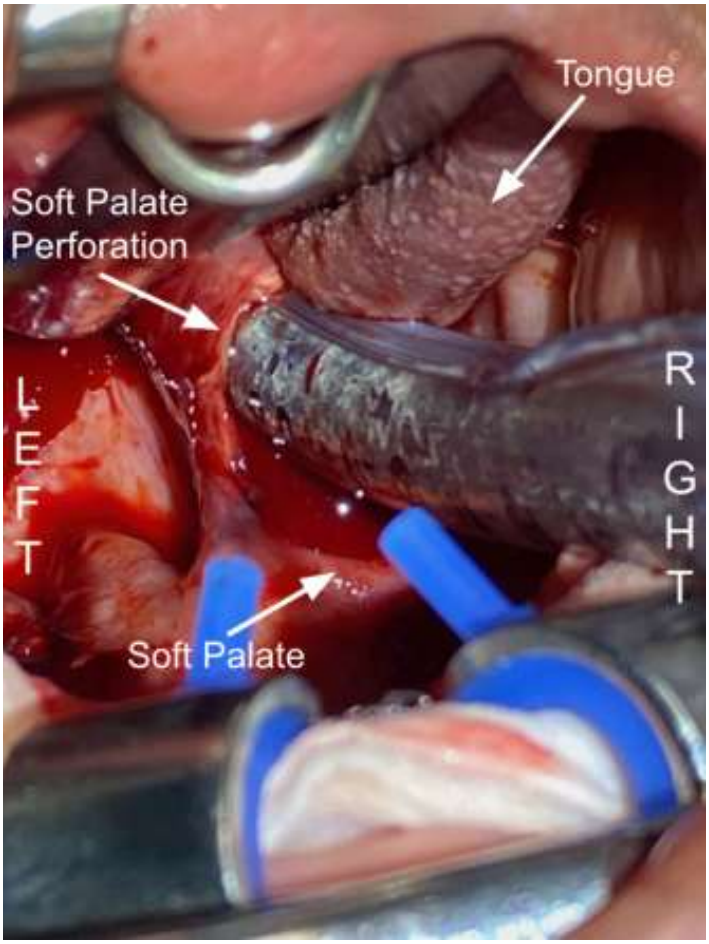


Image. Endotracheal tube perforating the soft palate.

CPC-EM Capsule

What do we already know about this clinical entity?
Perforations and other injuries to the soft palate during endotracheal intubation are uncommon complications that are reported more often during video laryngoscopy.

What is the major impact of the image(s)?
The image and video show the location of the perforation with the endotracheal tube caught in the soft palate and relevant labels to help orient the viewer.

How might this improve emergency medicine practice?
The proposed mnemonic Mouth–Monitor–Mouth–Monitor–Mouth indicates where clinicians should look to prevent and identify injuries during video laryngoscopy.

DISCUSSION

Recent literature demonstrates that videolaryngoscopy–guided endotracheal intubation improves first-pass success rates and laryngeal views. Consequently, this approach has become the default technique in EDs and operating rooms.[1,2] However, injuries to the soft palate and surrounding structures occur more frequently with video laryngoscopy than with direct laryngoscopy.[3,4] This case report describes an uncommon injury to the soft palate and anterior tonsillar pillar during endotracheal intubation.

A mnemonic designed to improve accuracy and prevent complications during video laryngoscopy–guided intubation is the "Mouth–Screen–Mouth–Screen" sequence.[5] However, this technique does not emphasize reevaluation of the mouth after intubation. Therefore, a more detailed mnemonic is recommended to prompt clinicians to look at the "5 M's" (Mouth–Monitor–Mouth–Monitor–Mouth: (1) Look into the patient's mouth while inserting the laryngoscope blade; (2) look at the monitor display of the glottis to position the camera; (3) look again into the mouth to insert the endotracheal tube and avoid perforations with the rigid stylet; (4) look at the monitor to position the tube before popping the stylet, advancing through the vocal cords, and withdrawing the stylet once the tube is in its proper position; and (5) look directly at the mouth while removing the laryngoscope to evaluate for any injuries while ensuring the tube is secured.

Video. This video shows the perforation at the junction of the soft palate and the right anterior tonsillar pillar by the endotracheal tube.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Samuel J. Thomas, BS, Indiana University School of Medicine, Department of Emergency Medicine, 1234 N Notre Dame Ave, South Bend, IN 46617. Email: sajathom@iu.edu.

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Gaze-Evoked Vomiting After Minor Orbital Trauma: White-Eyed Blowout Fracture

Naoki Yamada, MD*
Yukinori Kato, MD, PhD†

*University of Fukui Hospital, Department of Emergency Medicine and General Medicine, Fukui, Japan
†University of Fukui Hospital, Department of Otorhinolaryngology–Head and Neck Surgery, Fukui, Japan

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Case Presentation: A 16-year-old girl presented to the emergency department after being struck in the right eye by another player's elbow during a basketball game. She had only minimal periorbital swelling but developed persistent nausea, repeated vomiting, and diplopia with upward gaze. Visual acuity, visual fields, and pupillary light reflexes were normal. Extraocular movement testing showed mild limitation of upward gaze of the right eye, and upward gaze reproducibly provoked diplopia, marked nausea, and vomiting. Computed tomography (CT) showed no intracranial hemorrhage, and orbital CT was initially interpreted as showing no obvious orbital floor fracture or extraocular muscle entrapment. Given the discrepancy between the mild external appearance and the severe ocular symptoms, white-eyed blowout fracture with trapdoor-type entrapment and an oculocardiac reflex was suspected. She underwent emergent surgery, after which her vomiting, diplopia, and ocular motility disturbance improved immediately.

Discussion: White-eyed blowout fracture is a trapdoor-type orbital floor injury seen mainly in children and adolescents and may present with minimal external signs and subtle or negative-appearing CT findings.[1-3] Gaze-evoked nausea or vomiting, diplopia, and relative bradycardia are important bedside clues to extraocular muscle entrapment with an oculocardiac reflex.[1-3] This case highlights that white-eyed blowout fracture may be detected by careful bedside examination even when CT does not clearly show an orbital floor fracture. Emergency physicians should recognize this entity as a clinically important, easily missed diagnosis in pediatric orbital trauma. [Clin Pract Cases Emerg Med. 2026;10(4):654–656.]

Keywords: *white-eyed blowout fracture; orbital trauma; trapdoor fracture; oculocardiac reflex; diplopia.*

CASE PRESENTATION

A 16-year-old previously healthy female was brought to the emergency department after being struck in the right periorbital region by an opponent's elbow during a basketball game. She was alert and hemodynamically stable: blood pressure, 126/73 mm Hg; heart rate, 56 beats per minute; respiratory rate, 18 breaths per minute; oxygen saturation, 99% on room air; and temperature, 36.8 ºC. Examination showed only minimal swelling around the right eye without marked ecchymosis. Visual acuity, visual fields, and pupils were normal. Extraocular movement testing showed mild limitation of upward gaze of the right eye. Upward gaze reproducibly provoked diplopia, marked nausea, and repeated vomiting. Head computed tomography (CT) showed no traumatic intracranial findings. Orbital CT was initially interpreted as showing no obvious orbital floor fracture or extraocular muscle entrapment (Image 1).

