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Clinical Practice and Cases in Emergency Medicine

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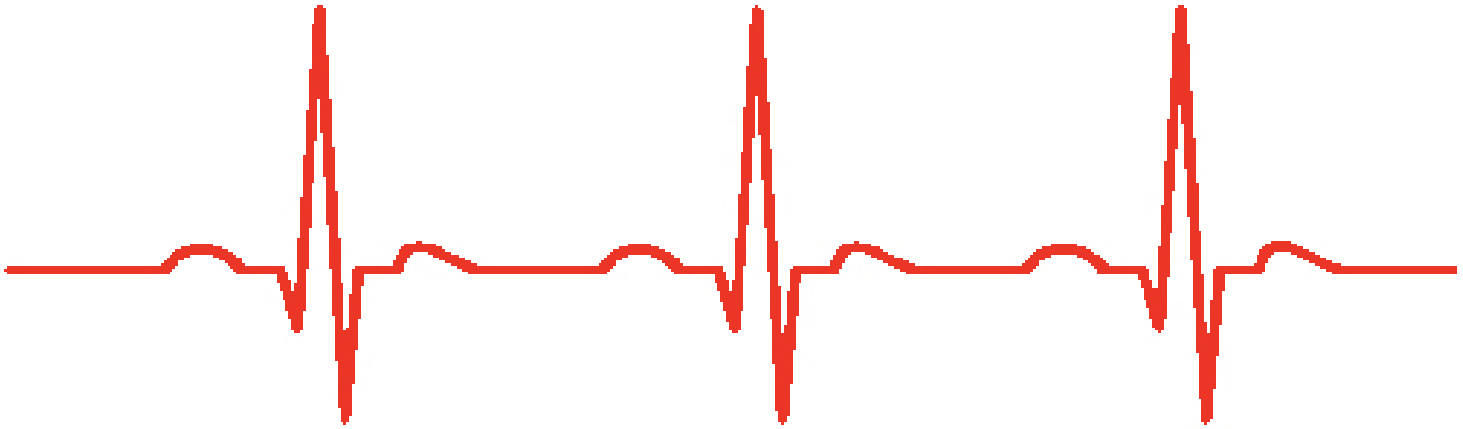
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CPCEM Clinical Practice & Cases

Bell Palsy Mimics: Lessons from Four Malpractice Cases

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Introduction: Bell palsy, an idiopathic dysfunction of the seventh cranial nerve, is the leading cause of unilateral facial paralysis, although other more serious entities such as stroke, infection, and tumor may present similarly, leading to both medical and legal risks in cases of misdiagnosis.

Case Series: We present four malpractice cases revolving around misdiagnosis of Bell palsy. These cases alleged failure to diagnose, failure to obtain informed consent, and failure to provide appropriate discharge instructions. Outcomes ranged from a jury verdict in favor of the physician, to an out-of-court settlment for $400,000, to a jury verdict in favor of the patieint for over $3.1 million.

Conclusion: Bell palsy is the most common cause of unilateral facial paralysis. While the diagnosis can be made at the bedside without advanced testing, doing so requires a clear understanding of the pathophysiology of the disease, an appreciation for the role of advanced diagnostics, and thorough documentation of a supportive history and physical exam. Misdiagnosis or mismanagement confers both clinical and legal risks. [Clin Pract Cases Emerg Med. 2025;19(2):125-128.]

Keywords: *Bell palsy; malpractice; medicolegal; lawsuit.*

INTRODUCTION

Bell palsy, often diagnosed in the emergency department (ED), is a relatively benign, idiopathic condition that manifests as unilateral facial weakness, thought to be secondary to inflammation of the seventh cranial (facial) nerve.[1,2] It is the most common cause of unilateral facial paralysis, a symptom also seen in other serious conditions such as stroke, infections, and tumors.[1] The diagnosis generally can be made based on history and physical examination, which often demonstrates fairly abrupt onset of unilateral symptoms involving weakness or paralysis of facial muscles including the forehead, a sensation of facial numbness, alterations in taste, and hyperacusis. Differential diagnoses include any other conditions that may affect the cranial nerve including stroke, tumors, vasculitides, and infections such as Lyme disease and herpes viruses.[3]

Given the broad spectrum of causes of facial paralysis, the opportunities to misdiagnose Bell palsy are numerous. The literature suggests the misdiagnosis rate to be anywhere from 1%-20%.[3,4,5] Given the multiple dangerous and treatable etiologies that may mimic Bell palsy, the clinical and legal risks of misdiagnosis are high. Here, we discuss four medical malpractice cases centered around Bell palsy, drawing attention to common clinical characteristics of missed diagnoses and steps clinicians can take that may mitigate their malpractice risks.

CASE SERIES

Case 1: *Hamilton*

A 60-year-old male presented to the ED with slurred speech, left-sided facial weakness, and deviation of his tongue. The defendant emergency physician diagnosed the patient with Bell palsy. After discharge, the patient's symptoms worsened, and he was subsequently diagnosed with a stroke. He alleged that the physician's failure to diagnose this at his first visit led to his poor outcome, including permanent motor and cognitive impairment. The case settled for $400,000.[6]

Case 2: *Taite*

A 42-year-old male with a history of hypertension and diabetes presented to the ED with right-sided facial droop, slurred speech, and dizziness. He was initially examined by a resident physician, who diagnosed him with Bell palsy based on his lack of arm or leg weakness. No imaging was obtained, a supervising physician agreed with the assessment, and the patient was discharged. The next day he returned with more severe symptoms and was found to have had a large hemorrhagic stroke, from which he subsequently died five days later. After two years of litigation and a jury trial, the physicians in this case were ultimately found not negligent in the care provided.[7]

Case 3: *Jandre*

A 53-year-old male presented to the ED with dizziness, slurred speech, and facial weakness. The defendant emergency physician diagnosed the patient with Bell palsy and discharged the patient. The patient developed permanent neurologic deficits secondary to a stroke and sued the physician both for failing to diagnose and treat the stroke and for failing to inform the patient of additional options for diagnosis. The case went to trial, and a verdict was rendered for $3,106,433.[8]

Case 4: *Neu*

A 59-year-old female was diagnosed with Bell palsy after presenting for evaluation of ear pain and facial weakness. No imaging was performed. No follow-up was recommended. The patient's symptoms persisted, and she was ultimately diagnosed with parotid gland cancer eight months later, which had widely metastasized by that time. The lawsuit, alleging failure to obtain appropriate imaging and failure to diagnose, was ultimately unsuccessful as it was filed after the statute of limitations.[9]

CPC-EM Capsule

What do we already know about this clinical entity?
Bell Palsy, often diagnosed in the emergency department (ED), causes unilateral facial weakness due to facial nerve inflammation but can mimic serious conditions like stroke or tumors.

What makes this presentation of disease reportable?
Four malpractice cases on Bell Palsy highlight characteristics of missed diagnoses and key steps clinicians can take to reduce liability.

What is the major learning point?
Clinicians should identify atypical Bell Palsy symptoms, like tongue deviation and dizziness, and recognize risk factors for conditions like acute stroke.

How might this improve emergency medicine practice?
Careful history, exam, and documentation, including shared decision-making and discharge instructions, can aid accurate diagnosis and reduce legal risks.

DISCUSSION

We present four cases involving serious conditions that were misdiagnosed as Bell palsy. While Bell palsy is generally considered a clinical diagnosis, meaning a condition that can be identified based on a clinical history and physical exam, being able to make this diagnosis confidently requires thoroughly understanding the pathophysiology of the condition and recognizing when a bedside exam is not sufficient. As stated above, Bell palsy is thought to be due to inflammation of the seventh cranial nerve, which normally controls a) muscles of the face (raising eyebrows, shutting eyelid, wrinkling nose, smiling); b) muscles of the middle ear that regulate sound; c) taste on the anterior two-thirds of the tongue; d) output from the submandibular and sublingual salivary glands; and e) output from the lacrimal ducts.

Traditionally, Bell palsy is diagnosed in patients with dysfunction of the seventh cranial nerve who may present with unilateral facial weakness or paralysis, hyperacusis, change in normal taste perception or saliva production, and change in tear production. Some patients endorse pain near the ear on the affected side overlying the facial nerve itself. These symptoms tend to appear rather abruptly and reach maximum intensity within hours to days. Symptoms that fall outside this constellation should prompt clinicians to consider alternative diagnoses. Treatment consists of corticosteroids to address the inflammation with or without concomitant antiviral therapy.[10] The majority of patients will recover full function, while about 5-20% will have persistent deficits.[3] Those who do not show response to treatment within three weeks warrant additional evaluation to assess for alternative causes.[11] Treatable diagnoses that may present with overlapping symptoms include ischemic stroke, hemorrhagic stroke, intracranial tumor, extracranial tumor, viral infections (eg, herpes), and bacterial infections (eg, Lyme disease). Being aware of these mimics is critical in not overlooking their presence.

Clinicians should be adept at recognizing patients who have symptoms that are not consistent with Bell palsy. For example, in the first case, the patient presented with tongue deviation. The muscles of the tongue are controlled by the hypoglossal nerve, cranial nerve XII, not the facial nerve, as is affected in Bell palsy. Identification of tongue deviation in the

setting of a unilateral facial paralysis suggests a central process affecting multiple cranial nerves and warrants a more thorough evaluation. Maintaining a solid knowledge of the facial nerve anatomy and function is crucial in recognizing these subtle differences, and meticulously documenting this exam is critical for supporting a bedside diagnosis.

In the second and third cases, a patient presented with unilateral facial weakness and slurred speech, symptoms that could be consistent with Bell palsy, but they also endorsed dizziness. Dizziness is the most overlooked symptom in missed diagnosis of strokes and is not consistent with the diagnosis of Bell palsy.[12] Any co-occurrence of dizziness should prompt evaluation for alternative diagnoses. A review of 69 malpractice cases of reported dizziness as a missed symptom of acute stroke revealed that patients had poor outcomes due to a missed or delayed central nervous system diagnosis, most commonly in the ED setting.[13] The majority of these patients exhibited at least one additional neurologic symptom in addition to dizziness, as did the patient in this malpractice case.[13] These findings underscore the importance for emergency physicians to consider alternative explanations in patients presenting with dizziness or any other symptoms that do not fit into the constellation of symptoms caused by dysfunction of the facial nerve.

The patient in the second case also had multiple risk factors for stroke, including hypertension, diabetes, and polysubstance use. One study analyzing ED misdiagnoses of Bell palsy found that more than one-quarter of these misdiagnoses were ischemic stroke.[5] While the diagnosis of Bell palsy can usually be made without advanced diagnostics, all patients—especially those with stroke risk factors—require careful consideration and documentation. In this case, we are told that physicians relied on the patient's lack of arm or leg weakness to exclude a stroke. This is not a sufficient clinical history or exam in any patient, but especially not in a patient with a higher pre-test probability for stroke based on his risk factors. History and exam focused on ruling out more dangerous etiologies *and* thorough documentation of both of these is crucial.

In the third case, from Wisconsin, the court found the physician to be negligent in part for not discussing the option of advanced imaging with the patient whose symptoms were atypical for Bell palsy. That court determined that the physician should have engaged in an informed consent discussion with the patient, stating that the physician has a duty to disclose diagnostic options that reasonable patients would want to know to make informed decisions about their care.[8] This finding is remarkable in that it seemingly extends the scope of traditional informed consent beyond the historical focus on treatments and also applies it to diagnostic tests. While this court decision applies only in Wisconsin, it may be indicative of a larger shift in the courts toward a more patient-centered standard of care in which patients are expected to be invited to be a part of the decision-making. For this reason, having a low threshold to engage in shared decision-making (and documenting these discussions) may also serve to mitigate legal risks.

In the fourth case, a patient had symptoms including unilateral facial weakness and ear pain that could be consistent with Bell palsy. However, she did not respond to treatment over several months and received no instructions for follow-up. While some patients with Bell palsy will have permanent deficits, the majority will recover full function, and those who do not recover warrant additional evaluation. Failing to appreciate or communicate these follow-up parameters to patients at discharge can put them at risk, as occurred when this patient's undiagnosed parotid gland cancer metastasized. While this additional evaluation is generally beyond the scope of the ED, discussing that such an evaluation may be necessary *is* the responsibility of the diagnosing physician. Documenting this discussion may help thwart any later allegations that such a conversation did not occur, and when this documentation is provided in the discharge instructions, it can help ensure patients have an avenue to revisit and better understand those recommendations, too.

CONCLUSION

Bell palsy is the leading cause of unilateral facial paralysis, often presenting with a variety of symptoms consistent with dysfunction of the seventh cranial nerve. Patients with facial paralysis frequently seek emergency care due to the sudden onset and distressing nature of the condition. Given that more serious and potentially treatable conditions can also present with facial weakness, it is essential to conduct a comprehensive history and physical examination to differentiate Bell palsy from life-threatening causes, including acute stroke. From a legal perspective, fully documenting the care provided, including any shared decision-making and complete discharge instructions, may help mitigate downstream risks.

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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Ultrasound-Guided Erector Spinae Plane Block for Breakthrough Pancreatic and Hepatobiliary Malignancy Pain in the Emergency Department: A Case Series

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Introduction: Breakthrough pain is frequently experienced by patients with gastrointestinal malignancies and is a common reason for presenting to the emergency department (ED). After ruling out acute pathology, ED management typically consists of intravenous opioids, although high doses of opioids can be associated with potentially severe adverse events and complications in certain high-risk populations. Regional anesthesia strategies, such as the erector spinae plane block (ESPB), have been shown to be effective for several etiologies of non-malignant visceral abdominal pain. In this case series we sought to evaluate whether the ESPB can be effective for ED patients with breakthrough pancreatic and hepatobiliary cancer pain.

Case Series: Three patients with breakthrough hepatopancreatobiliary cancer pain underwent successful ESPBs performed by an emergency physician in the ED. All patients reported considerable reduction in their pain. Two patients with cancer of the pancreatic head reported complete pain relief and were able to be discharged from the ED. The third patient with metastatic colorectal cancer involving the hepatobiliary system was admitted for further medical workup, although he did not require any additional analgesics for nearly 13 hours after the block.

Conclusion: The erector spinae plane block appears to be a safe and effective strategy for managing breakthrough pain related to pancreatic and hepatobiliary malignancy in the ED. [Clin Pract Cases Emerg Med. 2025;19(2):129-133.]

Keywords*: ultrasound-guided regional anesthesia; erector spinae plane block; cancer pain; pancreatic cancer; emergency department.*

INTRODUCTION

Nearly half of all patients with gastrointestinal (GI) cancer suffer from chronic abdominal pain.[1] In particular, pancreatic cancers and certain hepatobiliary malignancies are among the most painful cancers, with as many as 80% experiencing considerable pain.[2] Even among patients with well-controlled background pain, more than half will have breakthrough pain. Cancer patients with breakthrough cancer pain frequently present to the emergency department (ED) for control of this pain. Breakthrough pain is the most common complaint for patients with cancer who present to the ED.[3] Emergency department management typically consists of intravenous opioids, although many cancer patients still have considerable pain despite this treatment. Furthermore, opioids are associated with significant adverse effects, including dependence and tolerance, delirium, nausea and vomiting, and respiratory depression.[4]

The erector spinae plane block (ESPB) is a relatively new regional anesthesia technique that is increasingly being used for analgesia and anesthesia in the ED due to its favorable

safety profile.[5] Outside the perioperative setting, the ESPB is most commonly used for managing pain associated with rib fractures and has been effective in reducing pain and decreasing opioid utilization among hospitalized patients with rib fracture.[6] A recent randomized controlled study of ED patients with acute hepatopancreatobiliary pain demonstrated that ESPBs performed by emergency physicians (EP) resulted in significantly lower pain scores and a reduced need for rescue analgesia than patients managed with standard analgesia alone.[7] As has been documented in a few case reports, the ESPB has also been used to manage refractory breakthrough malignancy pain in patients with colon cancer[8,9] and cholangiocarcinoma.[7,10] We present a series of ED patients who underwent an EP-performed ESPB for refractory pancreatic and hepatobiliary malignancy pain.

CPC-EM Capsule

What do we already know about this clinical entity?
The erector spinae plane block (ESPB) is more frequently being used in the emergency department (ED) for thoracoabdominal wall and abdominal visceral pain.

What makes this presentation of disease reportable?
These are the first reported cases of an ESPB used for management of breakthrough pain from pancreatic cancer.

What is the major learning point?
The ESPB might be an effective analgesia strategy to manage breakthrough pain from pancreatic cancer and other abdominal gastrointestinal cancers in the ED.

How might this improve emergency medicine practice?
The ESPB may enable emergency physicians to manage breakthrough cancer pain, thereby reducing the amount of potentially harmful opioid analgesics.

CASE SERIES

Procedure

The ESPB was performed as described in Forero et al 2016.[11] After consenting for the procedure, patients were placed on a cardiac monitor and positioned seated on the edge of the bed. A 5-15 megahertz curvilinear abdominal probe was placed on the right posterior trunk just lateral to the midline in a caudal-cephalad orientation with the indicator facing cranially (Image 1). The erector spinae muscle and transverse processes were visualized with ultrasound, and the ultrasound probe was centered at the sixth thoracic vertebra. The surrounding area was prepped with chlorhexidine in standard fashion, and a small wheal of lidocaine was raised in the skin at the spinal needle insertion site. A Quincke spinal needle was introduced through the skin in a cephalad to caudad direction and advanced in-plane under sonographic visualization until the needle tip reached immediately deep to the erector spinae muscle, slightly superior to the transverse process. The syringe was aspirated to ensure the needle tip was not placed intravascularly.

A small test injection of normal saline was used to confirm correct needle-tip placement within the fascial plane between the erector spinae muscle and the transverse process. Weight-based bupivacaine 0.5% was administered in 5 milliliter (mL) aliquots, while observing anesthetic spread within this fascial plane (Image 2). We expanded the volume of anesthetic by diluting it with normal saline to allow for maximal spread to target nerves, using between 15 and 30 mL of 0.9% normal saline. We opted for bupivacaine to allow for the greatest duration of analgesia. All patients were maintained on the cardiac monitor before blockade and were admitted to a telemetry unit.

Cases

Case 1. A 67-year-old man with a known history of unspecified primary malignancy of the pancreatic head and chronic lymphocytic leukemia presented with acute on chronic epigastric abdominal pain, 10/10 in severity, refractory to his outpatient analgesic regimen. He was hemodynamically stable, and laboratory and imaging workup did not reveal any acute abnormalities. He underwent a right-sided ESPB with 20 mL of 0.5% bupivacaine mixed with 20 mL of normal saline without complications. Following the block, he reported complete (0/10) improvement in his pain. He was discharged home and did not return to the ED until approximately one month later when he had another episode of breakthrough cancer pain.

Case 2. A 68-year-old woman, also with a known history of unspecified cancer of the pancreatic head, presented with severe 10/10 acute on chronic epigastric pain attributed to her cancer. After an otherwise unremarkable ED workup, she underwent a right-sided ESPB with 15 mL of 0.5% bupivacaine mixed with 15 mL normal saline, which completely resolved her pain. She was discharged from the ED, although she returned approximately 48 hours later due to recurrence of her cancer pain.

Case 3. A 68-year-old man with a history of colorectal cancer and known metastases to the liver and biliary system presented with severe epigastric and right upper quadrant abdominal pain. He was initially treated with 2 milligrams of hydromorphone, which improved his pain from 10/10 to 8/10.