Despite the mild external appearance and initially subtle imaging findings, the combination of gaze-evoked vomiting, diplopia, mild upward gaze limitation, and relative bradycardia

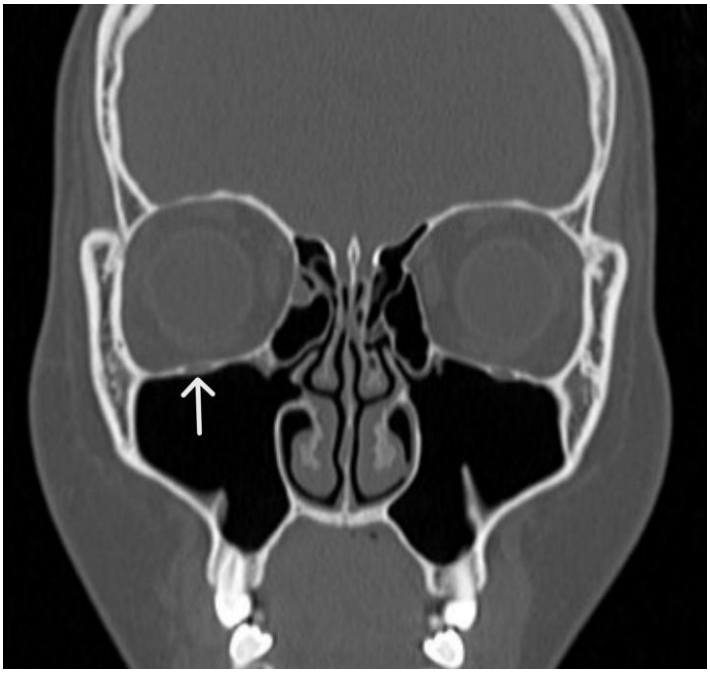


Image 1. Coronal orbital computed tomography at presentation. The arrow indicates the apparently intact right orbital floor, with no visible evidence of fracture or extraocular muscle entrapment.

CPC-EM Capsule

What do we already know about this clinical entity?
White-eyed blowout fracture can present with minimal external signs but cause extraocular muscle entrapment and oculocardiac symptoms.

What is the major impact of the image(s)?
The images demonstrate subtle computed tomography (CT) findings and operative confirmation of trapdoor entrapment despite minimal external trauma signs.

How might this improve emergency medicine practice?
Emergency physicians should suspect trapdoor fracture when diplopia, vomiting, or bradycardia follows minor orbital trauma, even with subtle CT findings.

raised concern for a white-eyed blowout fracture with trapdoor-type entrapment and an oculocardiac reflex. Otolaryngology was urgently consulted, and the patient underwent emergent surgery. Intraoperatively, a right orbital floor fracture with suspected orbital soft-tissue entrapment was identified and released (Image 2). Her vomiting, diplopia, and ocular motility disturbance improved promptly after surgery.

DISCUSSION

White-eyed blowout fracture is a trapdoor-type orbital floor injury seen in children and adolescents.[1-3] Pediatric facial fractures account for less than 15% of all facial fractures, and orbital blowout fractures constitute approximately 20% of pediatric facial fractures.[3] In trapdoor fractures, the elastic orbital floor may transiently open and recoil, entrapping the inferior rectus muscle or adjacent orbital soft tissue while leaving only minimal external signs of trauma.[1-3] Consequently, clinicians may miss the diagnosis when swelling or ecchymosis is minimal.

This case emphasizes that CT may underestimate or fail to clearly demonstrate pediatric trapdoor-type injury.[2] Although CT is the first-line imaging modality for orbital trauma, soft-tissue entrapment may not be apparent on initial interpretation. Parbhu et al reported a radiologic-intraoperative concordance rate of 50% in children with orbital floor fractures, compared with 87% in adults.[2] Therefore, extraocular muscle entrapment should be considered a clinical diagnosis rather than a purely radiographic one.

In young patients with orbital trauma, the mismatch between mild external findings and severe symptoms is an important clue. Diplopia, restricted upward gaze, nausea or vomiting triggered by eye movement, and relative bradycardia should raise concern for extraocular muscle entrapment with an oculocardiac reflex.[1-4] Early recognition is important because delayed release of entrapped tissue may lead to ischemia, fibrosis, persistent diplopia, and ocular motility dysfunction.[1,3] Missed diagnosis and pediatric trapdoor fractures with subtle CT findings have been reported previously.[4,5] This case adds to the emergency medicine literature by highlighting gaze-evoked vomiting as a bedside clue to white-eyed blowout fracture when CT findings are subtle or initially interpreted as negative.

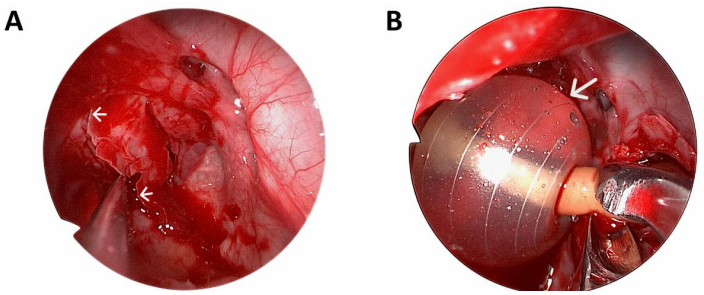


Image 2. Endoscopic transnasal surgical findings. A: Intraoperative endoscopic view demonstrating the orbital floor fracture line, indicated by white arrows. A forceps is advanced toward the fracture cleft. B: After release and reduction of the prolapsed orbital tissues, a balloon catheter, indicated by a white arrow, is placed to support the repaired orbital floor.

The authors attest that their institution does not require Institutional Review Board approval for publication of this image in emergency medicine. Patient consent has been obtained and filed for the publication of this image in emergency medicine.

Address for Correspondence: Naoki Yamada, MD, University of Fukui Hospital, Department of Emergency Medicine and General Medicine, 23-3 Shimoaizuki, Matsuoka, Eiheiji-cho, Yoshida-gun, Fukui 910-1193, Japan. Email: nyamada@u-fukui.ac.jp

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Mechanical Small Bowel Obstruction Secondary to Deflated and Migrated Intragastric Balloon

Luis Moas, MD* *Mount Sinai Medical Center, Department of Emergency Medicine, Miami Beach, Florida
Arturo Feraud, MD† †Mount Sinai Medical Center, Department of Internal Medicine, Miami Beach, Florida
Aliyah Baksh, BS‡ ‡Florida International University, Herbert Wertheim College of Medicine, Department of Emergency Medicine, Miami, Florida
Eric Boccio, MD, MPH*

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Case presentation: A 29-year-old woman presented to the emergency department three months after intragastric balloon placement with nausea, vomiting, constipation, and abdominal pain. Computed tomography (CT) demonstrated spontaneous deflation and migration of the device into the small bowel with resultant mechanical small bowel obstruction. The patient was managed conservatively with bowel rest, intravenous fluids, electrolyte repletion, and nasogastric decompression. Repeat CT on hospital day 2 confirmed distal migration of the balloon into the descending colon with interval decompression of the small bowel. On hospital day 3, the patient reported spontaneous passage of the balloon per rectum with complete resolution of symptoms.