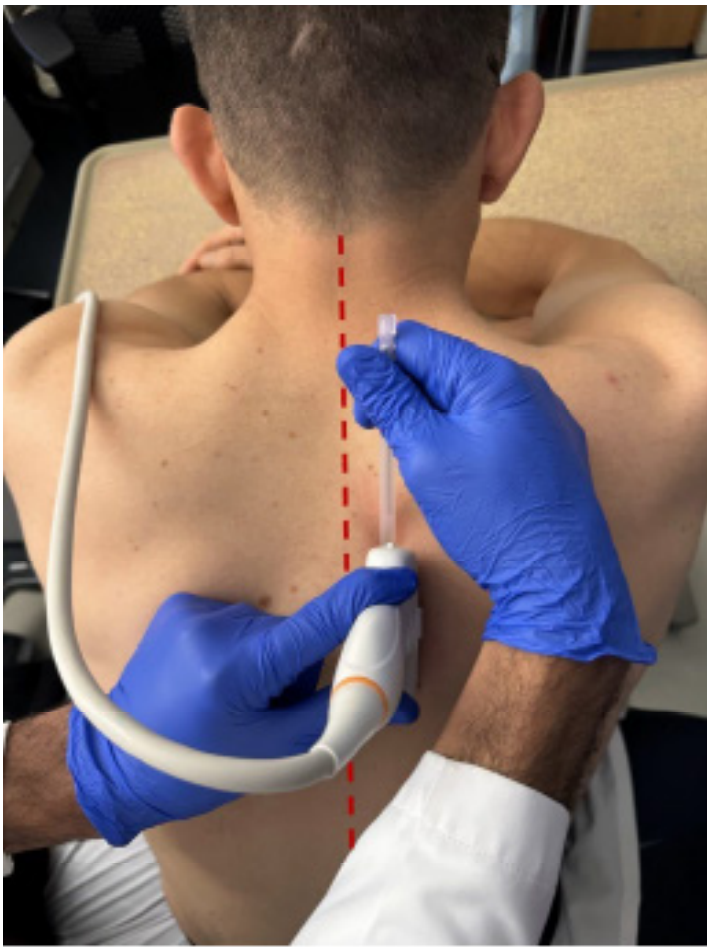


Image 1. Patient set-up and probe orientation for thoracic erector spinae plane block. The patient is seated on the edge of the bed with arms resting on a side table. A curvilinear probe is oriented on the right paraspinal region with the indicator facing cranially, while the needle is inserted in a craniocaudal direction. Midline is indicated on the patient model with a dashed line.

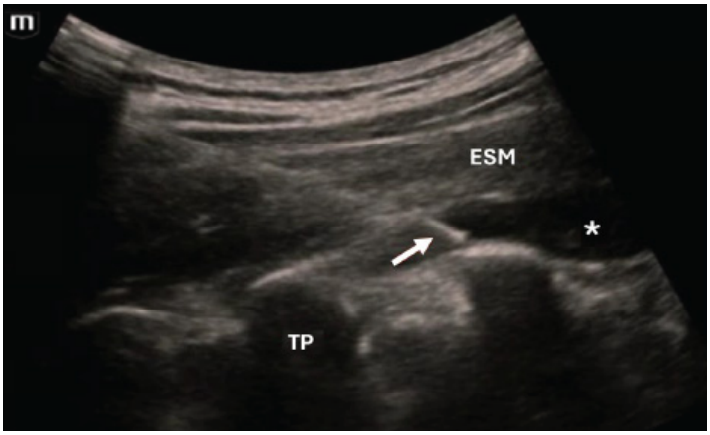


Image 2. Sonographic anatomy of the erector spinae plane block. The arrow points to the needle tip, with the asterisk indicating spread of anesthetic injected within the fascial plane between the erector spinae muscle and the transverse process. The "m" in the upper left of the figure shows the location of the probe indicator, which is facing cranially relative to the patient.
ESM, erector spinae muscle; *TP*, transverse process.

Given this negligible improvement, he underwent a right-sided ESPB with 25 mL of 0.5% bupivacaine plus 25 mL of normal saline. Following the block, his pain improved to 3/10, although given the complexity of his cancer and concern for worsening involvement within the biliary system, he was admitted to the oncology service for pain control and additional medical workup. Notably, he did not require any additional analgesics until 13 hours after the block.

DISCUSSION

In this case series, we demonstrate the successful use of the ESPB to manage breakthrough hepatopancreatobiliary cancer pain in the ED. To our knowledge, these are the first reported cases of EP-performed ESPBs used to manage breakthrough pain from pancreatic cancer. Moreover, we demonstrate another successful case of an EP-performed ESPB to manage refractory pain from colorectal carcinoma with hepatobiliary metastases.[8] Following the ESPBs, both patients with breakthrough pancreatic cancer pain were able to be discharged from the ED, avoiding hospitalization for pain control. The latter patient with metastatic colorectal cancer was ultimately admitted for additional medical workup, although he did not require additional analgesics for 13 hours after the block. Given how frequently cancer patients seek emergency care for breakthrough pain, the ESPB may be a promising component of a multimodal analgesia strategy to manage breakthrough abdominal malignancy pain in the ED.

Compared to other truncal nerve blocks that only target the somatic innervation of the thoracoabdominal wall, the ESPB also provides sensory analgesia to visceral abdominal organs through blockade of the sympathetic chain.[12] In an ESPB, local anesthetic is administered within the fascial plane between the erector spinae muscle and the transverse processes, where it can spread several vertebral levels cranially and caudally to anesthetize multiple spinal nerve roots with a single injection.[11] Additionally, anesthetic simultaneously diffuses anteriorly into the paravertebral space to surround the sympathetic chain, thereby enabling blockade of sympathetic sensory afferents from the gastrointestinal tract.[13] This allows the ESPB to provide analgesia for patients in significant pain from numerous abdominal visceral pathologies, including colorectal, hepatobiliary, pancreatic, and likely other sources of gastrointestinal malignancy.[7–9] Since the afferent sympathetic neurons from the abdominal viscera converge prior to splitting into the left and right thoracic sympathetic chains, only a unilateral ESPB is required to provide analgesia.

Cancers of the pancreas and hepatobiliary system are notoriously painful and often poorly responsive to standard

analgesia.[14] As a result, a variety of multimodal analgesia strategies, including celiac plexus blocks, have been explored for managing refractory pancreatic cancer pain, although their efficacy has been somewhat inconsistent.[2,14] These can occasionally lead to severe complications such as diaphragmatic paralysis, pneumothorax, retroperitoneal injuries, damage to abdominal viscera, and neurovascular damage due to the trajectory of the needle very close to several critical structures.[1] The superficial needle trajectory in the ESPB and its reliable diffusion of anesthetic to the sympathetic chain may allow for safe visceral analgesia without the need for advanced imaging equipment or the expertise of interventional radiologists or endoscopists.

With the early success of the ESPB for managing visceral abdominal pain, it would be reasonable to consider augmenting these blocks with dexamethasone or other adjuvants to potentially prolong the duration of analgesia. For select patients who will ultimately require admission, EPs can also consider coordinating with anesthesiologists to place regional nerve block catheters in the ED to initiate continuous infusion of anesthetic. These strategies may allow EPs to initiate early, safe, and effective analgesia for cancer patients in the ED, which will continue to provide benefit well into their hospitalizations.

Caring for patients with GI malignancies with breakthrough cancer pain requires a multidisciplinary approach between the EP and the consulting oncologists and gastroenterologists. For patients awaiting admission to the hospital for continued medical care, the EP should coordinate with consultants prior to performing a block to inform them of the planned procedure and to elucidate any potentially unknown contraindications. This will not only improve the lines of communication between the ED and consultants to improve continuity of care but also ensure these patients receive appropriate monitoring in the hospital.

The ESPB should only be performed by EPs with either prior experience in regional anesthesia or at least experience with in-plane, ultrasound-guided needle placement, such as is used in placing peripheral intravenous lines. Although a rare complication, the ESPB can lead to pneumothorax, especially if an EP employs an overly acute needle trajectory that could allow the needle to enter the pleura. However, the naturally obtuse needle trajectory with the ESPB usually causes an overshot needle to back-wall to the next transverse process. Furthermore, all physicians who perform regional anesthesia, especially plane blocks such as the ESPB, which employ large volumes of anesthetic to allow for maximal spread to distant nerves, should always be aware of how to mitigate local anesthetic systemic toxicity. This can usually be avoided by calculating the local anesthetic dose based on the patient's ideal body weight (maximum of 2.5 mg per kilogram for bupivacaine), aspirating prior to injecting, and only injecting increments of 5 mL of anesthetic at a time while monitoring for patient response. Lastly, patients being worked up for a surgical abdomen likely should not receive an ESPB due to masking their pain on physical exam, which may delay surgical treatment. However, patients who are being admitted for planned surgical intervention are still candidates for an ESPB for analgesia.

CONCLUSION

The erector spinae plane block appears to be a safe and effective strategy for managing breakthrough pain related to pancreatic and hepatobiliary malignancy in the ED. Implementation of the ESPB in the ED for patients with visceral malignancy may improve pain, reduce the use of opioids, and in some cases avoid the need for hospitalization. Future studies should evaluate the efficacy of the ESPB on ED patients presenting with breakthrough pain from other abdominal visceral malignancies. Moreover, future investigation should aim to better compare the efficacy of the ESPB to conventional analgesia strategies among ED patients with breakthrough GI malignancy pain.

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

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Chronic Nitrous Oxide Toxicity Despite Elevated Serum Vitamin B_{12} Level: A Case Report

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Introduction: Nitrous oxide (N_2O) toxicity is an uncommon but important-to-recognize presentation of neurologic deficits and hematologic abnormalities, which may never resolve in some patients. In the United States, nitrous oxide is legal to possess and easily obtainable for purchase in stores and online. Nitrous oxide abuse and its long-term sequelae must be recognized by the emergency physician to ensure proper follow-up and maximize neurologic outcomes.

Case Report: A 28-year-old male with past medical history of alcoholism and polysubstance abuse presented to the emergency department with progressive paresthesias, shortness of breath, and neurologic complaints following daily inhalation of N_2O for three weeks. He was diagnosed with N_2O toxicity due to functional vitamin B_{12} deficiency in the setting of elevated B_{12} levels from prophylactic self-supplementation.

Conclusion: While most recreational users of nitrous oxide will experience transient neurologic symptoms resolving within minutes of cessation, frequent or heavy users may develop permanent neurotoxicity. Exposed patients require close follow-up with neurology and vitamin B_{12} supplementation to maximize neurologic recovery. In this patient, there was persistence of neurologic symptoms over 24 hours after cessation of use despite self-supplementation of vitamin B_{12}. [Clin Pract Cases Emerg Med. 2025;19(2):134-137.]

Keywords: *case report; nitrous oxide toxicity; neurotoxicity; functional vitamin B_{12} deficiency.*

INTRODUCTION

Nitrous oxide (N_2O), also known as "laughing gas," is an inhaled compound commonly used for medical and dental procedures for anesthesia and anxiolysis. It is also used commercially for food preservation, fuel combustion, and aerosolization of food products (such as whipped cream), making it widely available to purchase. Nitrous oxide is abused recreationally due to its euphoric side effects and hallucinogenic properties, and the prevalence of abuse has increased in recent years.[1] The onset of effects occurs within seconds of inhalation. In the vast majority of users, the effects of N_2O dissipate within minutes. However, prolonged use may create dose-dependent effects resulting in persistent neurologic deficits after cessation of the drug.

These neurologic deficits are related to the drug's impact on the body's utilization of vitamin B_{12}, creating a functional B_{12} deficiency.[2] The mainstay of treatment involves cessation of the drug and supplementation with vitamin B_{12}. As the popularity of recreational use of N_2O rises, online communities of users have recommended prophylactically supplementing B_{12} to its members; however, as demonstrated by this case report, prophylactic vitamin B_{12} supplementation may not prevent neurotoxicity.

CASE REPORT

The patient was a 28-year-old male with a history of alcoholism, cocaine use, and marijuana use. He presented to the emergency department (ED) with a chief complaint of paresthesias and neurologic symptoms after prolonged use of recreational N_2O. The patient reported buying a commercial-sized tank of N_2O from an online retailer three weeks prior to presentation. He described inhaling the N_2O repeatedly since obtaining it and discontinuing its use just over 24 hours prior to presentation to the ED. He reported inhaling directly from the tank an estimated at 30-60 times per hour during awake hours daily. In addition to the N_2O use, he was also smoking marijuana daily and using cocaine and alcohol every few days.

He reported onset of progressive symptoms over the prior three to five days, consisting of numbness and tingling of the extremities, "brain fog," headaches, visual hallucinations, chest pain, dyspnea, and nausea. He reported using inhaled N_2O in the past without any prolonged symptoms. He was concerned that his symptoms were not resolving despite cessation of N_2O use over 24 hours prior to presentation. He reported symmetric ascending paresthesias initially involving the lower extremities, which were now present to a lesser extent in the hands and wrists, described as tingling and numbness without motor deficits. He complained of a holocephalic, pressure-like headache and intermittent visual hallucinations described as "shadows" in his peripheral vision. He reported that he was a member of an online community of N_2O users, and based on information provided on their forum, he had been prophylactically taking 1,000 micrograms daily of oral vitamin B_{12} supplementation for the prior three weeks.[3]

The patient was observed to be awake, alert, and fully oriented. He presented via private vehicle to the ED where his initial vitals were notable for mild tachycardia (heart rate of 108 beats per minute [bpm]), but otherwise within normal limits. He was noted to be anxious-appearing and tremulous. His neurologic exam revealed diminished sensation to light touch in the lower extremities below the knees and in the bilateral upper extremities distal to the mid forearms. His sensory deficits worsened distally in all extremities and were symmetric. His cranial nerves were intact. His motor function remained intact and was 5/5 globally. He ambulated with steady gait. His cardiopulmonary exam was unremarkable aside from tachycardia, and there were otherwise no positive findings on his remaining examination.

In the ED, intravenous (IV) access was obtained, and cardiac monitoring was established. He had blood and urine laboratory studies ordered, and neuroimaging was performed. The poison center was consulted. A 1,000 milliliter (mL) normal saline IV fluid bolus was given, as well as 4 milligrams (mg) ondansetron and 2 mg lorazepam IV. He was given 600 mg oral ibuprofen and 650 mg acetaminophen for his headache. A point-of-care glucose measurement was 109 mg per deciliter (dL) (reference range: 70-90 mg/dL). His laboratory studies revealed a complete blood count with differential without significant abnormalities and a comprehensive metabolic panel with a mildly elevated total bilirubin 1.4 mg/dL (0.7-1.2 mg/dL), a slightly elevated aspartate transaminase 37 Units (U) per liter (L) (5-34 U/L), and otherwise normal values. His magnesium level was minimally low at 1.5 mg/dL (1.6-2.6 mg/dL).

A venous blood gas revealed venous pH of 7.45 (7.35-7.43), partial pressure of carbon dioxide of 32 millimeters of mercury (mm Hg) (41-51 mm Hg), partial pressure of oxygen of 32 mm Hg (30-50 mm Hg), and bicarbonate of 24 millimoles (mmol) per L (24-28 mmol/L). A venous lactic acid level was 2.10 mmol/L (0.36-1.25 mmol/L). His total creatine kinase was 159 U/L (30-200 U/L). His ethanol, acetaminophen, and salicylate levels were undetectable. A urine toxicology screen was positive for cannabinoids and cocaine. Thyroid-stimulating hormone level was within normal limits. His methemoglobin level was 0.5% (0.5-1.5%). Vitamin B_{12} level was elevated at greater than 2,000 picograms (pg) per mL (213-816 pg/mL). A computed tomography of the head without IV contrast demonstrated no acute intracranial

CPC-EM Capsule

What do we already know about this clinical entity?
Chronic nitrous oxide (N_2O) abuse causes neurologic deficits, which may be permanent but improved with cessation of the drug and vitamin B_{12} supplementation.

What makes this presentation of disease reportable?
This chronic user of N_2O prophylactically self-supplemented with vitamin B_{12} but developed neurologic toxicity despite supratherapeutic levels of B_{12}.

What is the major learning point?
Nitrous oxide abuse creates a functional B_{12} deficiency, which makes prophylactic self-supplementation of B_{12} ineffective in preventing neurotoxicity.

How might this improve emergency medicine practice?
Prophylactic self-supplementation of B_{12} is not well-documented in the literature. This case helps broaden our understanding of the impacts of N_2O on patients.

abnormalities. An electrocardiogram showed a sinus tachycardia with a rate of 104 bpm, without any ischemic changes or notable abnormalities. A high-sensitivity troponin was less than 4 nanograms (ng) per L (less than or equal to 35 ng/L). A two-view chest radiograph demonstrated no acute cardiopulmonary disease.

He was given 2 grams IV magnesium supplementation and observed. Upon re-evaluation, his headache had resolved, and he was no longer tachycardic. He remained hemodynamically stable. He appeared less tremulous but still complained of persistent paresthesias, unchanged from his initial exam. His presentation and laboratory studies were discussed with the toxicology fellow at the Illinois Poison Center, who agreed that his presentation was consistent with chronic N_2O toxicity and recommended cessation of N_2O use plus empiric vitamin B_{12} supplementation of 2,000 mcg/day (despite an elevated vitamin B_{12} level on the patient's presenting labs).

The poison center advised close outpatient follow-up with either neurology or a toxicology clinic and to obtain outpatient magnetic resonance brain imaging. Furthermore, he was asked to monitor for progression or resolution of his symptoms. In this case, the toxicology clinic was located approximately three hours driving distance from the patient's home, so he preferred to follow up with a local neurology clinic. He was given a referral to neurology for follow-up and a dose of 2,000 mcg of oral vitamin B_{12} was given while in the ED. The prognosis was discussed with the patient, including possible permanent deficits, and he was urged strongly to discontinue N_2O use and all other illicit drug use. He was given a prescription for oral vitamin B_{12} supplementation of 2,000 mcg/day and agreed to follow up outpatient closely with neurology. Unfortunately, at the time of this case report completion, the patient had not responded to the neurology clinic's attempts to schedule him for a follow-up evaluation. Neither did he respond to the emergency physician's follow-up calls.

DISCUSSION

A literature review of this topic included mostly case reports with the patients presenting after prolonged use and with a clinical syndrome expected of vitamin B_{12} deficiency, including both hematologic and neurologic abnormalities. Most case reports in the current literature were associated with low to normal serum vitamin B_{12} levels.[4,6] There were a few case reports of sensory neuropathies occurring in patients with elevated serum vitamin B_{12} levels, as seen in this case. Our case was unique, given the patient's involvement in an online community of recreational N_2O users who recommend prophylactic self-supplementation of B_{12} to avoid adverse effects.[3]

Nitrous oxide toxicity is divided into acute and chronic phases. In the acute phase, users experience sensations of euphoria, hallucinations, and analgesia. The onset of effects occurs within seconds, typically peak at one minute, and completely resolve within several minutes.[2] The quick onset and resolution of effects makes N_2O an attractive drug for adolescents and young adults. Most users return to their baseline neurologic and functional status within minutes after single use. Some users with underlying cardiac or lung disease may have complications, including arrhythmias, hypoxia, pneumothorax, or pneumomediastinum.[4] Rarely, death attributed to arrhythmias, seizure, and asphyxiation has been reported, with postmortem analysis demonstrating pulmonary edema and visceral congestion.[4]

Toxicity related to chronic N_2O use is believed to be caused by irreversible oxidation of the cobalt ion in vitamin B_{12} leading to impaired conversion of homocysteine to methionine and S-adenosylmethionine in deoxyribonucleic acid (DNA) and myelin synthesis. Vitamin B_{12} exists in the body in two active forms, methylcobalamin and adenosylcobalamin, which act as cofactors for methionine synthetase and methylmalonyl coenzyme A mutase, respectively. Nitrous oxide acts to convert vitamin B_{12} from its active to its inactive form via irreversible oxidation, thus blocking its availability as a cofactor for DNA and myelin synthesis. This leads to demyelination of the central and peripheral nervous systems as well as megaloblastic anemia.[2]

This pathology may present clinically as sensory neuropathy, myeloneuropathy, and subacute combined degeneration.[5,6] While patients with vitamin B_{12} deficiency may be at greater risk of developing neurologic deficits, there are case reports of patients with normal to high serum vitamin B_{12} levels sustaining complications, suggesting the importance of vitamin B_{12} inactivation rather than deficiency that leads to pathogenesis. Some studies have suggested measurement of levels of methylmalonic acid (MMA) in exposures, rather than vitamin B_{12} levels. A rising MMA level signals a functional deficiency of vitamin B_{12}, as it is not being normally utilized.[8,9] The patient in this case reported experienced persistent sensory neuropathies and hallucinations despite an elevated vitamin B_{12} level due to prophylactic self-supplementation. This demonstrates a situation in which a normal-to-elevated vitamin B_{12} level may provide false reassurance to clinicians regarding neurologic outcomes. Further research is required to determine whether other endpoints such as folate, homocysteine, or MMA levels may provide more diagnostic reassurance or prognostication of neurologic outcomes.