Discussion: Spontaneous deflation and distal migration of an intragastric balloon causing small bowel obstruction is a rare but potentially life-threatening complication. Most reported cases require surgical or endoscopic intervention. This case highlights spontaneous transanal passage as a rare but favorable outcome achievable with conservative management in carefully selected, hemodynamically stable patients without signs of peritonitis, bowel ischemia or perforation, or failure of the intragastric balloon to progress. [Clin Pract Cases Emerg Med. 2026;10(4):657–659.]

Keywords: *intragastric balloon; small bowel obstruction; gastric balloon migration; clinical image.*

CASE PRESENTATION

A 29-year-old woman with obesity (body mass index, 32.8 kg/m^2) presented to the emergency department (ED) with a two-day history of intractable nausea, bilious vomiting, constipation, and diffuse abdominal pain three months after undergoing intragastric balloon placement at a facility in Mexico. She reported a 35-pound reduction in weight during that time but had no follow-up with the physician who inserted the device. She endorsed up to seven episodes of nonbloody, bilious vomiting per day, intolerance of oral intake, and no bowel movements for three days.

The patient's initial ED triage vital signs were within normal limits. Abdominal examination demonstrated mild distension with the point of maximal tenderness in the left lower quadrant; there was no rebound tenderness or guarding and bowel sounds were present. Laboratory evaluation was notable for hypokalemia (potassium, 3.3 mmol/L; reference range, 3.5-5.0 mmol/L), with an otherwise unremarkable complete metabolic panel, complete blood count, and lipase.

Noncontrast computed tomography (CT) of the abdomen and pelvis demonstrated a deflated and displaced intragastric balloon with associated bowel wall edema and a dilated loop measuring up to 3.2 cm, consistent with a developing small bowel obstruction (Images 1 and 2). General surgery and gastroenterology were consulted, and the patient was admitted and managed conservatively with nasogastric tube decompression, strict nothing per os status, maintenance intravenous fluid infusion, potassium repletion, and serial abdominal examinations. Repeat contrast-enhanced CT of the abdomen and pelvis on hospital day 2 demonstrated interval



Image 1. Coronal noncontrast computed tomography of the abdomen and pelvis obtained during the workup in the emergency department demonstrating the displaced intragastric balloon (arrow) within small bowel loops in the left lower quadrant consistent with a mechanical small bowel obstruction.

CPC-EM Capsule

What do we already know about this clinical entity?
Intragastric balloon deflation and migration can cause rare, life-threatening small bowel obstruction, traditionally requiring surgical or endoscopic removal.

What is the major impact of the image(s)?
Serial computed tomography shows balloon migration from obstructing the small bowel to spontaneous passage through the colon.

How might this improve emergency medicine practice?
Conservative management with serial exams and imaging in stable patients with progressing balloon transit is feasible.

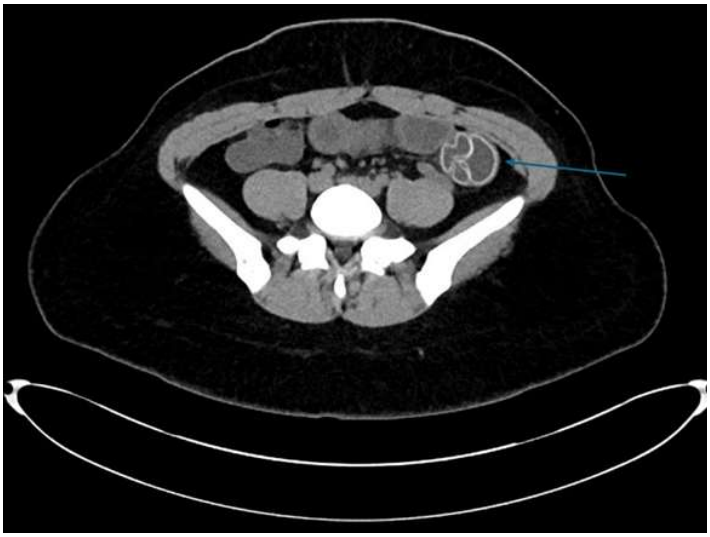


Image 2. Axial noncontrast computed tomography of the abdomen and pelvis obtained during the workup in the emergency department demonstrating the displaced intragastric balloon (arrow) within the small bowel of the left lower quadrant.

transit of the balloon into the descending colon with decompression of previously dilated small bowel loops and no evidence of perforation (Image 3). On hospital day 3, the patient reported spontaneous passage of the balloon per rectum with complete resolution of symptoms, and she was discharged in stable condition.

DISCUSSION

Intragastric balloon therapy has been used for weight loss since Nieben and Harboe introduced the procedure in 1982, offering a nonsurgical option for patients who do not qualify for bariatric surgery.[1] Spontaneous deflation and distal migration causing small bowel obstruction is a rare complication estimated to occur in fewer than 1% of cases.[2] Upon deflation, the collapsed silicone envelope may migrate through the pylorus and become impacted anywhere from the duodenum to the terminal ileum. Hay et al., in a review of 27 published cases, found that open or laparoscopic enterotomy and extraction was required in approximately 70% of cases, with the anatomical location of impaction guiding management.[2] Endoscopic retrieval is feasible when the balloon is accessible—most reliably from the duodenum—although successful endoscopic retrieval from the jejunum and retrograde colonoscopic retrieval from the terminal ileum have also been reported.[3-4] Timely removal is critical given the risk of progressive complications including bowel ischemia and perforation.

Conservative management without operative or endoscopic intervention is reasonable in hemodynamically stable patients