The patient in this case was lost to follow-up, and it is not known whether he adhered to treatment.

CONCLUSION

Recreational use of nitric oxide is increasingly prevalent, especially among adolescents and young adults, and it is important to recognize due to the potential for permanent neurologic deficits. Cessation is the mainstay of treatment, but it is important to provide vitamin B_{12} supplementation, even in cases when serum vitamin B_{12} levels are normal or even

elevated. These patients require close follow-up with neurology for magnetic resonance imaging and serial evaluations of neurologic function to maximize outcomes. Even when adherent to treatment, patients may never have full neurologic recovery.

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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Perinatal Stroke Presenting as Arm Swelling: A Case Report

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Introduction: Perinatal stroke is a rare but clinically significant condition that can present in a variety of ways and can result in diagnostic challenges in a particularly vulnerable population. We present the case of a term neonate who presented with left arm swelling, ultimately diagnosed with perinatal stroke.

Case Report: A term male neonate presented to the emergency department with left arm swelling noted the day prior, with abnormal tone of the left arm since birth. Physical examination revealed mild erythema and edema localized to the left upper extremity, with the arm held in flexion. Neurological examination was otherwise unremarkable. Further evaluation, including imaging studies, demonstrated thrombi in the left axillary and subclavian arteries, as well as an infarct involving the right middle cerebral artery and anterior cerebral artery with diffusion restriction, consistent with perinatal stroke.

Conclusion: Through this case report, we aimed to increase awareness of perinatal stroke among healthcare professionals and highlight the importance of prompt recognition and appropriate management in optimizing outcomes for affected infants. [Clin Pract Cases Emerg Med. 2025;19(2):138-140.]

Keywords: *perinatal stroke; pediatric; ultrasound; magnetic resonance imaging; case report.*

INTRODUCTION

Perinatal stroke, which occurs between 20 weeks gestation and 28 days of life, is a neurological emergency associated with significant morbidity and mortality. Although the exact incidence remains uncertain, perinatal stroke is estimated to occur in 1 in 1600-2300 live births.[1] Approximately half of perinatal strokes present acutely in the first days of life.[1,2] Perinatal stroke poses substantial challenges in diagnosis and management due to its diverse clinical presentations and limited evidence-based guidelines. Despite advances in medical imaging and diagnostic techniques, the diagnosis of perinatal stroke can be difficult, often leading to delays in recognition and management. In this case report, we discuss the presentation of a patient with perinatal stroke, highlighting the complexities in diagnosis and management.

CASE REPORT

A six-day-old term male presented to our emergency department (ED) with left arm swelling. He was born via spontaneous vaginal delivery to a 17-year-old, gravida one para one, at 38 weeks two days gestational age, weighing 2.466 kg. It was noted that he had acrocyanosis of the left arm at birth, resolved after several minutes on the warmer. Apgar scores were 8 and 9 at one and five minutes of life, respectively, out of a maximum score of 10. Nursing staff additionally noted that his arm had no spontaneous movement immediately after birth with decreased tone, and he was subsequently diagnosed with a presumed nerve palsy. Mother reported he had been "stuck in the birth canal" during delivery. Chest radiograph obtained while in the nursery was negative for fracture. Prior to discharge from the nursery at day two of life, he was noted to have improvement in his movement of

the left arm.

He was seen by his primary care physician (PCP) at four days of age, where he was noted to have improved movement of the left arm. At five days of age, his mother noted that his left arm appeared swollen. He was seen at an outside ED and diagnosed with ulnar nerve palsy. Following a routine visit to his PCP for bilirubin check on the day of presentation, the family was encouraged to present to our ED for further evaluation of arm swelling.



Image 1. Patient's left arm was held in a flexed position with an area of forearm erythema (black arrow) and distal extremity edema (white arrow).

On presentation to our ED, examination was remarkable for the left arm held in flexion at the elbow with edema to the forearm and hand (Image 1), but no appreciable tenderness to palpation. Capillary refill was brisk, and radial pulses were 2+ and symmetric. He was noted to have erythema to the left forearm that had been present since birth. Radiographs of the right humerus and forearm were unremarkable. Ultrasound of the left upper extremity was obtained due to concern for thrombosis and revealed a long non-occlusive thrombus within the left axillary artery measuring approximately one centimeter in length, as well as a few small, non-occlusive adherent thrombi anteriorly and posteriorly in the midportion of the subclavian artery, with a distal area of turbulent arterial flow favored to be a thrombus.

Hematology was consulted and they made imaging recommendations. A magnetic resonance imaging (MRI) of the brain demonstrated a large acute/subacute infarct involving the right middle cerebral artery (MCA) and anterior cerebral artery with significant diffusion reduction. A magnetic resonance angiogram of the brain demonstrated severe focal occlusion at the M1/M2 junction of the MCA with normal flow seen in the distal MCA branches. Magnetic resonance venography of the brain was normal (Image 2). Laboratory studies were remarkable for significant elevation in D-dimer of 1,248 nanograms per milliliter (ng/mL) (reference range: < 500 ng/mL), low fibrinogen level of 148 milligrams per deciliter (mg/dL) (200 to 393 mg/dL), and hypoglycemia of 49 mg/dL (70-105 mg/dL). Hypoglycemia was corrected with dextrose-containing fluids. The patient was then admitted to the neonatal intensive care unit (NICU).

Neonatology, neurology, and hematology came to a

CPC-EM Capsule

What do we already know about this clinical entity?
Neonatal stroke is a rare but significant neurological emergency that necessitates timely diagnosis and management.

What makes this presentation of disease reportable?
This presentation is unusual as the neonate presented with extremity swelling and was found to have a clot, which led to workup that identified a neonatal stroke.

What is the major learning point?
A high index of suspicion, comprehensive diagnostic evaluation, and multidisciplinary management is needed to optimize outcomes.

How might this improve emergency medicine practice?
Increased recognition of neonatal stroke among emergency physicians is essential for early intervention to improve the long-term prognosis of affected infants.

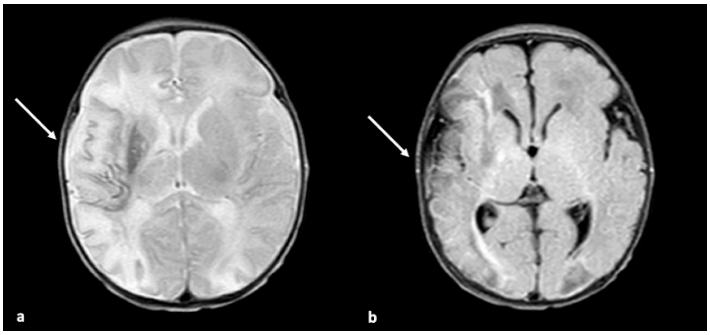


Image 2. Magnetic resonance imaging T2 (a) and fluid-attenuated inversion recovery (b) demonstrating hyperintensity and diffusion restriction involving the right middle cerebral artery and anterior cerebral artery distribution consistent with infarct (white arrows).

consensus on day one of hospitalization that heparin therapy should not be initiated as it was felt that any potential benefit of anticoagulation did not outweigh the risk of hemorrhagic transformation, with further increased risk due to the large infarct. On hospital day three, repeat ultrasound of the left upper extremity remained unchanged from the prior study. Head ultrasound showed increased echogenicity in the right ACA and MCA consistent with known infarcts without hemorrhage. Echocardiogram demonstrated a structurally normal heart, a patent foramen ovale with a small left to right shunt, and no intracardiac thrombus.

Interventional radiology was consulted on hospital day five and did not recommend angiography or arterial intervention as the arterial thrombus was small and non-occlusive on ultrasound, and he maintained good perfusion to the extremity on examination. Additional laboratory studies were obtained, including protein S activity, which was elevated at 70% and antithrombin activity, which was low at 48%, although noted to be expected in neonates. Protein C activity and protein S antigen were unable to be obtained and testing was deferred to hematology on an outpatient basis at a later date.

While in the NICU, the patient underwent physical therapy and had improvement in movement of the left arm with the exception of the hand, which he was unable to extend fully although finger movement was noted. Orthopedic surgery was also consulted and recommended outpatient follow-up in the brachial plexus clinic. He was discharged from the NICU on hospital day eight with outpatient follow-up scheduled with hematology, orthopedics, and neurology.

DISCUSSION

The diagnosis of perinatal stroke poses several challenges due to its non-specific clinical presentation and the limitations of neuroimaging modalities in neonates. Common presenting symptoms in neonates can include seizures, recurrent apnea or desaturations, alteration in tone, altered level of consciousness, and focal neurological deficits.[2] Cranial ultrasound is often the initial imaging modality used in perinatal stroke, but its sensitivity for detecting ischemic lesions is limited, especially in the early stages.[2,3] Magnetic resonance imaging with diffusion-weighted imaging is considered the gold standard for diagnosing perinatal stroke, offering superior sensitivity and specificity compared to other imaging modalities including computed tomography (CT). However, if MRI is not readily available or impractical, CT or ultrasound should be used.[3]

The management of perinatal stroke focuses on supportive care, seizure control, and prevention of secondary complications. Anticoagulation therapy, which is commonly used in adult stroke management, is controversial in neonates due to the risk of intracranial hemorrhage and is not routinely indicated.[2] Instead, management strategies often include hydration, temperature regulation, and physical and occupational therapy to optimize neurodevelopmental outcomes.[2,3]

Long-term outcomes following perinatal stroke vary widely, ranging from complete recovery to severe neurodevelopmental disabilities.[2,4] Early identification of neurodevelopmental delays and prompt initiation of early intervention services are crucial in optimizing outcomes for affected infants.[1,4] Multidisciplinary collaboration and follow-up involving neurology, hematology, and rehabilitation specialists is essential.

CONCLUSION

Perinatal stroke is a neurological emergency that necessitates timely diagnosis and management. Further study is needed to elucidate the underlying mechanisms, refine diagnostic approaches, and develop targeted therapeutic interventions. Increased awareness among clinicians is essential to facilitate early intervention and improve the long-term prognosis of affected infants. This case underscores the need for a high index of suspicion, comprehensive diagnostic evaluation, and multidisciplinary management to optimize outcomes in affected infants.

Documented patient informed consent and Institutional Review Board approval has been obtained and filed for publication of this case report.

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Case Report of Post-Appendectomy Fungal Osteomyelitis: A Rare Complication in a Healthy Patient

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Introduction: Osteomyelitis is a bone infection that presents with swelling, erythema, pain, and possible systemic symptoms. Immunocompromised patients are at higher risk of developing osteomyelitis. Fungal pathogens are a rare etiology for these infections with very few case reports published compared to infections due to bacterial pathogens. Work up should include imaging studies to investigate infections when there is clinical suspicion for osteomyelitis. Bone biopsy is performed to identify the causative agent with bacterial infections being the most common. Osteomyelitis can be treated both surgically with debridement or amputation and medically with extended courses of antimicrobials or antifungals. Our case describes fungal foot osteomyelitis after an uncomplicated appendectomy.

Case Report: A 19-year-old previously healthy female underwent laparoscopic appendectomy for nonperforated, non-gangrenous appendicitis. Fourteen days later, she developed gradually worsening right foot pain, swelling, and erythema. After multiple failed treatments for the management of osteomyelitis, bone biopsies and courses of antibiotics, patient was ultimately diagnosed with a rare osteomyelitis secondary to *Coccidioides* species, which was managed and improved with antifungals.

Conclusion: Bacterial osteomyelitis has been described in two English case reports as a postoperative complication of appendectomy, particularly when the appendicitis is perforated, gangrenous, or purulent. Fungal osteomyelitis is an even rarer cause of postoperative bone infection in immunocompetent patients. The goal for treatment is surgical intervention or pharmacologic management. Emergency department physicians should maintain a high suspicion for fungal osteomyelitis when otherwise healthy patients present multiple times for failing outpatient antibiotic regimens. [Clin Pract Cases Emerg Med. 2025;19(2):141-145.]

Keywords: *Fungal osteomyelitis; appendectomy; Coccidioides; hematogenous osteomyelitis; case report.*

INTRODUCTION

Osteomyelitis is an acute or chronic infection of bone tissue which occurs secondary to traumatic injury, open fractures, surgery or pyogenic organisms that spread through the bloodstream.[1] Patients with a history of smoking cigarettes or diabetes, complicated and open fractures, surgical implants, large wound sites, and other causes for poor wound healing have increased risk for osteomyelitis. Osteomyelitis can present with inflammation, swelling, erythema at the site of infection and associated constitutional

symptoms such as fever, chills, and malaise. Staphylococcus aureus is the most common bacterial infectious agent in osteomyelitis in adults and children; however, osteomyelitis can be caused by a variety of other bacterial or even fungal pathogens. Radiographs and magnetic resonance imaging (MRI) are standard methods of identification to detect soft tissue swelling, osteolysis, and other infection-related bone damage. Diagnostic criteria include positive cultures from bone or blood in conjunction with other signs and symptoms.[2] Bone biopsy is generally performed to identify the infectious pathogen. Bone biopsy cultures identify microbiological cause in 94% of cases.[3] Blood cultures are commonly negative except in cases in which osteomyelitis is caused by hematological spread.[4] Treatment involves a multi-disciplinary approach with medical and surgical specialists for prolonged antibiotic therapy, and possible surgical debridement or amputation.[5]

This case of foot fungal osteomyelitis is unique presenting in a healthy patient after uncomplicated appendectomy. There is a paucity of research on the topic of osteomyelitis after appendectomy with only two case reports found in the English language literature review. The reported cases had bacterial pathogens as the causative agent and were in patients with ruptured, perforated, or gangrenous appendicitis.[6,7]

CPC-EM Capsule

What do we already know about this clinical entity?
Osteomyelitis is an infection of bone that can occur after surgery. It is rarely caused by fungal species.

What makes this presentation of disease reportable?
This case report highlights an unusual presentation of fungal osteomyelitis after uncomplicated appendectomy.

What is the major learning point?
If a patient continues to worsen in their treatment of osteomyelitis despite initial antibiotics, an atypical infection should be considered.

How might this improve emergency medicine practice?
This article encourages clinicians to expand their differential of bony pain after surgery and consider atypical infections for patients who failed conventional osteomyelitis treatment.

CASE REPORT

A 19-year-old female with no significant past medical history presented to the Emergency department (ED) for progressively worsening right foot pain, swelling, and redness with associated nausea. The patient initially presented to the ED one month prior for abdominal pain, received pre-operative intravenous (IV) piperacillin/tazobactam, and underwent a laparoscopic appendectomy with findings of acute nonperforated appendicitis. She was discharged the same day with pain medications.

The patient followed up with the acute care surgery team in clinic on postoperative day 14. At the visit, she was determined to be doing well post-operatively. She expressed concern about swelling and soreness of her right foot without any history of trauma. Her physical exam noted that she was tender to palpation over the lateral right foot without any redness, wounds, bruising or any physical exam findings of trauma. The surgeon noted patient was able to bear weight and advised conservative management for possible foot sprain.

One month after the surgery, she presented to the ED because of persistent pain and swelling in the right foot. Initial vital signs were unremarkable. On physical exam, patient's right lateral foot noted swelling with overlying erythema, no drainage, tender to palpitation and the distal foot was neurovascularly intact (Image 1).



Image 1. Patient with right foot fungal osteomyelitis after an appendectomy noted to have swelling, erythema, and tenderness to palpation on exam.

She reported the symptoms started after her appendectomy. Significant labs include white blood cells (WBC), 8.18 x 109 per liter (L) (reference range: 4.8-11.80 x 109/L); erythrocyte sedimentation rate (ESR), 39 millimeters per hour (mm/hr) (0-20 mm/hr); C-reactive protein (CRP), 0.7 milligrams per deciliter (mg/dL) (0.0-0.8 mg/dL). Foot radiographs were concerning for osteomyelitis. Intravenous vancomycin was started in the ED, and she was admitted to the hospital. She had further radiologic studies. Computed tomography scan revealed findings suggestive of osteomyelitis with adjacent phlegmon and inflammatory changes and associated fifth metatarsal neck fracture. Magnetic resonance imaging reported findings consistent with osteomyelitis of the fifth metatarsal with surrounding phlegmon. Bone biopsy was negative to any growth.

Infectious disease was consulted and recommended IV cefazolin, thus a peripherally inserted central catheter (PICC) line was placed. She was discharged from the hospital with the diagnoses of osteomyelitis of the right fifth metatarsal and provided with a four-week course of cefazolin to be administered via her PICC line.

Eight days later, patient returned to the ED for worsening right foot pain, swelling, redness, and a new blister forming on lateral side of foot. Vital signs remained unremarkable. Her physical exam was significant for swelling, tenderness, and redness with a fluctuant ulcer draining serous fluid (Image 2). Despite cefazolin, the patient's symptoms had worsened.

Laboratory analysis revealed WBC, 7.05 x 109/L; ESR, 50 mm/hr; and CRP, 1.2 mg/dL. Acute care surgery was consulted for possible surgical debridement. Their team recommended internal medicine admission for IV antibiotics due to outpatient failure of cefazolin as well as infectious disease for the management of osteomyelitis. The patient was started on IV vancomycin and cefepime. Repeat MRI during this admission revealed worsening extensive osteomyelitis with regional phlegmon, developing abscess, myositis of the lateral forefoot and associated regional cellulitis. A second bone biopsy was performed and again had negative growth. Bone biopsy pathology reported marrow fibrosis with reactive changes but no acute inflammation or necrosis. Acute care surgery offered incision and drainage of the abscess and fifth digit amputation for source control which patient declined. As patient was deemed to have failed outpatient IV cefazolin therapy, infectious disease recommended four weeks of IV vancomycin and cefepime to be administered for four weeks via PICC line. Patient was discharged home with the regimen of antibiotics recommended by infectious disease.

Three months after her appendectomy and one month after the second hospitalization, the patient again presented to the ED. During the antibiotic course with vancomycin and cefepime, she did experience temporary improvement but not resolution of her symptoms. Briefly after completion of the course, her symptoms returned. She presented with worsening pain and swelling of the right foot. On physical exam, she was noted to have erythema of her right lateral foot with tenderness to palpation and a three centimeter circular wound with purulent drainage (Image 3).

Laboratory analysis revealed WBC, 7.36 x 109/L; ESR, 53 mm/hr; and CRP, 1.9 mg/dL. Radiograph of the foot was



Image 2. Patient with right foot fungal osteomyelitis after an appendectomy following a four-week course of cefazolin. She was noted to have worsening right foot pain, swelling, redness, with a new blister forming on exam.



Image 3. Patient with fungal osteomyelitis after an appendectomy following one month of cefepime and vancomycin. She was noted to have erythema of her right lateral foot with tenderness to palpation and a three-centimeter circular wound with purulent drainage on exam.

suggestive of acute osteomyelitis with persistent phlegmon. She was started on IV cefazolin and admitted to the hospital under the internal medicine service. During that admission, her wound culture grew filamentous fungi and fungal specific culture to *Coccidioides immitis* and C posadasii. Infectious disease recommended voriconazole by mouth and discharge.

Upon follow up in the infectious disease clinic, the patient's antifungal therapy was changed to fluconazole instead of voriconazole because of the side effects the patient experienced to voriconazole. As of writing this paper, the patient continues to take fluconazole for her improving foot infection.

After the patient's appendectomy and multiple admissions to the hospital, the patient continues to follow up in clinic. The infectious disease team documented they theorize the patient may have acquired an asymptomatic Coccidioides pulmonary infection as she lives in an endemic area with dissemination to the foot because of an immunosuppressive event from her appendectomy. It is possible the patient's osteomyelitis was autoimmune (chronic recurrent multifocal osteomyelitis) versus a secondary infection developing after multiple rounds of antibiotics, or related to skin breakdown, however a high suspicion for atypical fungal infection is critical in any patient diagnosed with osteomyelitis who does not improve with antibiotics.

DISCUSSION

Bacterial osteomyelitis after appendectomy is a rare complication that has been reported in only two case reports.[6,7] Fungal osteomyelitis is a rarer form of osteomyelitis with only case reports, case series, and narrative reviews in the published literature. From a recent (2023) systematic review of reported cases, the most common fungal pathogens in osteomyelitis are *Aspergillus* (26.5%), *Candida* (20.7%), and *Mucor* (16.8%) with *Coccidioides* osteomyelitis making up 5.6% of total cases. Vertebral fungal osteomyelitis is the most common (318 cases, 29.7%) while fungal foot osteomyelitis is rare (138 cases, 4.5%) and only 24 patients grew *Coccidioides* as the etiologic agent.[8,9] Our patient is the only known reported case of fungal osteomyelitis after uncomplicated appendectomy.

The first case of osteomyelitis after an appendectomy reported in the English literature was published in 2010. The patient was a previously healthy male who developed appendicitis. He underwent appendectomy and was found to have a perforated retrocecal gangrenous appendix with copious free pus. He had an uncomplicated hospital course involving surgical site infection that required additional antibiotics. The patient re-presented six weeks after discharge with symptoms of right lower quadrant pain, and inability to bear weight. He was diagnosed with iliac crest osteomyelitis caused by *Pseudomonas aeruginosa* and *Bacteroides*.[6]

In 2013, the only other reported case of osteomyelitis following appendectomy involved a patient with a perforated gangrenous appendix with free pus. On postoperative day 23, he presented with lower back, thigh, and buttock pain and difficulty weight bearing. He was found to have sacroiliitis and iliac bone osteomyelitis which improved with antibiotic administration.[7]

Fungal osteomyelitis is considered a rare disease and is often overlooked when creating the differential diagnosis. Symptoms are often subacute and mimic those of other etiologies, which leads to substantial delays in treatment as was the case with our patient.[8] Clinicians should consider fungal infection when osteomyelitis is of clinical concern. A recent (2023) systematic review of fungal osteomyelitis revealed the risk factors to be local surgery or local lesion, diabetes mellitus and disseminated fungal infection (which is theorized in our patient). However, fungal osteomyelitis can happen in an otherwise healthy patient as well.[9]

CONCLUSION

Fungal osteomyelitis in the foot is rare and may present in an otherwise healthy patient but more commonly occurs in the patient with local surgery, local lesion, diabetes mellitus or disseminated fungal infection. It is critical to keep a broad differential diagnosis on postoperative patients presenting to the ED with musculoskeletal complaints. Clinicians should maintain high clinical suspicion for fungal infections when considering osteomyelitis especially for those who have risk factors and fail to improve after appropriate antibiotic courses.

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

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Lipschütz Ulcers In 12-year-old Premenarchal Female Days After A Gastrointestinal Illness: A Case Report

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Introduction: Lipschütz ulcers are a rare immune-mediated reaction that commonly occurs in premenarchal females, usually associated with a recent viral illness.[1] The treatment for Lipschütz ulcers consists of pain relief, topical steroids, and, in severe cases, a course of systemic steroids.[1] A thorough history and exam, as well as an appropriate workup to rule out other causes of vaginal ulceration, should be completed.[2]

Case Report: A premenarchal, 12-year-old female presented to the emergency department (ED) with her mother due to significant vulvar pain. Two days prior, the patient had a gastrointestinal illness associated with vomiting, diarrhea, and fever. On exam, she had significant swelling of the labia minor, discoloration with a necrotic appearance of the introitus, and brown vaginal discharge. The patient denied sexual intercourse, concern for retained vaginal foreign body, or vaginal trauma. Gynecology suggested the diagnosis of a rare post-viral immune-mediated reaction causing acute genital ulcerations, also known as Lipschütz ulcers. The patient's treatment regimen included topical and systemic steroids, enteral opioid pain medication, and topical lidocaine. Her symptoms had resolved at her two-month follow-up visit.

Conclusion: In summary, this case report discusses a previously healthy 12-year-old premenarchal female who presented to the ED due to vulvar swelling, pain, and vaginal discharge in the setting of a recent viral gastrointestinal illness. The patient was seen in the ED by gynecology and diagnosed with Lipschütz ulcers. Lipschütz ulcers are an uncommon condition causing acute genital ulcers. [Clin Pract Cases Emerg Med. 2025;19(2):146-148.]

Keywords: *Acute genital ulcers; gynecology; emergency medicine; Lipschütz ulcer; case report.*

INTRODUCTION

Lipschütz ulcers are painful genital ulcers with unknown etiology. These ulcers present on the vulva and are often associated with recent bacterial or viral infections in adolescent females.[3] Due to the severity and acuity of the symptoms, the patients who experience these acute genital ulcers may present to the emergency department (ED), as did the patient described in this case report. The limited number of publications available within emergency medicine literature has led to a lack of awareness of this disease by emergency physicians and therefore may lead to underdiagnosis in the emergency department setting. There is a need for emergency physicians to be aware of this disease to limit unnecessary testing and inaccurate treatments.[4]

CASE REPORT

A 12-year-old premenarchal female presented to the ED with her mother due to vulvar pain and swelling. She was previously healthy, fully immunized for age, actively involved in volleyball, and had an average body mass index for age.

Two days prior to presenting to the ED, she experienced vomiting, diarrhea, and fever associated with gastrointestinal illness, suspected to be viral in nature, which had since resolved. She denied respiratory symptoms. The patient had trialed home treatment with acetaminophen, ibuprofen, and warm baths to help the vulvar swelling and pain, but these interventions did not improve the swelling, pain, or discomfort. This was the patient's first presentation for evaluation of this concern, and she had no prior evaluations by gynecology. She was premenarchal and denied sexual intercourse, a history of sexually transmitted infection, vaginal trauma, concern for retained foreign objects in the vagina, sexual trauma, or sexual assault.

On initial physician exam, the patient was afebrile, hemodynamically stable, well appearing, without increased work of breathing, however she was sitting very still in the hospital bed. The patient had mildly diffuse abdominal tenderness during the exam. On vaginal exam, there was swelling of the labia majora (left greater than right) with purple and necrotic ecchymosis, brown discoloration of the labia minor, and copious brown discharge flowing from the introitus. The patient had an initial workup in triage with negative swabs for SARS-CoV-2, influenza A, and influenza B. Broad workup was initiated and included a white blood cell count of 12 x 10^9 per liter (L) (reference range: 3.8 - 10.4 x 10^9/L) with a neutrophil count of 9.5 x 10^9/L (1.5 - 6.5 x 10^9/L), potassium of 3.5 millimoles (mmol)/L (3.6 - 5.2 mmol/L), sodium of 133 mmol/L (135 – 145 mmol/L), chloride of 99 mmol/L (102 – 112 mmol/L), creatinine of 0.79 milligrams per deciliter (mg/dL) (0.35 - 0.86 mg/dL). Urine microscopy showed red blood cells and bacteria, with a negative bacterial urine culture. Vaginal swabs were also collected in the ED and were negative for Chlamydia, gonorrhea, trichomonas, and herpes simplex virus (HSV). There was no growth on bacterial and fungal cultures.

Gynecology was consulted and upon examination they made the diagnosis of acute genital ulceration (Lipschütz ulcer). She was started on pain management with scheduled acetaminophen and ibuprofen. The patient was instructed to apply ice packs for twenty minutes once every hour to the area, topical clobetasol ointment two times daily, and topical lidocaine gel as needed. She was prescribed oral oxycodone. She was discharged from the ED with a follow-up visit with gynecology the following week.

At the follow up visit, gynecology started the patient on an oral prednisone taper, in addition to the topical steroids, given that the ulcers were still present and significant, although improved. She stopped using the clobetasol ointment after this visit due to pain with the application. She had another follow up with gynecology two weeks later and at that visit it was documented that her ulcers and pain had greatly improved. There were no concerns for a superimposed infection from appearance of the ulcers during the exam. The patient was instructed to follow up with her family medicine physician and discussion occurred regarding the increased likelihood of recurrence during times of stress, especially after viral infection or illness. It was explained that there would be no impact on her future fertility and no additional gynecology follow-up was warranted. The patient was prescribed topical lidocaine, topical clobetasol, and prednisone taper, if she experienced a flare in the future. The ulcers fully resolved at a follow up appointment with her primary care physician the following week.

CPC-EM Capsule

What do we already know about this clinical entity?
Lipschütz ulcers are painful, necrotic ulcers due to an immune-mediated reaction in premenarchal females, usually associated with a recent viral illness.

What makes this presentation of disease reportable?
The presentation of Lipschütz ulcers in a pediatric emergency department in the setting of recent gastrointestinal illness.

What is the major learning point?
Lipschütz ulcers are painful, necrotic ulcers that can occur after a recent viral illness. They are usually self limited, however they may require treatment with topical or systemic steroids.

How might this improve emergency medicine practice?
Emergency medicine physicians should be knowledgeable about this uncommon diagnosis to prevent unnecessary workups and incorrect treatments.

DISCUSSION

This case report highlights the importance of emergency physicians considering the diagnosis of Lipschütz ulcers in adolescent females who present with acute genital ulcers after a recent viral illness to prevent invasive unnecessary workups and ineffective treatments. Lipschütz ulcers are described in the literature after a viral or bacterial infection, including influenza, streptococcal pharyngitis, with limited case reports describing the presentation after the COVID-19 vaccination and infection.[5-7] Body aches, fatigue, and other nonspecific symptoms can occur during the presentation of the ulcers and

should be a diagnosis of exclusion.[2] Therefore, it is essential to take a thorough history and do a complete physical exam, along with any necessary workup to rule out other causes of acute genital ulcers, such as HSV, syphilis, chancroid, lymphogranuloma venereum, trauma, Crohn disease, psoriasis, trauma, fixed drug eruption, and Behçet syndrome.[1,3]

The ulcers are often described as bilateral "kissing lesions" that are painful, necrotic, with grayish exudate and signs of inflammation.[2,7] Lipschütz ulcers have been reported in children but are more commonly reported in adolescents and young adults. The ulcers are often bigger than HSV lesions and painful, as compared to the non-painful chancre of syphilis. There is discussion in the literature regarding diagnostic criteria, however further research is needed regarding the utility of using this criteria in the ED.[3] There is no role in biopsy for diagnosis, as case reports describe finding nonspecific inflammatory cell infiltration on biopsy.[6,8] Resolution of ulcers occurs over the course of two to six weeks, and they may recur, especially during times of stress and viral illnesses.[7,8]

This is an uncommon condition with limited literature, specifically from an emergency physician perspective, which may lead to the underdiagnosis of this condition when patients present to the ED. Patients and parents should be counseled that there is no evidence to suggest a correlation between Lipschütz ulcers and sexually transmitted infections.[8] Treatment is supportive with pain management, and the condition is self-limited. However, severe cases could be treated with topical corticosteroids with additional consideration for the use of systemic steroids.[1,2,6] More literature is needed regarding the use of steroids for symptom management in this self-limited condition.

CONCLUSION

In summary, this case report discusses the initial presentation of Lipschütz ulcers in a previously healthy 12-year-old premenarchal female who presented to the ED with her mother due to vulvar pain and swelling after a recent gastrointestinal illness. On vaginal exam, there was swelling of the labia majora (left greater than right) with purple appearing ecchymosis, brown discoloration of the labia minor, and brown discharger flowing from the introitus. The diagnosis of Lipschütz ulcer is uncommon; however, it should be considered in premenarchal females who deny sexual activity and have vulvar and vaginal ulceration on exam, especially in the setting of a recent viral illness. Misdiagnosis delays appropriate intervention and limit unnecessary workup. It is essential that emergency physicians make the correct diagnosis and arrange appropriate follow-up appointments with gynecology or primary care.

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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Fusobacterium necrophorum Brain Abscess Following Invasive Sinusitis in an Immunocompetent Adult: A Case Report

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Introduction: A brain abscess is a localized collection of purulent infection within the brain parenchyma. It most often occurs due to contiguous spread from sinus, otogenic, and odontogenic infections; however, it can also develop from direct intracranial contact via trauma or surgery. *Fusobacterium necrophorum*, an obligate anaerobic, gram-negative bacillus, is part of the normal flora of the oral cavity. Given its inherent location, F *necrophorum* has been shown to contribute to complications stemming from infection of the tonsils, pharynx, and teeth. Invasive infections of F necrophorum are seldomly seen in immunocompetent patients.

Case Report: We report a case of a previously healthy 20-year-old man who presented to our emergency department with headache, facial pain, and neck stiffness. He was ultimately found to have an *F necrophorum* intracranial abscess and underwent right frontal craniotomy with evacuation of epidural abscess and partial sinus obliteration. He was placed on broad-spectrum antibiotics, including vancomycin, cefepime, and metronidazole for six weeks. His treatment course was complicated by recurrence of intraparenchymal abscess requiring repeat craniotomy with abscess evacuation and advancement of antibiotic regimen to meropenem. To our knowledge, there are no reported cases in the literature of monomicrobial *F necrophorum* brain abscesses arising secondary to invasive sinusitis in immunocompetent adults.

Conclusion: This report highlights the clinical presentation, diagnostic strategies, management challenges, clinical outcomes, and complications of invasive sinusitis leading to brain abscess formation in an otherwise healthy adult male. [Clin Pract Cases Emerg Med. 2025;19(2):149-153.]

INTRODUCTION

A brain abscess is a localized collection of purulent infection within the brain parenchyma. It most often occurs from contiguous spread from sinus, otogenic, and odontogenic infections; however, it can also develop due to direct intracranial contact via trauma or surgery.[1-3] Intracranial abscesses typically contain aerobic and anaerobic bacteria, with anaerobes as the more common isolate—specifically, the gram-negative Fusobacterium species.[4] In instances where Fusobacterium brain abscesses or sinusitis are present, it is typical to observe the growth of *Fusobacterium nucleatum* species in cultures.[5-8] In contrast, the identification of *Fusobacterium necrophorum* in isolates is relatively rare.[6,7] Apart from sinusitis and brain abscesses, *F necrophorum* is a well-documented cause of a variety of other infections, including Lemierres syndrome, pharyngitis, meningitis, and septicemia. This bacterium can be found as part of the normal microbial flora of the mouth and throat in healthy individuals and can therefore contribute to invasive complications stemming from infection of the tonsils, pharynx, and teeth.

However, invasive infections of *F necrophorum* are seldom seen in immunocompetent patients.[7,9]

We report a case of a previously healthy man who presented to our emergency department (ED) with headache and was found to have an *F necrophorum* intracranial abscess. To our knowledge, there are no reported cases in the literature of monomicrobial *F necrophorum* brain abscesses arising secondary to invasive sinusitis in immunocompetent adults. Previously associated with high incidence of mortality, the outcomes for those with brain abscesses have improved with the advent of computed tomography (CT) and magnetic resonance imaging (MRI) (0.4-0.9 cases per 100,000).[10,11] However, despite these advancements, untimely identification and treatment continue to result in poor outcomes.[12] Thus, our work aims to highlight the clinical presentation, diagnostic strategies, management challenges, clinical outcomes, and complications of this rare infection.

CPC-EM Capsule

What do we already know about this clinical entity?
Fusobacterium necrophorum *is often linked to sinusitis or Lemierre's syndrome in immunocompromised patients and rarely causes brain abscesses.*

What makes this presentation of disease reportable?
This case is the first reported monomicrobial F. necrophorum brain abscess secondary to invasive sinusitis in an immunocompetent adult.

What is the major learning point?
Early recognition and aggressive multidisciplinary management of atypical pathogens like F. necrophorum can prevent severe complications and improve outcomes.

How might this improve emergency medicine practice?
Enhances awareness of rare brain abscess etiologies and emphasizes timely imaging and treatment for atypical, persistent sinusitis symptoms.

CASE REPORT

A 20-year-old man with no significant past medical history presented to the emergency department (ED) for evaluation of fever, headache, neck pain and stiffness, and facial pain. In the preceding month, he was initially diagnosed with influenza, which improved with supportive treatment until two weeks into his influenza diagnosis, when he began experiencing increased facial pain and intermittent fevers. For these new symptoms, he was diagnosed with bacterial sinusitis and was given a seven-day course of amoxicillin/trimethoprim which he took to completion. Symptoms initially resolved during the course of antibiotics, but by the end his symptoms returned. He represented to his primary care physician and was started on doxycycline. After three days of doxycycline, he presented to the ED with worsening headaches exacerbated by movement, neck pain and stiffness with flexion, continued facial pain, and intermittent fevers. He reported minimal relief from over-the-counter analgesics and antipyretics. He was fully vaccinated and had no recent wilderness exposure, insect bites, or travel.

In the ED, vital signs included a temperature of 37.8 °Celsius, heart rate of 109 beats per minute, respirations at 16 breaths per minute, blood pressure of 128/66 millimeters of mercury, and oxygen saturation of 100% on room air. On examination, he appeared uncomfortable and was wearing sunglasses due to photophobia. Inspection of the head and face revealed normal appearance without significant lesions. Palpation of the face and sinus cavities were positive for tenderness bilaterally. There was no salivary gland swelling or tenderness. Otoscopic exam revealed normal external auditory canals and tympanic membranes. Nasal inspection displayed normal nasal mucosa without evidence of septum abnormality. Oral inspection revealed oropharynx with normal mucosa and oral cavity negative for asymmetry, lesions, masses, erythema, exudates. Although his neck was no rigid, he had some stiffness and pain with flexion. Kernig and Brudzinski signs were negative, and neurologic examination revealed no focal deficits. He was alert and oriented to person, place, and time.

While awaiting further tests, the patient received intravenous (IV) fluids, analgesics, and antiemetics and received empiric treatment for meningitis with IV vancomycin, ceftriaxone, and dexamethasone. Initial laboratory studies revealed leukocytosis with 17.3 x 109 white blood cells per liter (L) (reference range: 3.4-9.6 x 109 cells/L) with neutrophilia of 94.8% (50.0-75.0%), C-reactive protein of 20.6 milligrams (mg)/L (<5.0 mg/L), and lactate of 1.7 millimoles (mmol)/L (0.5-2.2 mmol/L). Head CT with and without contrast demonstrated complicated right frontal sinusitis with intracranial extension and formation of an epidural abscess with extensive surrounding vasogenic edema and leftward midline shift (Image 1). A neurosurgeon was emergently consulted, and antibiotic coverage was broadened to IV vancomycin, cefepime, and metronidazole.