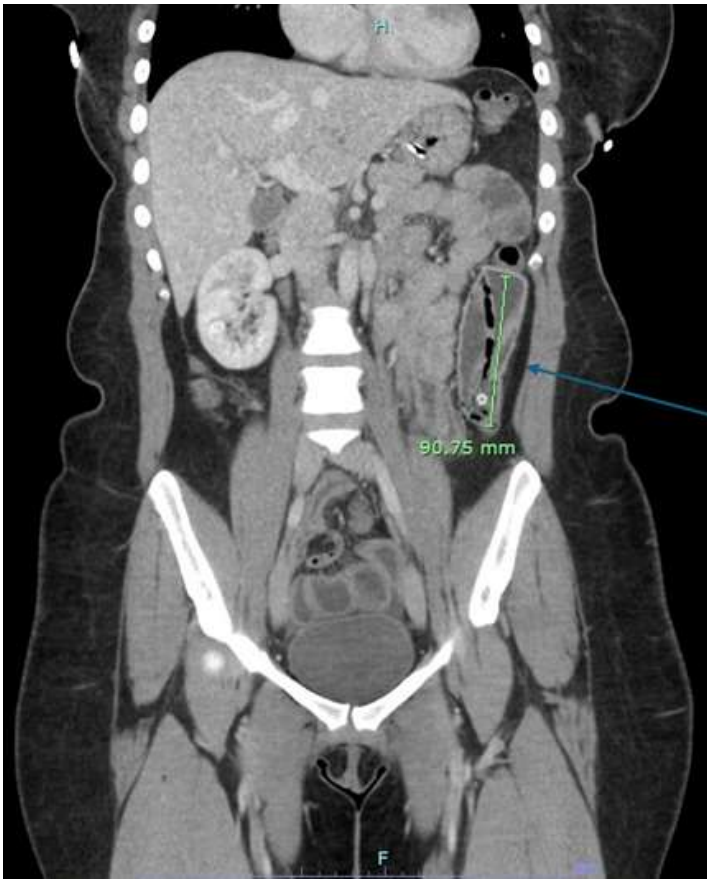


Image 3. Coronal contrast-enhanced computed tomography of the abdomen and pelvis obtained on hospital day 2 demonstrating the intragastric balloon (9.1 cm in the craniocaudal dimension), positioned within the descending colon with interval resolution of the small bowel obstruction (arrow).

with partial obstruction when spontaneous passage is anticipated.[5] Surgical removal is generally recommended if symptoms do not improve or the balloon fails to progress within 48 hours.[6] The specific intragastric balloon model, fill volume, and mechanism of deflation were unavailable because the procedure was performed outside the treating institution.

The present case demonstrates that spontaneous transanal passage is achievable with close clinical and radiographic monitoring and underscores the diagnostic value of serial abdominal examinations and CT imaging in guiding this decision. Emergency clinicians should maintain a high index of suspicion for mechanical small bowel obstruction in any patient with a history of intragastric balloon placement presenting with obstructive symptoms, regardless of time since placement, and a multidisciplinary approach that incorporates timely consultation with surgical and gastroenterological specialists remains essential.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Eric Boccio, MD, MPH, Mount Sinai Medical Center, Department of Emergency Medicine, 4300 Alton Road, Miami Beach, FL 33140. Email:bocciomedicalservices@gmail.com.

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Bedside Sonography Brings the Weiss Ring into Focus: Rapid Diagnosis of Posterior Vitreous Detachment

Joseph Betcher, MD
Matthew Shields, DO
Dakota Busch, DO

Michigan State University, Trinity Health Muskegon, West Michigan Emergency Medicine Residency, Muskegon, Michigan

Section Editor: Patrick Meloy, MD
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Case Presentation: A 51-year-old woman presented to the emergency department with blurred vision and a large, dark floater in her right eye. Point-of-care ultrasound (POCUS) revealed a Weiss ring, strongly suggesting a diagnosis of a posterior vitreous detachment.

Discussion: Identifying a Weiss ring with POCUS may be a reasonable and easier alternative for emergency clinicians when used in conjunction with a slit-lamp examination and may aid in timely ophthalmologic management of posterior vitreous detachments. [Clin Pract Cases Emerg Med. 2026;10(4):660–661.]

Keywords: *Weiss ring; posterior vitreous detachment; point-of-care ultrasound; POCUS; ocular ultrasound.*

CASE PRESENTATION

A 51-year-old woman presented complaining of blurred vision with a large, dark floater in her right eye. Examination demonstrated a slight decrease in visual acuity of the right eye. No pupillary abnormalities or ocular dysmotility was observed. There was no evidence of trauma or foreign body on fluorescein stain, and intraocular pressures were normal via tonometer. Laboratory tests and head computed tomography demonstrated no acute abnormalities. Point-of-care ocular ultrasound revealed the diagnosis (Image 1), which was later confirmed via fundoscopic examination with ophthalmology (Image 2).[1]

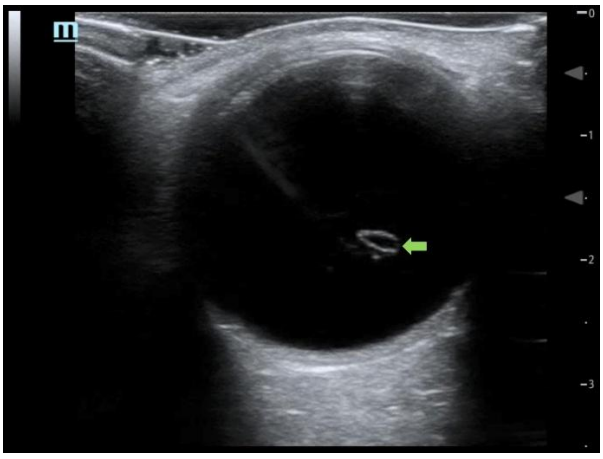


Image 1. Point-of-care ocular ultrasound demonstrates a hyperechoic annular structure, known as a Weiss ring, (arrow), which is highly suggestive of a posterior vitreous detachment.

DISCUSSION

We used point-of-care ultrasound (POCUS) to identify a Weiss ring (Video). This anatomic finding is formed when peripapillary glial tissue is torn from the optic nerve head and remains adherent to the posterior vitreous cortex.[2] The presence of a Weiss ring, when combined with a clinical presentation of sudden onset blurred vision with a floater, strongly suggests a diagnosis of a posterior vitreous detachment. The gold-standard for diagnosing posterior vitreous detachment is via slit-lamp biomicroscopy performed by ophthalmology at same-day visit.[3] Ocular POCUS demonstrates high specificity but poor sensitivity for posterior vitreous detachment.

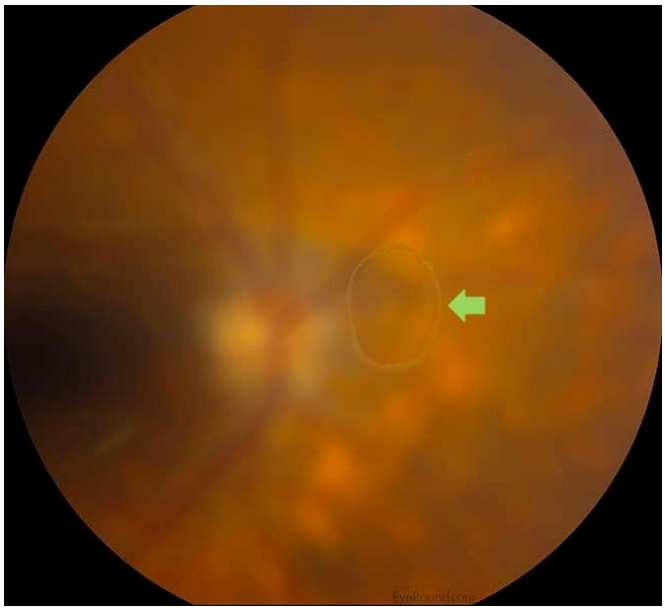


Image 2. Fundoscopic examination confirmed the presence of a Weiss ring, a ring-like opacity adjacent to the optic disc (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Posterior vitreous detachment is a common cause of flashes/floaters and may signal a vision-threatening retinal tear.

What is the major impact of the image(s)?
The images show a less commonly visualized variant of a posterior vitreous detachment known as a Weiss ring, which may be misconstrued as an artifact.

How might this improve emergency medicine practice?
Improved identification of this variant can expedite diagnosis and lead to proper follow-up and care for a posterior vitreous detachment.

In the largest prospective emergency department study, POCUS identified posterior vitreous detachment with a sensitivity of 42.5% and specificity of 96.0%, indicating that positive sonographic findings are highly suggestive of disease, whereas negative findings do not reliably exclude it. Although the presence of a Weiss ring may be considered a characteristic finding of posterior vitreous detachment, published data regarding the sensitivity and specificity of ultrasound for Weiss ring detection are currently lacking. When this finding is present, however, POCUS may be a reasonable and easier alternative for emergency clinicians when used in conjunction with slit-lamp examination and may aid in timely ophthalmologic management.

Video. Point-of-care ocular ultrasound recording aided in the diagnosis of a posterior vitreous detachment. Fanning back-and-forth shows the complete Weiss ring structure (arrow).

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Joseph Betcher, MD, Michigan State University, Trinity Health Muskegon, West Michigan Emergency Medicine Residency, 1500 E Sherman Blvd, Muskegon, MI 49444. Email: joebetcher@gmail.com.

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A 30-Year-Old Man With Rapid Floor-of-Mouth Swelling After Facial Trauma

Mohammed Naveeth Imran, DNB
Surendar Ravipragasam, MD
Shwetha Pujari Hemanthraj, MBBS

Jawaharlal Institute of Postgraduate Medical Education & Research (JIPMER), Department of Emergency Medicine and Trauma, Puducherry, India

Section Editor: Grace Hickam, MD, MEd
Submission history: Submitted: June 12, 2026; Revision received August 8, 2026; Accepted August 9, 2026
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Case Presentation: A 30-year-old man presented to the emergency department with severe blunt facial trauma following a fall from height. Examination revealed extensive floor-of-mouth ecchymosis and a large tense sublingual hematoma causing tongue elevation, dysarthria, and swelling mimicking Ludwig's angina, producing a traumatic pseudo-Ludwig appearance with marked airway crowding. Three-dimensional computed tomography demonstrated severely displaced bilateral mandibular fractures. Owing to pronounced segmental instability with mobile dentoalveolar fragments, temporary interdental bridle wire stabilization was performed in the emergency department. Despite the dramatic anatomical findings, serial airway reassessments confirmed sustained airway stability, permitting delayed definitive open reduction and internal fixation.

Discussion: Traumatic pseudo-Ludwig angina secondary to bilateral mandibular fractures is an uncommon presentation that may closely mimic life-threatening infectious floor-of-mouth swelling. Hemorrhage within the confined sublingual space can rapidly elevate and posteriorly displace the tongue, resulting in progressive airway compromise. This case highlights the importance of dynamic airway assessment, serial airway reassessment, and early multidisciplinary management, including temporary interdental bridle wire stabilization, in carefully selected patients with traumatic hemorrhagic floor-of-mouth emergencies. [Clin Pract Cases Emerg Med. 2026;10(4):662–664.]

Keywords: *traumatic pseudo-Ludwig angina; mandibular fracture; sublingual hematoma; Coleman sign; facial trauma.*

CASE PRESENTATION

A 30-year-old man presented to the emergency department with severe blunt facial trauma following a fall from height. On arrival, he was hemodynamically stable with vital signs as follows: blood pressure, 110/80 mm Hg; heart rate, 92 beats per minute; respiratory rate, 16 breaths per minute; and SpO_2, 97% on room air. The primary trauma survey identified no other immediately life-threatening injuries requiring urgent intervention. Examination revealed lower facial swelling, facial abrasions, ecchymosis, and marked mandibular instability. Intraoral examination demonstrated extensive floor-of-mouth ecchymosis and a large tense sublingual hematoma causing tongue elevation, dysarthria, and marked airway crowding, closely mimicking Ludwig's angina and producing a traumatic pseudo-Ludwig appearance (Image 1). Despite the dramatic anatomical findings, the patient maintained a patent airway, handled oral secretions without difficulty, and exhibited no stridor or respiratory distress.

Three-dimensional computed tomography demonstrated severely displaced bilateral mandibular fractures involving the parasymphysis and body-angle regions (Image 2). Owing to pronounced segmental instability with mobile dentoalveolar fragments, temporary interdental bridle wire stabilization was performed in the emergency department following consultation with the oral and maxillofacial surgery team (Image 3). The patient remained under close observation in the emergency department with serial airway reassessments,



Image 1. Extensive floor-of-mouth ecchymosis and massive sublingual hematoma causing tongue elevation and traumatic pseudo-Ludwig appearance (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Infectious Ludwig's angina is well known, but traumatic pseudo-Ludwig angina is an exceedingly rare mimic of deep neck infection.

What is the major impact of the image(s)?
Bilateral mandibular fractures produced a dramatic pseudo-Ludwig appearance from sublingual hemorrhage despite preserved airway physiology.

How might this improve emergency medicine practice?
Recognizing this traumatic mimic and using physiology-guided airway assessment may help avoid unnecessary airway escalation in stable patients.

which confirmed sustained airway stability and partial resolution of the sublingual hematoma over the subsequent 24 hours. Following reassessment by the oral and maxillofacial surgery team, he was discharged from the emergency department with planned delayed definitive fixation.

DISCUSSION

Traumatic pseudo-Ludwig angina secondary to mandibular fractures is an uncommon but potentially life-threatening presentation that closely mimics infectious Ludwig's angina. Most reported cases of pseudo-Ludwig angina are associated with anticoagulation, coagulopathy, or spontaneous sublingual hematoma, whereas trauma-related cases remain exceedingly rare.[1-3] Distinguishing traumatic pseudo-Ludwig angina from deep neck space infection is crucial because the underlying pathology is hemorrhagic rather than infectious; therefore, management prioritizes dynamic airway assessment, identification of the bleeding source, and timely hemorrhage control.