The patient underwent emergent right frontal craniotomy for evacuation of epidural abscess and obliteration of the connection between the frontal sinus and intracranial compartment. Bacterial cultures and Gram stain of a sample from the intracranial abscess were obtained, and the patient

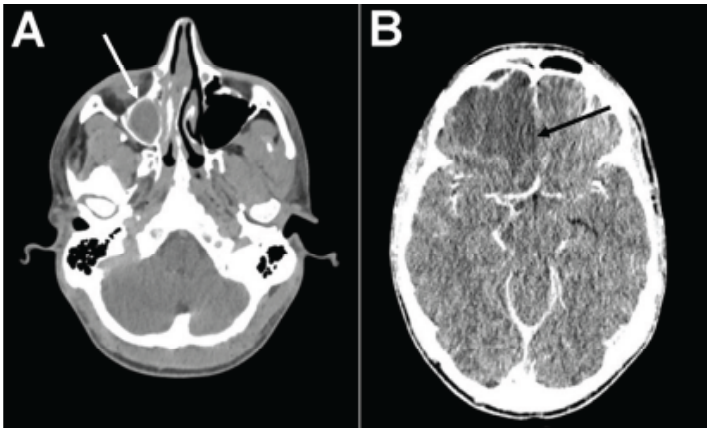


Image 1. Head computed tomography without (A) and with (B) intravenous contrast. Imaging demonstrated a complicated right frontal sinusitis (white arrow) with intracranial extension and formation of an abscess with extensive associated vasogenic edema and mass effect with two-millimeter leftward midline shift (black arrow).

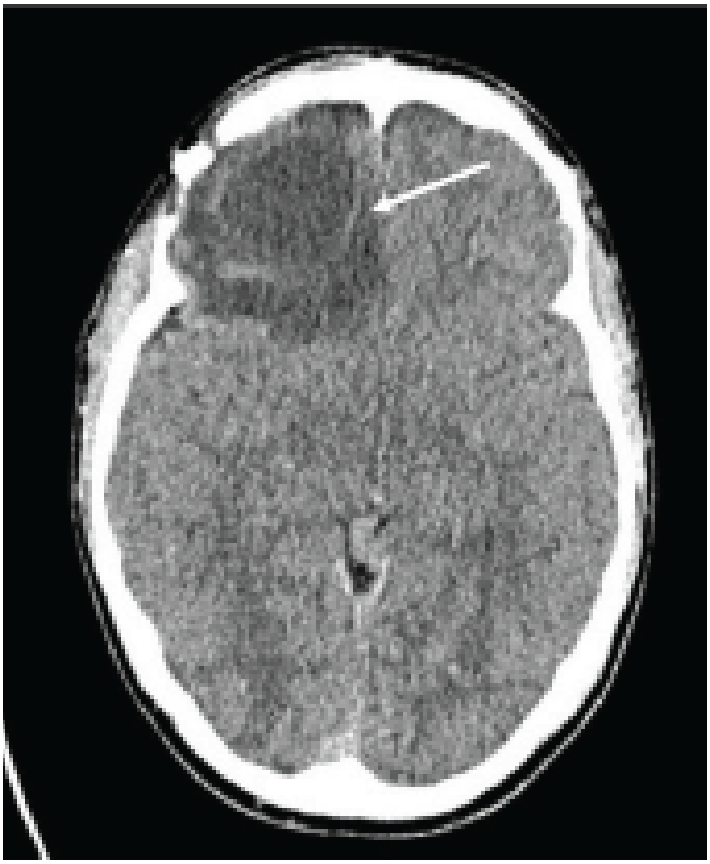


Image 2. On the patient's second emergency department visit for recurrence of symptoms, head computed tomography demonstrated interval development of a hypodense focus with hyperattenuating rim, concerning for a recurrent organizing intraparenchymal abscess with two millimeters leftward midline shift (arrow).

was admitted to the neurointensive care unit. On hospital day three, an otorhinolaryngologist performed a functional endoscopic sinus surgery with right maxillary antrostomy, right total ethmoidectomy, right sphenoidotomy, and right frontal sinusotomy with additional washout. During the procedure, the patient was noted to have severe inflammation of the mucosa of the ethmoid bulla and anterior ethmoidal cells, which appeared to be consistent with an ascending infection. Intraoperative cultures of purulent drainage from the maxillary and frontal sinuses were collected. At this time, the culture sample from the intracranial abscess showed growth of *F necrophorum*, so a peripherally inserted central catheter was placed in anticipation of long-term antibiotics.

On day of discharge, samples from the sinus showed no growth. The patient had marked improvement, and his leukocyte count was down to 8.7 x 10^9 cells/L. He was sent home on a corticosteroid taper, six weeks of oral metronidazole 500 mg three times daily, and six weeks of IV ceftriaxone 2 grams (g) twice daily with planned infectious disease outpatient clinic follow-up.

Approximately one month after discharge, the patient returned to the ED due to severe headache, retro-orbital pain, and intractable nausea and vomiting. Computed tomography without contrast and MRI with contrast of the head demonstrated intraparenchymal abscess and moderate lobulated opacification of the right maxillary sinus (Images 2 and 3). He was subsequently admitted to the neurointensive care unit and underwent repeat right frontal craniotomy for abscess evacuation. Home antibiotics of metronidazole and ceftriaxone were continued in their IV formulations, with the addition of vancomycin for broader coverage. Levetiracetam and dexamethasone were also added, and the patient was discharged five days later with close neurosurgical, otorhinolaryngologic, and infectious disease follow-up. At discharge, he was afebrile, hemodynamically stable, and free of retro-orbital pain, nausea, vomiting, and headache.

He returned to the ED ten days later with complaints of increased headache, fever, photophobia, intermittent bilateral hand numbness, and nausea. Given his history of brain abscess with recurrence, the patient was admitted to the neurosurgery service. Head CT with and without contrast and brain MRI with and without contrast demonstrated stable postsurgical changes without any abscess development. The infectious disease specialists advanced the antibiotic regimen to two g of IV meropenem every eight hours. The patient demonstrated clinical improvement and was discharged home three days later with close follow-up with specialty services and made a complete recovery in the outpatient setting. Brain MRI obtained two months after initial presentation revealed continued evolution of postsurgical changes within the right frontal region with no findings suggesting recurrent or progressive abscess.

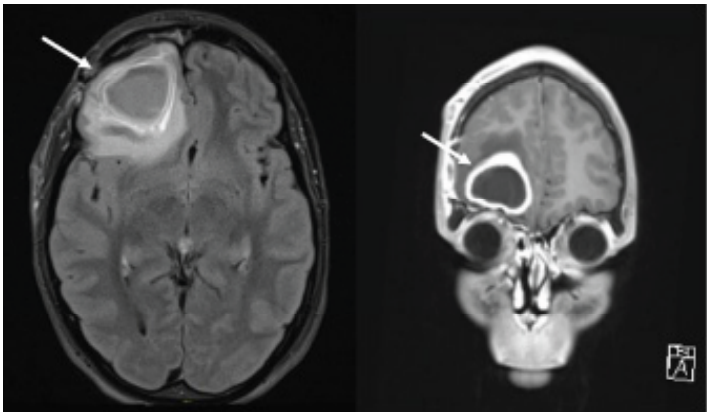


Image 3. On the patient's second emergency department visit for recurrence of symptoms, magnetic resonance imaging of the brain with contrast demonstrated recurrent large right inferior frontal lobe cerebral abscess with significant surrounding vasogenic edema.

DISCUSSION

Treatment of brain abscesses involves a multidisciplinary approach with various surgical and medical interventions depending on the infection source and microorganisms identified. With proper treatment, the overall mortality rate for brain abscesses has decreased to around 5% to 10%.[10] In this case, the positive culture for *F necrophorum* guided the selection of antibiotics. The patient's immunocompetent status, despite the severity of the infection, may have contributed to his resilience during treatment.

The conventional risk factors associated with the development of brain abscesses encompass previous head trauma, recent neurosurgical procedures, or persistent infections (such as otitis media, dental caries, and sinusitis) that may facilitate contiguous dissemination. Specific populations at heightened risk include immunocompromised patients (eg, HIV/AIDS), those suffering from chronic pulmonary diseases, and patients with diabetes mellitus or certain neoplastic disorders. Additionally, individuals traveling internationally may encounter infections like cysticercosis, which can lead to abscess formation.[1,4]

Regarding prognosis, early diagnosis and comprehensive treatment are associated with favorable outcomes, particularly in patients who present with preserved consciousness on admission.[5] However, the risk of recurrence of brain abscesses is high, with reports indicating a rate of approximately 10%, as seen in our case. Moreover, the progression from sinusitis to brain abscess is a rare and severe complication, occurring in approximately 0.4% of patients with sinusitis, particularly in those with untreated or inadequately managed bacterial sinusitis.[10] This statistic highlights the need for long-term follow-up and diligent monitoring, even for those without traditional risk factors.[11]

Emergency physicians should maintain high suspicion for brain abscesses in patients presenting with undifferentiated headache and sinus pressure, especially when accompanied by neurological signs such as neck stiffness, fever, or changes in mental cognition. Neuroimaging should be obtained in these cases to rule out brain abscess, especially when symptoms persist despite initial treatment for sinusitis or if there is severe systemic illness. Computed tomography is typically the first-line imaging modality due to ease and rapidity of results, but MRI is preferred for its increased sensitivity in diagnosing, monitoring, and characterizing the development of brain abscesses.[1] However, the time and cost associated with MRI may limit optimal care for some individuals. Another challenge in managing brain abscesses is identifying the causative microorganism and determining appropriate treatment. Common microorganisms include *Staphylococcus aureus*, Streptococcus species, and anaerobic bacteria like Fusobacterium.[1,2] Initial empirical antibiotic therapy should cover these, often starting with vancomycin for gram-positive coverage, combined with ceftriaxone or cefepime for broad-spectrum gram-negative coverage, and metronidazole for anaerobes.[2] In cases where *F necrophorum* is identified, as in this patient, treatment may be escalated to include meropenem or clindamycin based on susceptibility patterns.

In our patient, *F necrophorum* was identified through bacterial cultures and Gram stain of a cranial abscess sample. The isolation of this common oral flora from the cranial abscess and severe inflammation observed in the ethmoid sinus during surgery confirmed that the infection ascended from the sinuses to the brain. Bacterial cultures from intracranial surgery revealed the organism was susceptible to clindamycin, metronidazole, and penicillin, a typical susceptibility pattern for this bacterium species. Despite appropriate initial antibiotic therapy, the patient had symptom recurrence. This is believed to have been due to worsening inflammation from corticosteroid discontinuation rather than worsening infection from inadequate antibiotics because there was no abscess recurrence and repeat craniotomy cultures remained negative. Nonetheless, the patient was escalated to higher-intensity antibiotics and additional corticosteroids per neurosurgical recommendation. This case highlights a gap in our understanding of the pathogenesis of *F necrophorum* and proposed duration of the resultant cerebral inflammation, which is a potential area for further research.

Also of interest is the lack of growth on the initial sinus cultures, which may have occurred due to method of culturing or partial response of the infection to previously administered antibiotics.[8] In cases such as these, metagenomic next generation sequencing has been shown to rapidly reveal underlying microorganisms with reduced impact from prior antibiotic exposure.[13]

CONCLUSION

This case highlights the complexities involved in

diagnosing and managing sinusitis and its potential complications in healthy individuals. The insidious onset of symptoms, such as headache and fever without meningeal signs, can mimic other intracranial pathologies, leading to delays in diagnosis and management. Given risk of neurologic deterioration secondary to mass effect and shift changes with elevated intracranial pressure, CT should be obtained prior to considering lumbar puncture in patients in when both cerebral abscess and meningitis are in the differential diagnosis. Early recognition and prompt intervention is crucial to prevent severe outcomes, such as elevations in intracranial pressure, brain herniation, sepsis, reinfection, and death. It is important to consider atypical pathogens, such as *F necrophorum*, to identify pathogens quickly and accurately, allowing for optimization of antimicrobial management. Our case illustrates the importance of comprehensive diagnostic and management approaches by a multidisciplinary care team for patients faced with complex and illusive pathophysiology.

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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The Jaw-Locking Case of a Missed Tetanus Booster

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Introduction: Tetanus is a now rare disease due to the widespread administration of scheduled and prophylactic vaccines, making it exceptionally uncommon to appear in many emergency departments. Clinical suspicion alone is used to make the diagnosis as there are currently no immediately available diagnostic tests available to the clinician. If left unrecognized and untreated, however, tetanus can lead to airway compromise and death.

Case Report: We report a case of a young male who presented to the emergency department with intermittent full body spasms and stiffness of the masseter muscles in the setting of recent assaults and lacerations weeks prior who had not received tetanus since 2008. Immediate calls were placed to infectious disease consultants and the patient was treated with intravenous immunoglobulin, tetanus immunization, metronidazole, and ceftriaxone. Further work up revealed rhabdomyolysis, elevated lactate, and unremarkable imaging.

Conclusion: Following treatment, the patient's symptoms improved to resolution with completion of therapy, effectively confirming the diagnosis of tetanus. [Clin Pract Cases Emerg Med. 2025;19(2):154-156.]

Keywords: *Case Report; Tetanus; Infectious Disease; Immunization.*

INTRODUCTION

Tetanus is a serious infectious disease caused by *Clostridium tetani*. This bacterium forms a toxin which causes inhibition of gamma-aminobutyric acid neurotransmission, leading to muscle spasms resulting in airway obstruction and death. Rarely seen in countries with robust vaccination efforts, tetanus is a diagnostic challenge in the emergency department (ED). Although many patients present with open wounds, up to 30% of patients have no entry apparent.[1] Generalized tetanus is seen in 80-84% of cases, which is characterized by full body spasms and can progress to autonomic instability.[2] Lockjaw is the most common presenting symptom. Neonatal tetanus is much less common with current immunization guidelines for pregnancy and pediatrics. Local tetanus, making up between 2-13% of cases, is defined by muscle spasms proximal to the site of injury, and can either self-resolve over the course of weeks or progress to generalized tetanus.[2] Cephalic tetanus, the rarest form of tetanus accounting for approximately 1-3% of cases, is localized to the face and causes flaccid paralysis rather than spasm (Table).[3] Complications of tetanus include rhabdomyolysis, long bone

Table: Types of Tetanus

Type	Prevalence
Generalized	80-84%
Localized	2-13%
Cephalic	1-3%

fractures, respiratory compromise, and aspiration pneumonia.

CPC-EM Capsule

What do we already know about this clinical entity?
We understand the pathophysiology, causative organism, and different presentations of tetanus.

What makes this presentation of disease reportable?
This case is reportable due to the rare nature of tetanus. Reports such as this are often the only exposure most clinicians have to this pathology.

What is the major learning point?
This case discusses the immediate diagnosis and treatment of tetanus in the emergency department.

How might this improve emergency medicine practice?
This case calls to attention the opportunity for improved tetanus vaccination in the emergency department as well as provides a guideline for treatment of tetanus.

CASE REPORT

A 29-year-old male patient was brought to the ED by emergency medical services complaining of full body spasms and jaw pain. Upon initial evaluation, the patient was afebrile, and then suddenly became extremely diaphoretic, tachycardic, hypertensive and exhibited difficulty speaking. He was able to track clinicians in the room, shake his head yes or no, and was alert throughout the duration of the episode. His upper extremities and abdomen were clenched, back was arched, and neck was turned to the side. This episode resolved spontaneously, the patient was not post-ictal, and further evaluation continued. The patient denied taking any substances prior to presentation, denied medications, and denied past medical history.

Physical examination was notable for multiple wounds on the right hand, including a surgical site with suture on the dorsal fifth digit of the right hand, open wound on the right palm, and healing laceration of the left face. None of the wounds had erythematous bases or purulent discharge.

The differential for this patient included: serotonin syndrome, neuroleptic malignant syndrome, dystonic reactions, toxidrome, rabies, orofacial infection, and strychnine poisoning, all of which were less likely due to self-resolving episodes.[1,3] Seizure was considered less likely due to bilateral extremity involvement and the ability of patient to track and to respond throughout episodes. Benztropine therapy was a consideration to differentiate between tetanus and a drug-induced dystonic reaction.[4]

Labs were significant for elevated creatine kinase (CK) to 2678 Units per liter (U/L) (reference range: 0-200 U/L), creatinine 1.28 milligrams per deciliter (mg/dL) (0.6-1.20 mg/dL), lactate 9.2 millimoles (mmol)/L (0.5-2.2 mmol/L), and calcium 9.6 mg/dL (8.5-10.5 mg/dL). Computed tomography head showed no acute intracranial pathology.

Chart review demonstrated recent admission two months prior for a hand infection requiring operative washout and intravenous antibiotics, and other visits for intoxication over the past year. Per chart review, the patient had received full initial tetanus series in childhood and booster in 2008, 14 years prior to this presentation.

Approximately two hours after initial presentation, the patient had another episode of spasm, for which 2.5 mg midazolam was provided to cease the spasm. Infectious disease was consulted with concern for tetanus given open wounds, intermittent body muscle spasms with stiffness of the masseter muscles, back arching, and rigid paralysis. The infectious disease team agreed with the plan of intravenous immunoglobulin, tetanus immunization, and antibiotic coverage with metronidazole and ceftriaxone. Neurology was consulted as seizure was on the differential; on evaluation the neurology team agreed that seizure was unlikely, and recommended assessment for stiff person syndrome if there was no improvement with tetanus treatments. The patient was ultimately admitted to the critical care service for close airway monitoring.

After the patient's admission, he did not have any more episodes of muscle spasms, his CK down-trended and the patient was transferred to the medical floor. He was able to be transitioned to oral antibiotics and was provided strict return precautions and instructions for completing tetanus immunization series via return to the ED or through primary care provider referral provided. Five days after hospital discharge, the patient reported to the ED with leg spasms and reported he had been unable to pick up antibiotics from the pharmacy. He was provided additional antibiotics and prescriptions. No further encounters were recorded.

DISCUSSION

This case highlights several key points: early tetanus course can present as intermittent spasms with autonomic instability, aggressive management early can lead to favorable outcomes, and the ED has an opportunity for continued tetanus immunization, especially in vulnerable populations. There is data to suggest a decline in protective antibodies against tetanus with age, especially at age 60 and beyond.[5] Vaccination response declines with age as well, so despite having a booster within the last ten years, this population may

not be entirely protected with the standard schedule.[5] This population often presents to the ED with trauma and falls, and tetanus could be overlooked or not administered.[5]

Tetanus is a rare diagnosis in developed countries due to widespread vaccinations. However, tetanus should be included on the differential, particularly in emergency departments that treat immigrant populations, people who have limited access to consistent health care, or patients who have not been fully vaccinated. Tetanus can have a spectrum of presentations, including the above description of intermittent spasms.[2,3] Recognizing this disease process early can lead to initiation of treatment and more favorable patient outcomes. In this case, we were able to initiate treatment for tetanus prior to continuous spasms and airway compromise, leading to less invasive management of the patient. Metronidazole and ceftriaxone were chosen in conjunction with infectious disease consultation, for aerobic and anaerobic coverage, and with review of prior case reports. The role of antibiotics is to prevent multiplication of the bacteria, therefore limiting the toxin production and reducing mortality. We were able to provide resources for continuation of tetanus treatment and immunization.

An additional population which is at risk for tetanus includes patients involved in natural disasters. The emergency department is frequently activated for disaster scenarios, and therefore has a unique opportunity to evaluate and triage patients in the midst of a disaster. Tetanus outbreaks can occur in natural disasters due to risk for puncture and soft tissue injuries in conjunction with the spore forming nature of the causative organism, *Clostridium tetani.*[6] Recognizing the need for immunization in addition to triage and treatment of injuries is important in the setting of natural disease, as well as maintaining a high clinical suspicion for tetanus the weeks following natural disasters in patients who had exposure.

CONCLUSION

Despite vaccination rates and education, certain populations are still under-immunized or not fully protected from tetanus. These include patients who have not been compliant with vaccination status, have not had access to routine care for scheduled immunizations, victims of a natural disaster and the elderly, who are often under protected despite being vaccinated.[5] The emergency department provides a unique opportunity to increase immunity for at-risk groups and recognize the spectrum of presentations of tetanus. This case demonstrates, however, a patient who had had regular encounters with the healthcare system, including for treatment of the wounds that are presumed to have been the entry source for the tetanus toxin, and yet his tetanus vaccination status had been overlooked. This illustrates the need for attention to the mundane topic of tetanus vaccine status and the role of the ED in preventative healthcare.

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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BRASH Syndrome in the Absence of Chronic Kidney Disease: A Case Report

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Introduction: Bradycardia, Renal failure, Atrioventricular nodal blockade, Shock, Hyperkalemia (BRASH syndrome) is commonly misdiagnosed in the emergency department, which can lead to a delay in care and poor patient outcomes.

Case Report: We present a case of BRASH syndrome in a patient with no underlying renal disease, which further complicated diagnosis and delayed treatment.

Conclusion: Prompt recognition of the underlying pathophysiology in cases of BRASH syndrome is essential to guide treatment and avoid delays in care. [Clin Pract Cases Emerg Med. 2025;19(2):157-160.]

Keywords: *Case report; BRASH; shock.*

INTRODUCTION

Bradycardia, Renal failure, Atrioventricular nodal blockade, Shock, Hyperkalemia (BRASH syndrome), first described in 2016, refers to a cascade of physiological abnormalities that is often misdiagnosed in the Emergency Department as either hyperkalemia or bradycardia. Anchoring solely on treating one of these aspects of the full syndrome is common and can lead to a delay in adequate care and worsening of the full clinical picture, with increasing likelihood of cardiovascular collapse in these patients.[1] Understanding of the synergistic effects of hyperkalemia, atrioventricular nodal blockade, and renal failure allows timely and effective treatment of this syndrome. The following case describes a patient without chronic kidney disease who developed BRASH syndrome and severe hemodynamic instability. This case illustrates the importance of prompt recognition and holistic treatment of this novel syndrome, even in patients without underlying renal dysfunction.

CASE REPORT

An 81-year-old female with a past medical history of hypertension, congestive heart failure, atrial fibrillation, diabetes mellitus, and asthma on twice daily 50 mg metoprolol and once daily 25 mg lisinopril presented to the emergency department via medics for evaluation of fatigue. The patient had a left total hip arthroplasty performed four days prior to arrival. She complained of fatigue over the two days prior to presentation. Upon arrival to the emergency department, the patient was notably fatigued to the point of requiring multiple verbal prompts to answer questions. She was oriented to person, place, and time. The patient denied any associated symptoms, solely complaining of severe fatigue. Initial vital signs included mild bradycardia at 57 beats per minute as well as hypotension with a blood pressure of 71/56 millimeters of mercury (mm Hg). On physical exam, mild pitting edema of the bilateral lower extremities was noted as well as mild wheezing in all lung fields. The patient was noted to be hypoxic with oxygen saturation of 88%. She was noted to

have increased work of breathing and was placed on supplemental oxygen via nasal cannula with improvement to 95% oxygen saturation.

Patient's workup revealed an acute kidney injury with a creatinine of 3.1 milligrams per deciliter (mg/dL) (reference range 0.44 – 1.03 mg/dL) from a baseline of 1.08 mg/dL per chart review (notably the reference range from that facility was 0.6-1.3 mg/dL). Pertinent lab values included a potassium of 5.5 millimoles per liter (mmol/L) (3.5 – 5.5 mmol/L), high sensitivity troponin of 393 picograms (pg)/mL (0 – 12 pg/mL), lactic acid of 3.1 mmol/L (0.5 – 2.0 mmol/L), beta natriuretic peptide of 736 pg/mL (0 – 100 pg/mL). Twelve lead electrocardiogram showed atrial fibrillation with slowed ventricular response. (Image) Her blood pressure initially improved to 90 mm Hg systolic after one liter normal saline bolus however her bradycardia worsened with the heart rate intermittently lowering into the 30s. One mg intravenous atropine was administered with no effect. While awaiting lab results, the patient's mentation and blood pressure began to decline, leading to central line placement and administration of norepinephrine with subsequent improvement in both heart rate and blood pressure, though the patient remained mildly altered. Cardiology was consulted and with concern for unintentional overdose of metoprolol recommended holding this medication and continuing supportive care. The patient was then admitted to the intensive care unit for further management.

Overnight the patient's renal function continued to decline despite intravenous fluid resuscitation. With concern for possible sepsis, blood cultures, respiratory culture, and urine culture were all collected, but all showed no growth at 48 hours. Her hospital course was complicated by a course of hyperkalemia (serum potassium level was 6.4 mmol/L) and she received standard treatments for hyperkalemia of calcium, insulin, dextrose, and sodium zirconium cyclosilicate. At this point, with low suspicion of septic shock and rising concern for BRASH syndrome, she received further intravenous fluids and was continued on vasopressor support. After five days of continued care focused on holding atrioventricular nodal blocking agents and medical treatment of hyperkalemia, the patient's renal function returned to baseline and she was ultimately discharged home.

CPC-EM Capsule

What do we already know about this clinical entity?
Bradycardia, renal failure, atrioventricular nodal blockade, shock, and hyperkalemia (BRASH) syndrome syndrome is a vicious cycle of worsening renal function and bradycardia, fueled in part by atrioventricular nodal blockade and hyperkalemia, eventually leading to cardiogenic shock.

What is the major learning point?
The image shows the severity of bradycardia that can be induced, while the figure shows a simplified flowchart of the pathophysiology of BRASH syndrome

How might this improve emergency medicine practice?
More awareness of this syndrome, in particular even in patients without underlying renal

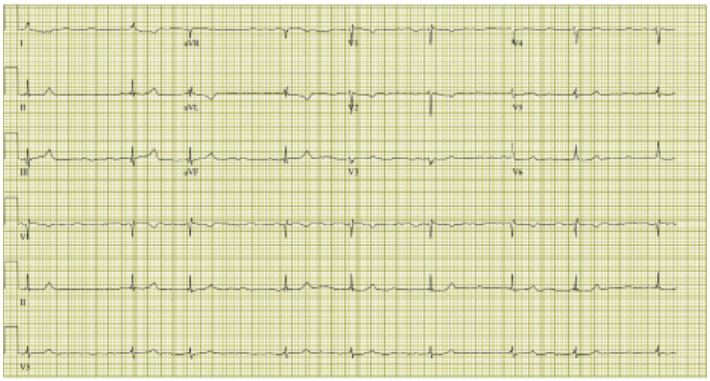


Image. Electrocardiogram taken in Emergency Department depicting atrial fibrillation with slowed ventricular response.

DISCUSSION

Bradycardia, renal failure, atrioventricular nodal blockade, shock, and hyperkalemia syndrome is an emerging cascade of symptoms that can lead to significant morbidity and mortality if not correctly diagnosed and treated. The mixture of atrioventricular nodal blockade, often due to a beta blocker or calcium channel blocker, with renal disease leads to a vicious cycle of decreased clearance of said atrioventricular nodal blocker, which therefore leads to worsening bradycardia and ultimately cardiogenic shock. As cardiac output decreases, renal perfusion is further compromised, leading to a deterioration in renal function (Figure). Misdiagnosis of the etiology for the patient's cardiogenic shock can lead to less optimal treatment and can literally be a fatal mistake. While it can be challenging to parse through the multilayered pathophysiology that leads to the cardiogenic shock, anchoring on only one portion of BRASH syndrome can lead

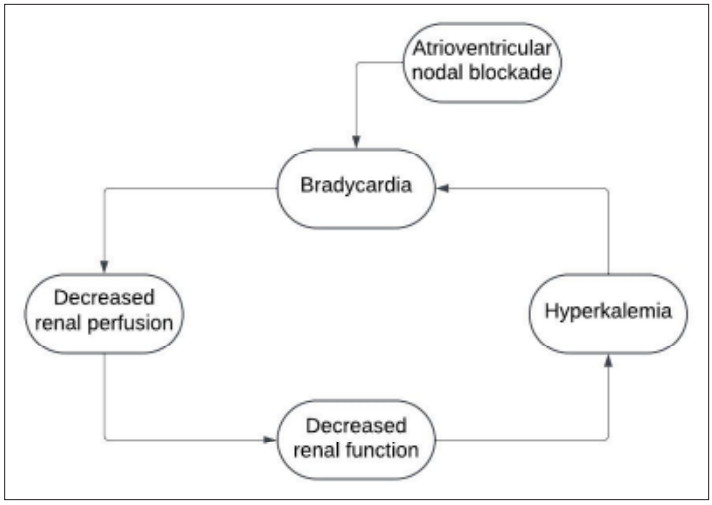


Figure. Flowchart illustrating synergistic effects of this syndrome that lead to hemodynamic collapse.

to a delay in the wholistic treatment needed for these patients and poor outcomes. This emphasizes the importance of having a high index of suspicion for BRASH syndrome when presented with a patient that is exhibiting bradycardia and/or signs of shock when sepsis and other common causes have been determined to be less likely.

Curiously, our patient had exhibited no renal dysfunction prior to this presentation and admission. Generally, patients suffering from BRASH syndrome will have some aspect of chronic kidney disease[1] that contributes to the spiraling effects of the syndrome. This case illustrates that even patients without prior kidney disease are at risk for this syndrome. The elderly population in particular are at elevated risk, as these patients are often prescribed atrioventricular nodal blockade medication and are particularly susceptible to prerenal acute kidney injury.

It is important to note that hemodynamic instability can arise from seemingly mild lab abnormalities due to the synergistic nature of this syndrome. While the initial potassium for this patient was technically within normal limits, in the setting of her decreased renal function and atrioventricular nodal blockade this still led to life threatening bradycardia and cardiogenic shock. Subsequent testing showed an elevated potassium, leading to confirmation of the diagnosis, prompt treatment, and a positive outcome for the patient. However, it is easy to dismiss this syndrome if the patient has not yet crossed the threshold into true hyperkalemia, contributing to the difficulty of diagnosis.

While atrioventricular nodal blockade plays an obvious role in decreasing cardiac output via bradycardia, it is unclear if the declining renal function leads to decreased clearance of the atrioventricular blocking agent. For example, metoprolol is commonly found on the medication lists of patients with BRASH syndrome, however declining renal function does not seem to effect the clearance of metoprolol.[2,3] Atenolol, the clearance of which is effected by renal dysfunction,[4] has also been the atrioventricular blockade agent in prior documented cases of BRASH. Further research is required to ascertain whether the renal dysfunction observed in these patients contributes to more significant bradycardia and/or hypotension, contingent on the specific atrioventricular blocking agent being used.

Determining which aspect of this syndrome actually initiates the cascade of events in a given case can be challenging. The patient in our report had been on beta-blocking medications for years without complication. Renal insult is often the first step towards this syndrome, caused by either renally dosed medications or dehydration in patients with prior kidney disease.[3,5] The patient's only complaint was fatigue, which is often the only reported symptom in cases of BRASH syndrome. Given the patient's history of recent surgery and decreased activity level at home, it is reasonable to presume that simple dehydration with resultant pre-renal azotemia is what began her decline, which has been seen in some cases of BRASH syndrome.[5,6]

Treatment for BRASH syndrome is focused on the treatment of hyperkalemia and improving cardiac output/renal perfusion via vasopressor support. Treatment of hyperkalemia should follow standard protocols while vasopressors and inotropes can be used concomitantly to transiently restore hemodynamics. Intravenous fluids should also be considered for those patients appearing volume responsive. There is often no need for transcutaneous or transvenous pacing if the syndrome is diagnosed quickly and treated correctly. Atropine is often attempted however given atropine's mechanism of action (antagonism of muscarinic receptors) is not targeting the atrioventricular nodal blockade nor helping to clear potassium it will have no effect in this syndrome. Overall, treatment of BRASH syndrome is relatively simple; diagnosing this rare and deadly disease state is where the difficulty lies.

CONCLUSION

A newly emerging clinical entity, BRASH syndrome can be difficult to diagnose and deadly if not treated promptly. While this syndrome is most commonly seen in patients with history of renal disease, this case illustrates that even patients with normal renal function at baseline are at risk. A high index of suspicion must exist, even in patients with normal baseline renal function, in order for this syndrome to be properly diagnosed and treated correctly.

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

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Gastrosplenic Fistula in the Setting of Undiagnosed Lymphoma: A Case Report

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Introduction: A gastrosplenic fistula (GSF) is a pathologic connection between the spleen and stomach that can lead to life-threatening complications. A GSF can arise spontaneously but is often secondary to a variety of etiologies. Most commonly, GSFs arise from gastric or splenic non-Hodgkin diffuse large B-cell lymphomas (DLBCL). Only 46 cases of GSFs have been published to date, and due to its rarity extensive literature review is insufficient for characterization of GSFs.

Case Report: This case discusses a patient with intermittent abdominal pain and weight loss, which led to the diagnosis and treatment of a GSF and DLBCL. The patient later went into remission for his DLBCL but succumbed to respiratory failure from a secondary abdominal-pleural fistula formation. Gastrosplenic fistulas have the potential to cause fatal, massive, upper gastrointestinal hemorrhages, infections, other fistulas, or esophageal obstructions. A delay in diagnosis corresponds with a higher morbidity and mortality; thus, prompt detection and treatment are imperative. The management of GSFs is complex and requires a multidisciplinary approach to care.

Conclusion: In this report we review GSFs in the emergency care setting with the goal of increasing awareness to facilitate their diagnosis. [Clin Pract Cases Emerg Med. 2025;19(2):161-164.]

Keywords: *case report; fistula; splenomegaly; lymphoma; psoriasis.*

INTRODUCTION

While some anatomic fistulas are well known and even expected in certain disease states, gastrosplenic fistula (GSF) is a rare and potentially fatal entity. Among the various disease states that have been shown to cause a GSF, diffuse large B-cell lymphoma (DLBCL) is the most common.[1] One potential explanation for this may be due to the lack of a desmoplastic reaction in lymphomas allowing rapid growth, gastric wall invasion, and tumor necrosis, a constellation not seen in adenocarcinomas.[2] Other causes of GSF include chemotherapy-induced tumor lysis syndrome, diffuse histiocytic lymphoma, splenic abscess, peptic ulcer disease, Hodgkin lymphoma, gastric adenocarcinoma, extranodal natural killer/T-cell lymphoma, Crohn disease, sleeve gastrectomy, and trauma.[1] Development of a GSF has been documented in less than 1% of gastric cancers.[2] Diffuse large B-cell lymphoma can arise in any tissue, but the most common site of extranodal disease is found in the stomach.[3]

Formation of a GSF is often facilitated by splenic enlargement and its proximity to the gastric fundus.[4] The GSF is often forged through a chronic process involving tissue necrosis secondary to lymphoma and infiltration into the gastric wall and splenic capsule.[1] This direct communication between the stomach and spleen allows for the passage of gastric contents into the spleen, leaving the organ vulnerable to damage due to the acidity of gastric contents. Enlargement of the spleen due to inflammation and intraparenchymal air produced by tissue necrosis can irritate the diaphragm, potentially leading to pleural effusions, splenic perforation, abscesses, or splenopleural fistula formation.[5]

Clinically, GSFs have a wide spectrum of presentations from asymptomatic to hemorrhagic shock, which makes detection and diagnosis difficult. However, the most commonly reported symptom is abdominal pain.[6] A worse prognosis is associated with an initial presentation of gastrointestinal (GI) bleeding, which often involves the splenic artery.[6] Complications of GSF may include gastric perforation, infection, spleen destruction, abscess, pleural effusion, fistula formation with other organs, and more rapid metastasis.[6]

Abdominal computed tomography (CT) with contrast is the preferred imaging method for diagnosing GSF, but upper GI endoscopy can also provide direct visualization.[2,6] Significant findings indicating a GSF include air in the spleen, splenomegaly, gastric ulceration, and communication between the spleen and stomach.[2] Treatment varies depending on the presentation, extent of fistula formation, and organ damage. Currently, an approved treatment for hemodynamically stable, active bleeding is with splenic artery embolization performed by an interventional radiologist.[6] Surgical resection including splenectomy and gastrectomy for treatment of GSF is generally considered first-line treatment, but due to the rarity of the condition there is no clear consensus on this.[2]

CPC-EM Capsule

What do we already know about this clinical entity?
Gastrosplenic fistulas (GSF) are rare and often form in the presence of lymphoma. Initial symptoms can range from generalized abdominal pain to massive gastrointestinal hemorrhaging.

What makes this presentation of disease reportable?
This patient was previously undiagnosed, making it an unusual first presentation of diffuse large B-cell lymphoma.

What is the major learning point?
Due to the rarity and potentially fatal outcome of a GSF, it is imperative to expedite early detection and develop improved therapeutic strategies.

How might this improve emergency medicine practice?
Physicians should maintain a high index of suspicion for GSF, particularly in patients with diffuse large B-cell lymphoma.

CASE REPORT

A 59-year-old White male with a past medical history of hypertension, coronary artery disease, obesity, and psoriatic arthritis presented to an urgent care with intermittent left upper and lower quadrant abdominal pain, dark urine, fatigue, and nausea, which had gradually increased in severity over five weeks. Over the course of two months, the patient reported unintentional weight loss of 25 pounds but denied any melena, hematochezia, or tenesmus. Upon his arrival to the urgent care, he had a blood pressure of 145/88 millimeters of mercury, heart rate of 90 beats per minute, respiratory rate of 18 breaths per minute, temperature of 37 °Celsius, and 99% oxygen saturation on room air. The patient had moderate upper and lower abdominal tenderness on physical exam with normal bowel sounds and no palpable masses or organomegaly. Laboratory testing revealed a white blood cell count of 15.0 x10^3 cells/microliter (μL) (reference range: 5-10 x 10^3 cells/μL). All remaining lab values including hemoglobin, platelet count, basic metabolic panel, amylase, lipase, and coagulation studies were within normal limits.

A CT of the abdomen and pelvis with and without contrast was performed revealing a large heterogeneous-appearing spleen with gas appearing to extend from the body of the stomach into the splenic hilum (Images 1 and 2). Image 3 shows a small, left-sided pleural effusion with atelectasis or infiltrate in the left lung base.