Hemorrhage within the confined sublingual space can rapidly elevate and posteriorly displace the tongue, resulting in dysarthria, dysphagia, drooling, and progressive airway compromise. Coleman sign, a bluish, tense swelling of the floor of the mouth caused by sublingual hemorrhage, is an important clinical clue to mandibular fracture and should alert emergency physicians to the possibility of an evolving airway emergency. Computed tomography is the imaging modality of choice for confirming mandibular fractures and delineating associated injuries.[3,5]

From an emergency medicine perspective, management should prioritize early recognition of dynamic airway compromise following the primary trauma assessment. Although marked tongue elevation and floor-of-mouth swelling may appear alarming, the decision to secure the airway should be guided by physiologic deterioration rather than anatomical appearance alone. Progressive stridor, inability to handle oral secretions, hypoxemia, worsening dysphonia, or rapidly expanding swelling warrant early definitive airway management with preparation for a surgical airway when indicated.[4] In the absence of these features, carefully selected patients may be managed with close observation, serial airway reassessment, CT evaluation, and early oral and maxillofacial surgery consultation while definitive fracture management is coordinated.[5]

Despite marked tongue elevation and significant airway crowding, our patient remained clinically stable throughout repeated bedside airway assessments, allowing definitive airway intervention to be safely deferred. Temporary interdental bridle wire stabilization reduced movement of the displaced fracture segments, potentially limiting ongoing soft tissue injury while facilitating continued observation before definitive fixation. Although temporary stabilization is not a substitute for definitive airway protection or fracture fixation when clinically indicated, this case highlights the

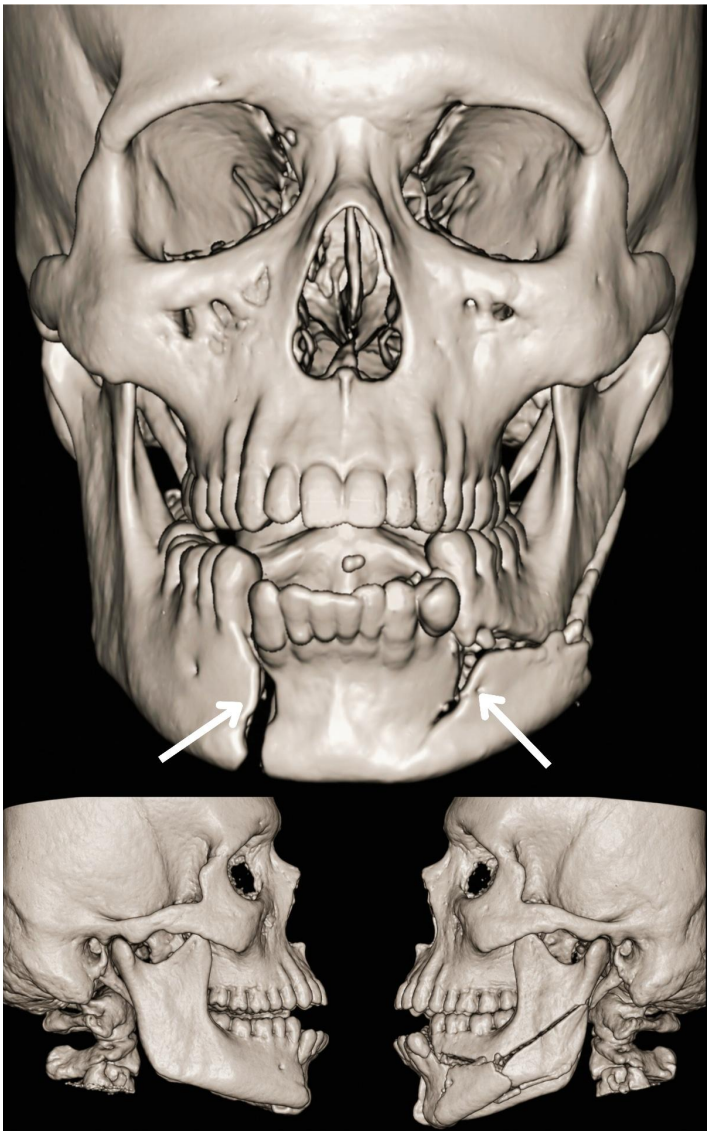


Image 2. Three-dimensional computed tomography demonstrating severely displaced bilateral mandibular fractures involving the parasymphysis and body-angle regions (arrows).

importance of repeated bedside airway evaluation, multidisciplinary collaboration, and timely temporary fracture stabilization in facilitating safe delayed definitive management in carefully selected patients with traumatic pseudo-Ludwig angina.[3-5]

Patient consent has been obtained and filed for the publication of this case report.

Address for Correspondence: Mohammed Naveeth Imran, MBBS, DNB, Department of Emergency Medicine and Trauma, Ground Floor, Emergency Medical Services Block, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Dhanvantari Nagar, Puducherry 605006, India. Email: jr8954@jipmer.ac.in; imran16497@gmail.com.

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Image 3. Temporary interdental bridle wire stabilization performed in the emergency department for mandibular segmental instability (arrows).

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Subarachnoid Pneumocephalus Following Endoscopic Transsphenoidal Surgery

Willie Dong, MD
Roger Rothenberg, MD

Temple University Hospital, Department of Emergency Medicine, Philadelphia, Pennsylvania

Section Editor: Alexander John Scumpia, MD
Submission history: Submitted April 11, 2026; Revision received June 3, 2026; Accepted June 3, 2026
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Case Presentation: A 26-year-old man with past medical history of prolactinoma presented two weeks status post endoscopic transsphenoid prolactinoma excision with a positional headache and cerebrospinal fluid rhinorrhea; he was found to have diffuse subarachnoid air.

Discussion: There are few reports of subarachnoid pneumocephalus in the emergency medicine literature. Pneumocephalus, particularly tension pneumocephalus, typically occurs in the setting of traumatic skull fractures. However, emergency physicians should be aware of subarachnoid pneumocephalus in the setting of endoscopic skull base surgeries. [Clin Pract Cases Emerg Med. 2026;10(4):665–667.]

Keywords: *pneumocephalus; subarachnoid; case image.*

CASE PRESENTATION

A 26-year-old man with past medical history of prolactinoma status post endoscopic transsphenoid excision with neurosurgery, about two weeks prior, presented to the emergency department (ED) for headache. The patient awoke on the morning of presentation with a sharp bilateral headache that worsened when sitting up. He also reported clear nasal drainage with a salty taste and postnasal drainage into the posterior oropharynx. He denied fever or other infectious symptoms. He had received postsurgical discharge instructions to present to the ED for headache or neurological symptoms. His vital signs on arrival were within normal limits. He was in no acute distress. Neurologic examination was nonfocal aside from a baseline temporal hemianopsia of the left eye attributable to his prolactinoma. Noncontrast computed tomography (CT) of the head demonstrated diffuse subarachnoid air (Images 1-3).