The diagnosis of a GSF was made, and the patient was admitted to the hospital where he underwent treatment for the next few weeks. During that time, an esophagogastroduodenoscopy was performed showing inflammation at the gastric fundus and a perforated ulcer with abscess formation and abnormal mucosa. A biopsy was obtained and tested positive for tumor markers and pathology consistent with DLBCL. During the surgery, a large 7.0 x 6.0-centimeter gastric tumor three inches inferior to the gastroesophageal junction was visualized. Perforation into the splenic capsule caused splenomegaly and tumor invasion to the diaphragm on the left, the pancreatic tail; the left lobe of the liver was also identified and treated during the procedure. The patient underwent a splenic embolization followed distal esophagectomy, total gastrectomy, omental pedicle flap, Roux-en-Y esophagojejunostomy, splenectomy, distal pancreatectomy, feeding jejunostomy tube placement, small bowel resection, and partial left hepatectomy. He tolerated the procedure well and was later treated by oncology for stage-III DLBCL with chemotherapy. Despite achieving remission, the patient died of complications three years later.

DISCUSSION

This patient had a GSF secondary to DLBCL. Gastrosplenic fistulas have a wide range of presentations, which could be a potential cause of delayed treatment. Documented cases of GSFs have included the following presenting symptoms: abdominal pain, splenomegaly, constitutional

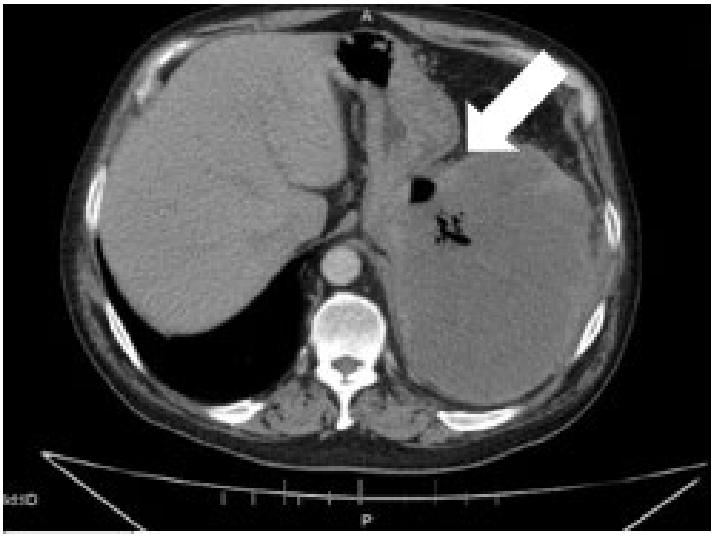


Image 1. Computed tomography abdomen and pelvis axial view displaying the presence of gas (arrow) within the spleen.

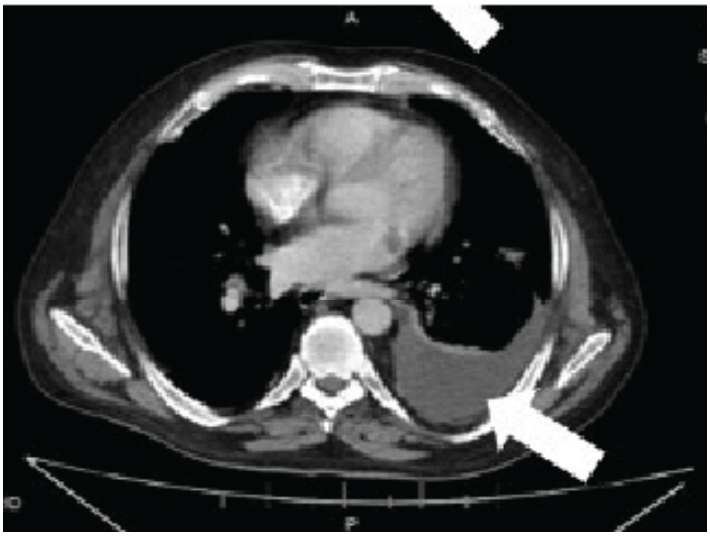


Image 3. Computed tomography abdomen and pelvis showing a pleural effusion (arrow) of the left lung base with atelectasis.

symptoms, hematemesis, melena, gastric hematoma, hemorrhaging, splenic abscess, nausea, and weakness.[1] Most documented cases of GSFs have been in males with an average age of 50 years and history of lymphoma.[1]

While GSF remains a rare occurrence, DLBCL is associated with numerous risk factors including the presence of DLBCL.[3,7] Current data indicates that DLBCL occurs most frequently in males with a median age of 55 years who are White, and individuals with a body mass index greater than 30 kilograms per meter squared (kg/m^2) (18.5-24.9 kg/m^2), all of which put the patient in this case at increased risk.[3] Recent studies have also shown a significant increase in incidence and mortality of DLBCL in individuals with an underlying history of autoimmune disease.[8] While this association has often been connected to B-cell mediated diseases such as rheumatoid arthritis or systemic lupus erythematosus, one study from 2019 showed decreased survival rates due to non-Hodgkin lymphoma (NHL) in patients with psoriatic arthritis, a T-cell mediated disease.[8] While it remains controversial, articles dating back to 1993 have provided evidence that methotrexate has been shown to cause lymphoproliferative diseases such as NHL that later went into remission with cessation of the drug; this phenomenon is a disease now termed methotrexate-associated lymphoproliferative disorder.[7] There is some evidence of increased risk of lymphoma specifically in patients with psoriatic arthritis being treated with methotrexate.[9] The patient in this case had been taking methotrexate to treat his psoriatic arthritis for five years, potentially placing him at increased risk for DLBCL.



Image 2. Computed tomography abdomen and pelvis coronal image showing the presence of gas (arrow) within the spleen.

Patients with a GSF treated with surgery generally have a good prognosis but often have a propensity for developing complications from surgery due to a weakened immune system.[1,10] Surgical treatment of GSF varies but frequently involves both splenectomy and gastrectomy.[2] Potential complications of a splenectomy include increased risk of infection with encapsulated organisms, bleeding, venous thromboembolism, and many cancers.[10,11] Gastrectomy complications include anastomotic leakage, stenosis, dumping syndrome, abscesses, perforation, renal dysfunction, respiratory complications, and anemia.[12] In a systematic review conducted in 2017, the most common cause of death from GSFs was gastric perforation followed by progression of lymphoma and pulmonary infection with multi-organ failure.[1]

Overall, when the initial presentation of the GSF is not in the setting of massive GI hemorrhaging, patients have an 82% survival rate.[6]

CONCLUSION

Because they are rare, insufficient data exists regarding gastrosplenic fistulas; thus, it is important to document cases to expedite early detection and foster the development of improved therapeutic strategies. It is essential to keep a high level of suspicion for GSF in patients with DLBCL to prevent fatalities. Further studies should be conducted to increase awareness of GSFs and improve patient care.

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

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De Garengeot Hernia with Acute Gangrenous Appendicitis Case Report

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Introduction: A De Garengeot hernia is defined as a femoral hernia that contains the vermiform appendix. While femoral hernias carrying the appendix are uncommon, strangulation of the appendix in the hernial sac with concurrent acute appendicitis is an extremely rare and life-threatening condition often presenting with an atypical clinical picture.

Case Report: A 51-year-old man presented to the emergency department with two weeks of persistent right inguinal pain after heavy lifting. Imaging revealed suspicion for an Amyand hernia, an inguinal hernia containing a portion of the appendix. However, intraoperative findings revealed a strangulated De Garengeot hernia with gangrenous appendicitis.

Conclusion: De Garengeot hernias are femoral hernias containing the appendix. They are diagnostically challenging and require urgent surgical evaluation and intervention given high risk for strangulation. [Clin Pract Cases Emerg Med. 2025;19(2):165-168.]

Keywords: *De Garengeot hernia; Amyand hernia; acute gangrenous appendicitis; case report.*

INTRODUCTION

Femoral hernias are protrusions of bowel or abdominal tissue across a weakened abdominal wall into the femoral canal, which is bordered by the inguinal ligament anterosuperiorly, the femoral vein laterally, and the lacunar ligament medially. While femoral hernias make up approximately 5% of all hernias and are not as common as inguinal hernias, 15-20% of all femoral hernias strangulate.[1] Additionally, an estimated 0.5-5% of femoral hernias contain the appendix, specifically referred to as a De Garengeot hernia.[2,3] Of note, 0.08-1.013% of De Garengeot hernias are complicated by appendicitis.[2,3] Although the combination of physical exam and computed tomography (CT) have proved valuable in assisting with the diagnosis of De Garengeot hernias, the rate of misdiagnosis remains high.[4] Preoperative diagnosis of De Garengeot hernia is only made in approximately one-third of patients, thus posing diagnostic challenges to the emergency physician.

CASE REPORT

A 51-year-old male presented to the emergency department (ED) with two weeks of sudden onset, persistent right inguinal pain after heavy lifting. The patient noted that he experienced a constant dull ache with intermittent sharp pain in the right inguinal region and right lower abdominal quadrant, emphasizing that the symptoms seemed similar in nature to his history of kidney stones. He also noticed a bulge in the right inguinal area, which had developed overlying erythema. He had a recent urgent care visit and was prescribed tamsulosin for a presumed kidney stone with no imaging or laboratory studies obtained at the time. The tamsulosin minimally helped with resolution of symptoms. In addition to the inguinal and abdominal pain, he also complained of subjective fevers and chills, intermittent nausea, multiple episodes of non-bloody, non-bilious vomit, and dysuria. Review of systems was otherwise negative for headache, chest pain, shortness of breath, hematochezia, melena, and testicular pain.

Past medical history was significant for multiple sclerosis, attention-deficit/hyperactivity disorder, hypertension, gastroesophageal reflux disease, and chronic lower back pain. Surgical history was notable for laparoscopic gastric fundoplication approximately 10 years prior and tibial open reduction and internal fixation. The patient used marijuana daily. Outpatient medications included amphetamine/dextroamphetamine, cyclobenzaprine, famotidine, hydrocodone and acetaminophen, and tamsulosin.

Upon arrival, the patient had a heart rate of 103 beats per minute, blood pressure of 134/83 millimeters of mercury, respiratory rate of 22 breaths per minute, oxygen saturation of 99% on room air, and temperature of 36.4 °Celsius. The head, eyes, ear, nose and throat exam was unremarkable. Cardiovascular exam was normal, and pulmonary exam revealed clear and equal breath sounds bilaterally. Abdominal exam was nondistended but notable for a firm abdomen with a tender, nonreducible area of swelling in the right inguinal fold with no guarding or rebound tenderness. Skin exam exhibited overlying erythema in the right inguinal region (Image 1).

Initial laboratory studies obtained included a complete blood count, comprehensive metabolic panel, and urinalysis. Complete blood count revealed normal white blood cell count, hemoglobin, and platelet count. Comprehensive metabolic panel showed normal electrolytes and creatinine, and no anion gap. Furthermore, albumin, alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase, and total bilirubin were all within normal limits. Urinalysis showed some ketones and trace leukocyte esterase. Abdominal and pelvis CT with intravenous and oral contrast showed the appendix extending into a fat-containing right groin hernia with evidence of thickening of the appendix and periappendiceal inflammatory change, as well as inflammatory changes around the hernial sac (Images 2, 3).

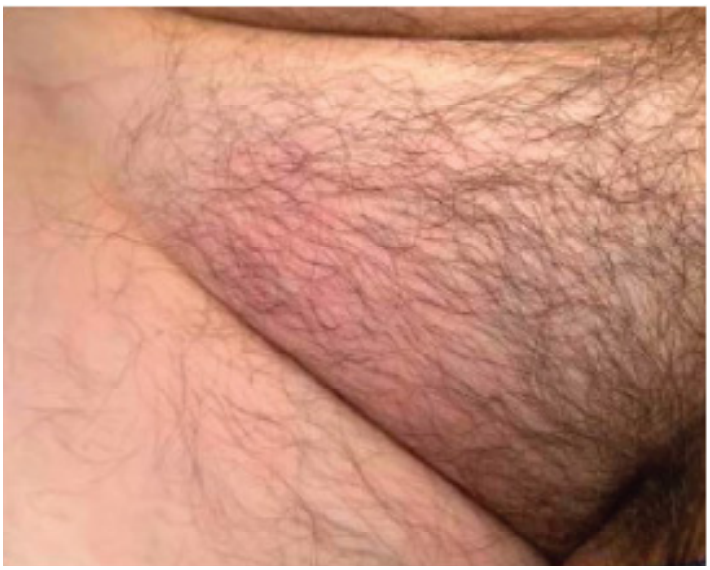


Image 1. Right groin with overlying erythema and swelling.

Appendiceal diameter was noted to be 1.3 centimeters. Other incidental findings included hepatic cysts, a right renal cyst, and diverticulosis. The radiologic diagnosis was an inguinal hernia containing the appendix (Amyand hernia) with concurrent presence of acute appendicitis.

The patient was started on piperacillin-tazobactam. General surgery was consulted and urgently took the patient to the operating room (OR) with a plan for a laparoscopic appendectomy with possible incarcerated right inguinal hernia repair. Intraoperative findings instead showed a femoral hernia with a strangulated gangrenous appendix, known as a De Garengeot hernia. He underwent a successful laparoscopic appendectomy with primary repair of the right femoral hernia. His postoperative course was uncomplicated with the patient tolerating diet and voiding and ambulating independently. He was deemed medically stable and ultimately discharged on postoperative day two.

CPC-EM Capsule

What do we already know about this clinical entity?
While femoral hernias make up approximately 5% of all hernias and are not as common as inguinal hernias, 15-20% of all femoral hernias strangulate.

What makes this presentation of disease reportable?
We present a rare case of femoral hernia and a unique case of a De Garengeot hernia with the presence of acute gangrenous appendicitis.

What is the major learning point?
Given the diagnostic challenges, clinicians should be aware that gangrenous appendicitis is an uncommon, life-threatening complication of the femoral hernia.

How might this improve emergency medicine practice?
Early surgical consultation is imperative, as preoperative diagnosis of a De Garengeot hernia is associated with improved patient outcome.

DISCUSSION

While lifetime occurrence of a groin hernia is 27-43% in men and 3-6% in women, femoral hernias typically have a



Image 2. Axial view of computed tomography of the abdomen pelvis showing the appendix within a femoral hernia (arrow).

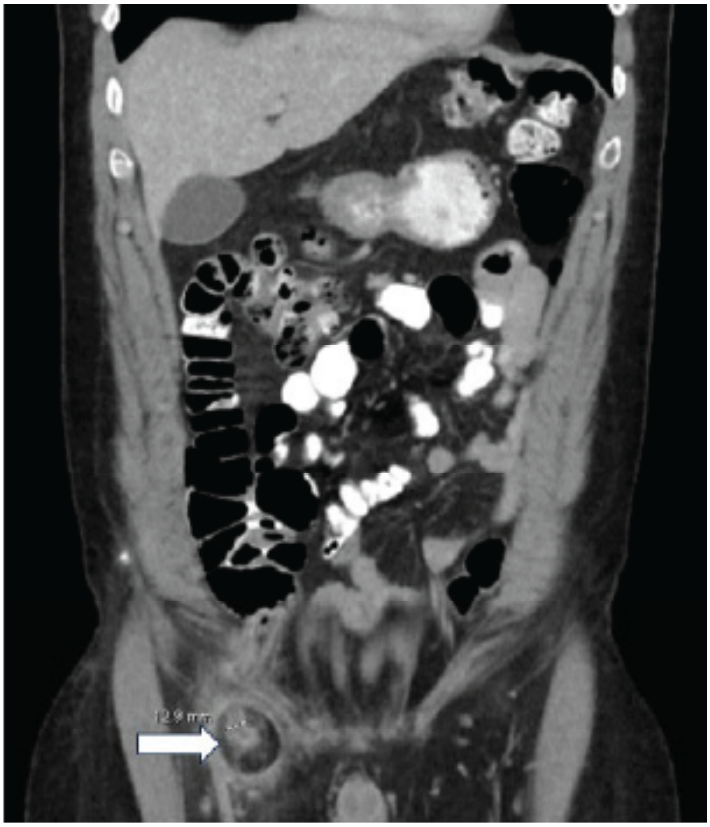


Image 3. Coronal view of a computed tomography of the abdomen pelvis with a dilated appendix at 12.9 millimeters (arrow) and inflammatory changes around the appendix and hernia.

female-to-male ratio of 10:1.[1] Femoral hernias make up only approximately 5% of all groin hernias with the incidence of De Garengeot hernias being less than 5% of all femoral hernias. Consequently, incarceration and strangulation of the appendix within a De Garengeot hernia with concurrent acute appendicitis is an exceedingly rare complication.[2,3] Here we not only present an uncommon case of femoral hernia in a male patient but a unique case of a De Garengeot hernia with the presence of acute gangrenous appendicitis. While De Garengeot hernias have been noted in previous literature, very few cases have documented radiographic and postoperative inconsistencies in diagnosis of this hernia.

Based on CT and physical exam, an Amyand hernia, a protrusion of the appendix into an inguinal rather than a femoral hernia, was the initial clinical suspicion.[5] Amyand hernias are more common than De Garengeot hernias, representing 0.4-1% of all inguinal hernias with 0.1% of cases being complicated by acute appendicitis.[6,7] Although both Amyand and De Garengeot hernias may present as abdominal protrusions with erythema in the inguinal area, this largely remains a non-specific differentiating finding. Even with the incorporation of modern imaging techniques, CT has only been shown to be diagnostic of De Garengeot hernia 44% of the time.[4] Given the radiographic challenges in distinguishing inguinal from femoral hernias, preoperative diagnoses of De Garengeot hernias poses diagnostic challenges and uncertainty to the emergency physician where final diagnosis is often only made by chance in the OR.[4]

Although the clinical presentation of a De Garengeot hernia can be fairly atypical and misdiagnosed as an inguinal hernia, high clinical suspicion of a De Garengeot hernia, especially one with concomitant appendicitis, should prompt urgent surgical consultation and evaluation, as the narrow neck of the femoral canal predisposes femoral hernias to high risk of strangulation and further complications as compared to inguinal hernias.[4,8] De Garengeot hernias should not be reduced in the ED due to the complex nature of the hernia and its risk for appendicitis.[4] Emergency physicians should be aware that if left untreated, the inflammation may progress to life-threatening complications, which include necrosis of the hernia contents, bowel obstruction, necrotizing fasciitis, and abscess formation.[4] Imaging studies can aid in the diagnosis and help guide clinical decision-making, particularly when considering the possibility of incarceration, strangulation, or concurrent acute appendicitis. However, clinical presentation is non-specific, and precise knowledge of hernia sac content preoperatively is not mandatory when considering surgical intervention.[8]

As evidenced in this case, the De Garengeot hernia with gangrenous appendicitis was not made until the patient was in the OR where he underwent laparoscopic appendectomy and open, right femoral hernia repair. Notably, preoperative diagnosis of a De Garengeot hernia is associated with a lower complication rate and shorter hospital length of stay than intraoperative diagnosis.[4] Given the lack of efficacy in radiographic methods for diagnosing a De Garengeot hernia, particularly with CT being less than 50% diagnostic, prompt surgical exploration should be pursued if there is high clinical suspicion for an incarcerated or strangulated femoral hernia with abdominal contents.

CONCLUSION

A De Garengeot hernia is defined as a femoral hernia that contains the vermiform appendix. Given its diagnostic challenges, emergency physicians should know that acute gangrenous appendicitis is an uncommon and life-threatening complication of this femoral hernia.

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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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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Successful Treatment of Paradoxical Vocal Cord Motion with Sub-dissociative Dose Ketamine: Case Report

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Introduction: Paradoxical vocal cord motion (PVCM) is a primarily neuropsychiatric condition that causes inappropriate adduction of the vocal cords during respiration. This condition is commonly misdiagnosed and treated as refractory asthma or upper airway obstruction requiring intensive care unit-level of care. Recent expert opinion suggests that ketamine administration may promote PVCM symptom resolution; however, this phenomenon has not yet been documented in the literature.