In the ED, neurosurgery was consulted following CT acquisition and prior to formal radiology interpretation because of the abnormal imaging findings. The patient was subsequently admitted to the neuro-intensive care unit, and a lumbar drain was placed for persistent cerebrospinal fluid (CSF) leak. On hospital day 4 he returned to the operating

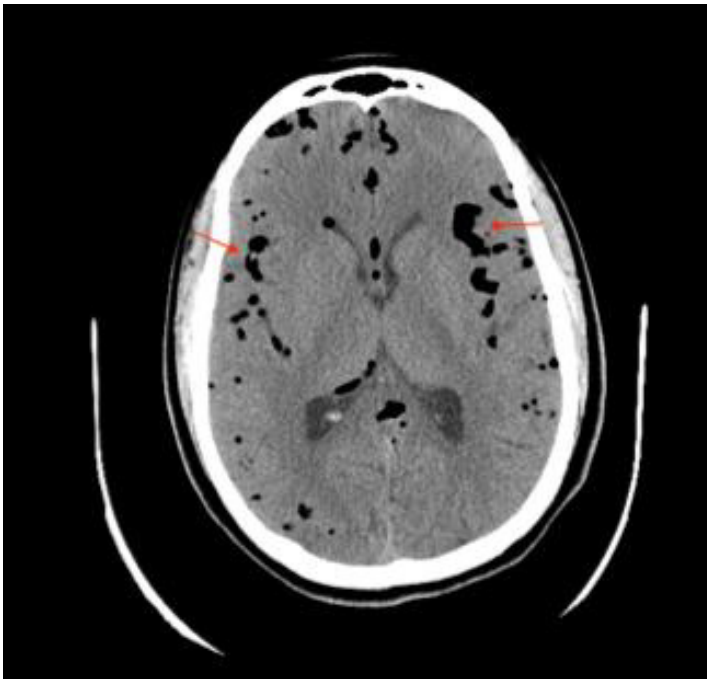


Image 1. Axial view from head computed tomography without contrast demonstrating subarachnoid air. Red arrows indicate collections of hypoattenuation, representing air in the subarachnoid space.

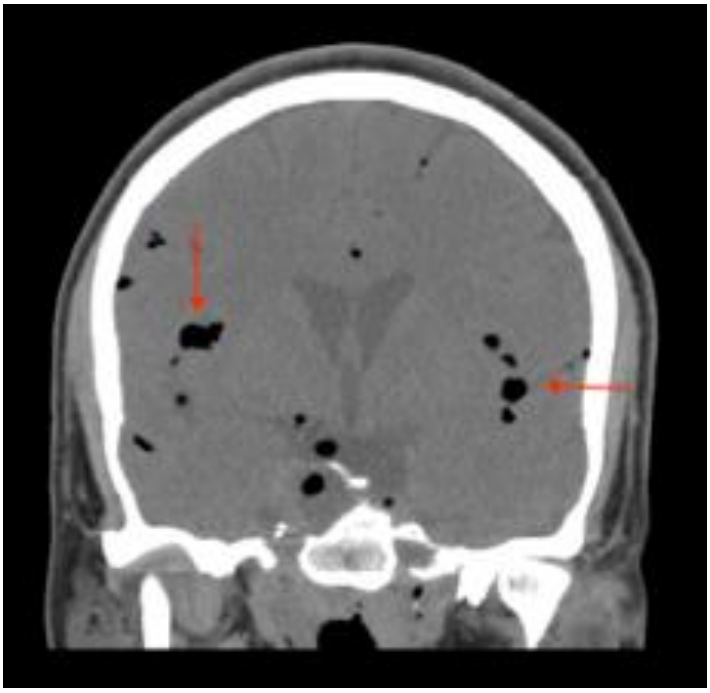


Image 2. Coronal view from head computed tomography without contrast demonstrating subarachnoid air. Red arrows indicate collections of hypoattenuation, representing air in the subarachnoid space.

CPC-EM Capsule

What do we already know about this clinical entity?
Pneumocephalus is uncommon and usually follows trauma or skull fracture. Tension pneumocephalus is a neurosurgical emergency.

What makes this presentation of disease reportable?
Diffuse subarachnoid pneumocephalus after endoscopic transsphenoidal surgery has not been reported in emergency medicine literature.

What is the major learning point?
Positional headache and salty rhinorrhea after skull-base surgery should raise suspicion for pneumocephalus and cerebrospinal fluid leak.

How might this improve emergency medicine practice?
Early recognition of red-flag symptoms and computed tomography findings can expedite neurosurgical consultation and definitive management.

room for an endoscopic CSF leak repair. The lumbar drain was removed on hospital day 10. Recurrence of CSF rhinorrhea on hospital day 14 necessitated placement of a second lumbar drain on hospital day 17 and repeat endoscopic CSF leak repair on day 19. The second drain was removed on hospital day 25. He was discharged on hospital day 27 and continues outpatient follow-up without recurrent complications.

DISCUSSION

Pneumocephalus is an uncommon finding in the ED, with most cases occurring in the setting of skull fractures.[1] There are few reports of pneumocephalus in the emergency medicine literature, and only one prior report of subarachnoid pneumocephalus following an epidural steroid injection.[2-10] Reports of pneumocephalus following endoscopic skull base surgery are similarly rare and largely confined to the otolaryngology and neurosurgical literature.[11-14] To our knowledge, no prior cases of diffuse subarachnoid pneumocephalus following endoscopic skull base surgery have been reported in the emergency medicine literature.

Clinical diagnosis of pneumocephalus can be challenging because patients may be asymptomatic or present with nonspecific symptoms, such as headache, that overlap with many other diagnoses.[1] In contrast, tension pneumocephalus is a neurosurgical emergency that may present with signs of elevated intracranial pressure, deteriorating mental status, focal neurologic deficits, cerebrospinal fluid rhinorrhea, or

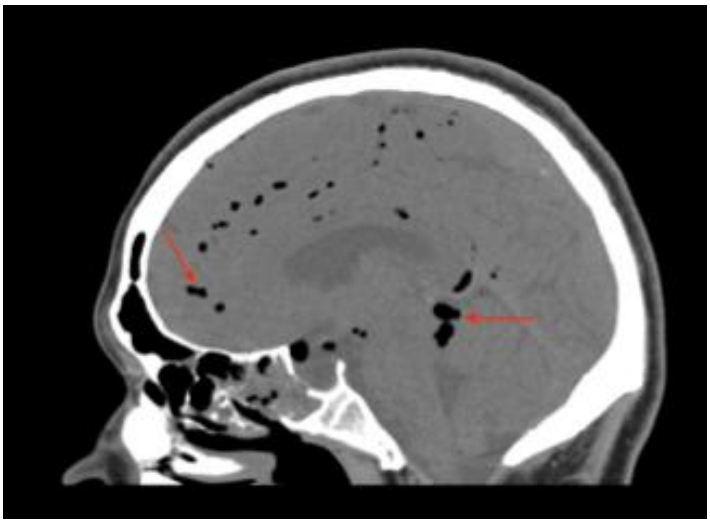


Image 3. Sagittal view from head computed tomography without contrast demonstrating subarachnoid air. Red arrows indicate collections of hypoattenuation, representing air in the subarachnoid space.

seizures. Computed tomography remains the gold standard for diagnosis of pneumocephalus and tension pneumocephalus. Classic CT findings suggestive of tension physiology include the "Mount Fuji sign" and "peaking sign" in the setting of subdural pneumocephalus or the "air bubble sign" for subarachnoid pneumocephalus.[7,8,10-14]

As demonstrated in this case, the air bubble sign is characterized by multiple air bubbles distributed throughout multiple cisterns and the subarachnoid space. Consistent with previously described "red flag" headache features, this patient reported a positional headache[15] and recent surgical intervention, prompting early neurosurgical consultation and advanced imaging. As with subarachnoid hemorrhage, emergency physicians should be able to identify this diagnosis on CT, and care should not be delayed for radiologist interpretation. Emergency physicians should maintain a high index of suspicion for pneumocephalus in postoperative patients with skull base surgeries. This consideration is particularly important in postoperative skull base surgery patients presenting with concerning features such as a positional headache, salty or metallic-tasting rhinorrhea, maximal headache on awakening, and worsening pain with Valsalva. Additional diagnoses that may present similarly, including subarachnoid hemorrhage, spontaneous intracranial hypotension, infection, and malignancy, should also be considered.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

Address for Correspondence: Willie Dong MD, Temple University Hospital Department of Emergency Medicine, 3401 N. Broad Street, Philadelphia PA, 19140. Email: Willie.Dong@tuhs.temple.edu.

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Amphetamine-induced Reverse Takotsubo Syndrome: Further Implications

Kenan Yalta, MD
Murat Gok, MD
Trakya University, School of Medicine, Cardiology Department, Edirne, TÜRKİYE

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Dear Editor:

In their recently published case report, Rabil et al[1] describe a case of amphetamine-induced reverse takotsubo syndrome accompanied by arterial thrombosis at multiple sites in a young female patient.[1] In this context, we would like to underscore further implications of amphetamine use, particularly in the context of takotsubo syndrome based on this interesting case.

First, atypical takotsubo syndrome variants have been attributed to high-risk clinical scenarios characterized by extreme levels of adrenergic discharge, potentially leading to malignant arrhythmogenesis, delayed takotsubo syndrome recovery, and coronary microvascular dysfunction.[2] In this context, disproportionate sinus tachycardia on admission might also substantiate the presence of extreme adrenergic discharge in the patient.[1] Beta blockers have been regarded as first-line agents for the management of sinus tachycardia, particularly in the setting of takotsubo syndrome episodes with supposedly extreme levels of adrenergic discharge (in the context of pheochromocytoma, etc).[3] However, initiation of ivabradine might be deemed a safer alternative to beta blockers in takotsubo syndrome patients with compensatory sinus tachycardia and/or pulmonary edema.[3] On the other hand, sinus tachycardia in the patient could be primarily attributed to metamphetamine impact,[1] which might have been more effectively reversed with the use of sympatholytic agents including short-acting beta blockers. Intravenous ultra-short acting cardioselective beta blockers such as landiolol have been used for the management of various tachyarrhythmias even in the setting of left ventricular dysfunction (in reduced doses).[4]

Second, we hold the opinion that invasive coronary angiogram should have been performed in the patient on admission due to the overt ST-segment elevation pattern on electrocardiogram, existing high-risk clinical features, and low interTAK score, all of which might denote co-existing acute coronary syndrome.[3] Mechanistically, excessive adrenergic discharge may elicit simultaneous evolution of takotsubo syndrome and spontaneous coronary artery dissection (SCAD), particularly in the female population.[5] Moreover, SCAD may arise as a primary phenomenon that might subsequently lead to takotsubo syndrome evolution in the setting of extreme stressors.[5] In this context, amphetamine use, besides its impact on the induction of arterial thrombus formation, was previously reported to induce SCAD as well as dissections in other arterial structures.[6,7] Accordingly, we recommend the evaluation of coronary arteries and other arterial structures on further imaging modalities including computed tomography coronary angiogram in the post-discharge setting.

Finally, amphetamine use may also be associated with other forms of myocardial injury through various mechanisms including direct myocardial toxicity, coronary vasospasm, etc.[1,7] Therefore, the patient may have incurred adverse myocardial remodeling and possible dilated cardiomyopathy in the long term due to the amphetamine-induced irreversible myocardial injury, potentially warranting close monitoring of the patient's cardiac function following discharge.

In conclusion, amphetamine use might induce life-threatening cardiovascular conditions that could have important diagnostic, prognostic, and therapeutic implications.

Address for Correspondence: Kenan Yalta, MD, Trakya University, School of Medicine, Cardiology Department, Balkan Yerleşkesi, 22030, Edirne, TÜRKİYE. Email: kyalta@gmail.com.

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Before Hyperemesis Gravidarum Is Blamed for Thiamine Deficiency, Alternative Causes Should Be Ruled Out

Josef Finsterer, MD, PhD Neurology & Neurophysiology Center, Vienna, Austria

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Dear Editor:

We read with interest the article by Kreutzer et al about a 22-year-old pregnant woman who developed Wernicke encephalopathy (WE) in the context of hyperemesis gravidarum at 13 weeks of gestation.[1] Thiamine deficiency was detected and attributed to the hyperemesis gravidarum.[1] The WE resolved completely after thiamine supplementation.[1] The study is promising, but some points should be discussed.

First, we disagree with the statement in the highlights that only a single case of hyperemesis gravidarum and WE has been described to date.[1] In addition to the case described, WE in association with hyperemesis gravidarum has also been reported by Kotha et al,[2] Kantor et al,[3] Bensaid et al,[4] and many other authors. A PubMed search using the search terms "WE" and "hyperemesis gravidarum" yielded over 200 results. These findings do not explain why only a single case of WE due to hyperemesis gravidarum was identified.

Second, the patient's usual diet was not documented.[1] Was there any evidence of malnutrition or alcohol dependence? Thiamine deficiency is particularly common in patients who consume refined carbohydrates such as polished white rice, white flour, and white sugar, because the outer bran and germ layers of grains are rich in thiamine. A diet high in carbohydrates and low in protein can also cause thiamine deficiency. Similarly, severely malnourished individuals and those with eating disorders are known to often have insufficient thiamine intake. Furthermore, foods high in freshwater fish, fresh shellfish, or certain ferns can degrade and destroy thiamine because they are rich in thiaminases.

Third, the patient's current medications (cefdinir, doxylamine, ondansetron, pyridoxine, and loratadine) were not considered as a possible cause of the confusion. Ondansetron, in particular, is known to induce confusion.[5] Furthermore, it is conceivable that at least cefdinir, or the combination of the four substances, impaired intestinal thiamine absorption, although these medications are not known to directly cause thiamine deficiency. We should also know which medications the patient took for hyperemesis gravidarum in the days prior to admission.

The fourth point is that the duration of the vomiting was not documented.[1] Since the patient was discharged after only five days, it is very likely that it was short-lived. Therefore, it is rather unlikely that hyperemesis gravidarum was actually responsible for the low thiamine level. Since the patient developed confusion during the five-day hospital stay, it cannot be ruled out that this was due to the urinary tract infection, its treatment, or the concurrent hyperemesis. Because thiamine deficiency can cause vomiting, it should be ruled out that the vomiting was already a manifestation of a pre-existing thiamine deficiency.

In summary, vomiting must be prolonged to be considered as a cause of thiamine deficiency. Before thiamine deficiency is considered as the cause of vomiting, other causes must be excluded.

Address for Correspondence: Finsterer J, MD PhD, Neurology & Neurophysiology Center, Postfach 20, 1180 Vienna, Austria. Email: fifigs1@yahoo.de.

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