Case Report: This is the case of a 23-year-old female who presented to the emergency department (ED) with acute PVCM exacerbation. After failing to respond to standard-of-care therapies including benzodiazepines, the patient was administered intravenous, sub-dissociative dose ketamine, which led to symptom resolution and discharge.

Conclusion: Sub-dissociative dose ketamine may be a safe and effective therapy for PVCM exacerbations in the ED. In this report we explore the patient factors that likely mediated the clinical outcome in this case. [Clin Pract Cases Emerg Med. 2025;19(2):169-172.]

Keywords: *paradoxical vocal cord motion; ketamine; case report.*

INTRODUCTION

First documented by clinicians in the late 1800s in the setting of women's "hysteria," paradoxical vocal cord motion (PVCM) describes inappropriate adduction of the vocal cords during the respiratory cycle. Cases of PVCM can be divided into subtypes according to their pathophysiologic drivers. In primary PVCM, neuro-psychological pathology such as depression and anxiety drive symptom exacerbations. In secondary PVCM, neuromedical processes such as post-viral encephalopathy and gastroesophageal reflux trigger PVCM symptoms.[1]

Paradoxical vocal cord motion is commonly misdiagnosed as refractory asthma or upper airway obstruction, leading to inappropriate pharmacotherapy, intubation, and critical care utilization. Based on expert opinion, the authors of a 2017 review suggested that ketamine administration may be helpful in emergency department (ED) management of PVCM.[2] The use of ketamine in ED management of PVCM has not previously been published. Here, we present a case of successful treatment of PVCM in the ED following administration of intravenous sub-dissociative dose ketamine. The antidepressant and synaptogenic effects of ketamine in combination with standard-of-care treatment likely mediated symptom resolution in this case.

CASE REPORT

A 23-year-old female presented to the ED with a chief complaint of wheezing. Her past medical history was significant for PVCM, prior pulmonary embolism, tracheobronchomalacia, gastroesophageal reflux disease (GERD), depression, anxiety, and long-term sequelae of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection. She endorsed central chest pain without pleurisy and denied recent illness, sick symptoms, and tobacco or vape use. The patient expressed that her current symptoms felt similar to prior PVCM exacerbations.

Six months prior to presentation, the patient was diagnosed with PVCM in the setting of SARS-CoV-2 infection. Since then, she had experienced worsening depression and anxiety and had frequent presentations to the ED with subjective respiratory distress resulting in multiple admissions to the intensive care unit (ICU). She was followed in the outpatient setting by an otolaryngologist and previously received botulinum toxin vocal cord injections with mild improvement in symptomatology. She was also in the process of seeking care from a gastroenterologist for severe gastroesophageal reflux disease.

On exam, the patient was afebrile and tachycardic to 106 beats per minute with a blood pressure of 140/90 millimeters of mercury. Her respiratory rate was 18 breaths per minute with an oxygen saturation of 98% on room air. The patient's lungs were clear to auscultation. Intermittent grunting sounds were present on laryngeal auscultation, and the oropharynx was clear and without pharyngeal swelling. Despite her forced exhalation, the patient was able to speak in complete sentences and had normal work of breathing.

Laboratory evaluation was notable for a mildly elevated leukocyte count of 12.6 x 10^3 cells per microliter (μL) (reference range: 4.0-11 x 10^3 cells/μL). Other lab values, including a D-dimer, two high-sensitivity troponin levels, and a basic metabolic panel were within normal limits. A pregnancy test and a 22-pathogen respiratory viral panel were negative. Chest radiograph was without aberration, and a 12-lead electrocardiogram showed sinus tachycardia without evidence of acute ischemia. A diagnosis of PVCM exacerbation was made and nasopharyngoscopy deferred.

The patient was encouraged to breathe deeply and received 1 milligram (mg) of intravenous (IV) lorazepam without improvement. Given lack of response, the patient received a second dose of lorazepam, IV pantoprazole 40 mg, and oral solution containing 1% lidocaine, aluminum hydroxide, and magnesium hydroxide. Her symptoms persisted, after which IV ketamine 0.15 mg per kilogram diluted in 400 milliliters (mL) of normal saline was administered over 15 minutes. On patient re-evaluation 10 minutes after the initiation of ketamine infusion, mild clinical improvement was appreciated, with a decrease in severity of patient distress. On patient re-evaluation 30 minutes after the initiation of ketamine infusion, clinical resolution of PVCM exacerbation was noted. Ninety minutes following ketamine infusion, the patient expressed symptom resolution and the desire to be discharged to ED staff. She was observed for 120 minutes from the time of ketamine administration, demonstrating the ability to safely ambulate and a return to mental status baseline at which time she was discharged. She was counseled to follow up with her otolaryngologist, psychiatrist, and gastroenterologist and to return to the ED if she developed respiratory distress.

CPC-EM Capsule

What do we already know about this clinical entity?
Paradoxical vocal cord motion (PVCM) is primarily driven by psychologic distress, although anatomic, neurologic, and medical factors may also contribute.

What makes this presentation of disease reportable?
This is the first reported case of emergency department (ED) management of PVCM using sub-dissociative dose ketamine.

What is the major learning point?
PVCM may be well controlled with sub-dissociative dose ketamine in the ED, particularly when symptoms are refractory to standard treatment.

How might this improve emergency medicine practice?
We were able to discharge the patient following ketamine administration, preventing misuse of critical care resources and limiting inappropriate medical therapy.

DISCUSSION

Here, we present the first case report on the successful treatment of PVCM using sub-dissociative dose ketamine. This condition can be divided into categories based on underlying pathophysiology (Figure).

Primary PVCM, which represents 75% of cases, is driven by neuropsychological pathology such as depression and post-traumatic stress disorder.[3] Exacerbations are frequently precipitated by an increase in acute life stressors, and while many cases are attributed to airway obstruction, the majority of patients presenting to the ED with PVCM do not have respiratory failure.[2] Secondary PVCM, which constitutes 25% of cases, results from a neurologic, respiratory, gastroesophageal, or other medical process.[3]

The patient presented here was likely experiencing a PVCM attack of mixed etiology. She had previously been diagnosed with depression and anxiety, esophageal reflux, and tracheobronchomalacia, all of which may contribute to the development of PVCM symptoms. Despite her history, her symptoms were refractory to topical anesthesia, acid suppression, and anxiolysis. Recent neurologic injury from infection with SARS-CoV-2 may have contributed to her symptom severity as well, as coronaviruses are well known to enter the cerebrospinal fluid, cause damage to brain tissue, and may precipitate or exacerbate psychiatric and functional neurological pathology.[4,5] Both the patient and her family observed that her depression, anxiety, and PVCM symptoms worsened following her SARS-CoV-2 infection.

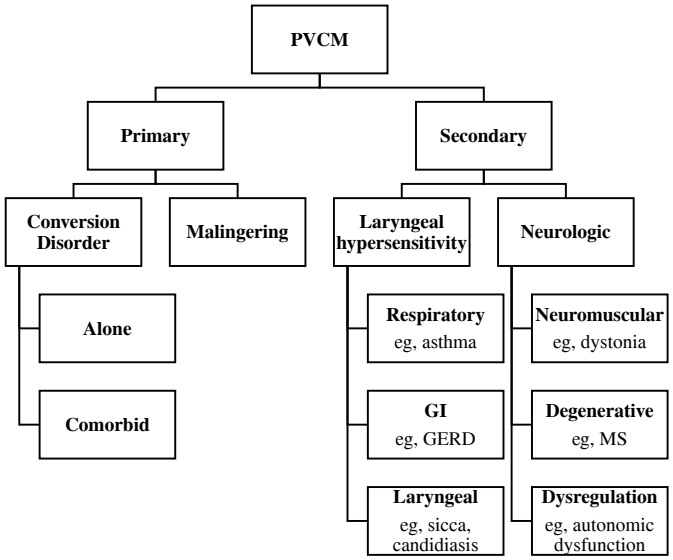


Figure. Subtypes of paradoxical vocal cord motion divided by primary driver.
PVCM, paradoxical vocal cord motion; *GI,* gastrointestinal; *GERD,* gastroesophageal reflux disease; *MS,* multiple sclerosis.

Depression, anxiety, and possibly post-viral neurologic injury were the likely acute and semi-acute drivers of PVCM in this case, with congenital tracheobronchomalacia likely conferring a baseline anatomic risk as well. A variety of therapies have previously been shown to be effective in decreasing acute PVCM symptomatology, including benzodiazepines and, in one case report, haloperidol.[6,7] Symptoms may respond to acid suppression, upright positioning, and topical lidocaine, especially when tracheobronchomalacia is present. Heliox, a mixture of helium and oxygen, and non-invasive positive pressure ventilation may decrease symptoms by promoting laminar, unobstructed airflow.[2,8] Botulinum toxin injection and superior laryngeal nerve block are attempted by otolaryngologists in refractory cases, and the most severe cases are managed with tracheostomy.[2] However, unless patients experience symptom resolution and stability following a short treatment course, many of these strategies are insufficient to promote hospital discharge from the ED. In the case presented, treatment with benzodiazepines was attempted twice without symptom resolution. In addition, treatment for acid reflux and laryngeal irritation were ineffective. Intravenous sub-dissociative dose ketamine produced timely relief of the patient's symptoms and allowed her to be discharged home.

Ketamine is a rapid acting antidepressant observed to be clinically effective within hours of administration, which increases synaptogenesis and neuronal growth factor release.[9] Ketamine's neuroprotective mechanism of action likely explains its clinical efficacy relative to benzodiazepines in this case. Benzodiazepines, while anxiolytic, are not antidepressants and do not promote neuroplasticity. In this patient with depression and possible recent neurologic injury from SARS-CoV-2 infection, ketamine likely offered the greater therapeutic benefit. Laryngospasm may rarely occur with ketamine administration, and this phenomenon is correlated with high-dose IV administration.[11] Sub-dissociative dosing is important to minimize possible iatrogenic harm in this population.

Paradoxical vocal cord motion is associated with risk of misdiagnosis, iatrogenic harm, and inappropriate allocation of medical resources.[1] Use of asthma medication in this group has been shown to be as high as 85%, and patients with primary PVCM account for approximately 10% of patients at specialized centers seeking treatment for refractory asthma.[2,11] In addition, patients with PVCM may be admitted to the ICU for airway monitoring, resulting in high cost/low value use of critical care resources.[1] In one study, as many as 28% of patients presenting to the ED with PVCM were subject to inappropriate intubation.[12] Early identification of PVCM exacerbations vs asthma/other respiratory dysfunction in an emergency setting and management with ketamine could limit iatrogenic harm, systems waste, and improve patient outcomes.

CONCLUSION

A young woman presented to the ED with a chief complaint of wheezing and was diagnosed with exacerbation of paradoxical vocal cord motion. We demonstrate the novel management of a case of PVCM using sub-dissociative dose ketamine allowing for discharge from the ED. Ketamine's antidepressant neuroprotective effects likely mediated successful treatment of PVCM symptoms in the case presented. Ketamine may be valuable in achieving symptom resolution and discharge of patients with acute PVCM exacerbation presenting to the emergency department.

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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.

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Unraveling an Enigmatic Triad: A Case Report of Concurrent Neurosyphilis, Ocular Syphilis, and Otosyphilis in a Patient with HIV

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Introduction: Patients with HIV disease, regardless of the phase of infection, can present with overlapping stages and less distinct signs of syphilis, complicating diagnosis and treatment. *Treponema pallidum*, the bacterium responsible for syphilis, can lead to neurosyphilis, ocular syphilis, and otosyphilis when left untreated. Therefore, early detection of syphilis coinfection in HIV patients and timely treatment have demonstrated prompt improvement of symptoms, mitigating the risk of serious complications.

Case Report: We report the case of a 39-year-old previously incarcerated male with a significant history of HIV on antiretroviral therapy and previous methamphetamine abuse referred to the emergency department from an ophthalmologist with a diagnosis of anterior uveitis and papilledema. The patient reported experiencing blurry vision, tinnitus, and memory difficulties. A thorough history and physical examination, along with diagnostic procedures, including lumbar puncture and cerebrospinal fluid analysis, corroborated the diagnosis of neurosyphilis with otic and ocular involvement. The patient underwent a 14-day course of intravenous aqueous crystalline penicillin G, resulting in symptom improvement.

Conclusion: Given the prevalence of syphilis and its diverse manifestations, clinicians must maintain a high index of suspicion in patients who engage in high-risk behaviors to facilitate early diagnosis and treatment, which are crucial for optimal outcomes and enhanced prognosis. [Clin Pract Cases Emerg Med. 2025;19(2):173-177.]

Keywords: *case report; HIV; neurosyphilis; ocular syphilis; otosyphilis.*

INTRODUCTION

Patients with HIV, irrespective of the stage of infection, can present with atypical features and potentially overlapping stages of syphilis co-infection. Once exposed, the primary stage of syphilis emerges typically between 9-90 days with the hallmark syphilitic chancre and other primary lesions in the genitalia.[1] Secondary syphilis develops subsequently over the next months, often marked by a multitude of symptoms including mucocutaneous eruptions, generalized lymphadenopathy, condyloma lata, and nephrotic syndrome.[2] Latent syphilis is a phase of the disease characterized by a lack of overt symptoms, which presents a diagnostic challenge due to the absence of typical clinical manifestations associated with secondary syphilis. The latent stage can be further subdivided into early and late latency, with early latency marked by subtle signs and symptoms that may easily be overlooked, while late latency is defined by the complete absence of primary and secondary symptoms. Although rare, tertiary syphilis may occur years to decades after infection and is associated with potentially devastating complications, such as aortitis, coronary artery disease, and late neurological issues.[3] Given the spirochete's ability to rapidly invade multiple tissues in the early stages of syphilis, it is crucial to maintain a high level of suspicion for neurosyphilis in immunocompromised patients.

CASE REPORT

A 39-year-old male with a history of HIV, prediabetes, and umbilical hernia presented to the emergency department (ED) complaining of bilateral, non-painful, non-pruritic eye redness with mild tearing, headache, and tinnitus with decreased hearing for three weeks. The patient reported persistent visual disturbances, characterized by haziness in the right visual field and black floaters with a diagonal line in the left visual field. Additionally, the patient's partner reported that the patient began to exhibit signs of confusion and difficulty hearing around the same time. Approximately a week before the ED visit, the patient had presented to an urgent care and was diagnosed with bacterial conjunctivitis, but the prescribed ophthalmic drops failed to alleviate his symptoms. He subsequently visited an ophthalmologist, who reported evidence of papilledema as well as anterior uveitis, and referred the patient to the ED for further evaluation.

The patient disclosed he had recently experienced joint and muscle pain that he attributed to his antiretroviral therapy (ART) regimen, which had prompted him to discontinue his medication for approximately three weeks prior to onset of symptoms. The patient's social history included tobacco and methamphetamine use, previous incarceration, and an HIV diagnosis made during incarceration. Importantly, the patient's ART resulted in a significant improvement in his HIV viral load (from 268,000 per milliliter [mL] down to 30 copies/mL) (reference range: 0 copies/mL) and cluster of differentiation 4 (CD4+) cell count (from 142 per microliter [μL] up to 508 cells/μL) (430-1800 cells/μL) within six months of treatment initiation. The patient reported being in a monogamous relationship with a woman since his release and denied any history of sex with men, intravenous drug use, or recent travel. His primary care physician monitored his health and ART response. After a thorough review, the patient endorsed night sweats, subjective fever, and desquamation of the soles of his feet.

Upon arrival to the ED, his initial vital signs were blood pressure 120/82 millimeters of mercury, heart rate 95 beats per minute, respiratory rate 19 breaths per minute, temperature 97.9 °Fahrenheit, saturation of peripheral oxygen 100% room air, and body mass index 26.39 kilograms per cubic meter (kg/m^3) (18.5-24.9 kg/m^3). Physical examination was remarkable for a male who appeared generally healthy but was experiencing mild visual distress due to recent use of ocular dilating medications at ophthalmologic appointment but no photophobia. There was moderate bilateral conjunctival injection, pale sclerae, and no discharge. Pupils were dilated, equal, round, and reactive with direct and consensual responses. Extraocular movements were intact. Visual acuity testing was 20/50 in the right eye, but he was only able to count fingers with the left eye, reporting severe blurriness. Peripheral visual field testing was intact bilaterally. Cardiac exam was unremarkable with normal rate and rhythm without murmurs. Equal and bilateral lung sounds were appreciated without wheezing, crackles, or rhonchi. There was no nuchal rigidity present. There was mild non-erythematous, non-pruritic, whitish desquamation on the soles of his feet, more on the right. There was no focal weakness in any extremity, and he had a normal gait. Cranial nerves II-XII were grossly intact.

Comprehensive metabolic panel showed normal electrolytes, kidney function, and transaminases with mildly elevated glucose. Serum non-treponemal and fluorescent treponemal antibody testing were reactive, with a subsequent serum reactive quantitative and confirmatory Venereal Disease Research Laboratory (VDRL) with a titer of 1:1024, which resulted during patient's admission. Lumbar puncture was technically challenging but successfully performed in the ED without a measured opening pressure. Cerebrospinal fluid (CSF) was clear in appearance with mild pleocytosis. Cerebrospinal fluid VDRL quantitative and confirmatory tests were reactive, confirming diagnosis. FilmArrayME meningoencephalitis CSF panel was negative. The CSF culture ultimately did not grow any organisms and did not demonstrate presence of malignant cells. Computed

CPC-EM Capsule

What do we already know about this clinical entity?
Prompt recognition and treatment of neurosyphilis, ocular syphilis, and otosyphilis are crucial to prevent complications.

What makes this presentation of disease reportable?
This case of an uncommon triad of syphilis in an HIV patient highlights the need to consider syphilitic infections in complex cases with seemingly unrelated symptoms.

What is the major learning point?
Clinicians should maintain a broad differential diagnosis and multidisciplinary collaboration when assessing HIV patients with complex neurological symptoms.

How might this improve emergency medicine practice?
Recognizing this rare triad of syphilitic infections in the era of a surge in syphilitic infections globally can improve clinical vigilance.

Table. Lab findings of patient hospitalized for treatment of neurosyphilis with ocular and otic involvements.

Blood count	Basic metabolic panel	Liver function test	Microbiology (serum-PCR)	Cerebrospinal fluid
Hemoglobin 12.3 g/dL (Ref: 14-18)	Sodium 138 mEq/L (Ref: 135-145)	ALT 25 U/L (Ref: 11-66)	RPR positive	Color: clear
Hematocrit 39.3% (Ref: 42.0-52.0)	Potassium 4.2 mEq/L (Ref: 3.5-5.2)	AST 28 U/L (Ref: 5-40)	VDRL 1:1024	RBC 11 mm^3 (Ref: 0)
WBC 6.1 thou/mm^3 (Ref: 4.8-10.8)	Chloride 103 mEq/L (Ref: 98-111)	ALP 106 U/L (Ref: 38-126)	HIV-1 RNA 112 cpy/mL (Ref: 0)	Nucleated cells 11/ mm^3 (Ref: 0-5)
Platelets 263 thou/mm^3 (Ref: 130-400)	Bicarbonate 23 mEq/L (Ref: 23-33)	Bilirubin, total 0.3 mg/dL (Ref: 0.3-1.2)	EBV negative	Lymphocytes 93% (Ref: 0-90)
Lactate 1.2 mmol/L (Ref: 0.5-1.9)	BUN 12 mg/dL (Ref: 7-22)	Albumin 3.6 g/dL (Ref: 3.5-5.1)	TB Quant negative	Glucose 65 mg/dL (Ref: 40-80)
Absolute CD4+ 190/µL (Ref: 430-1800)	Creatinine 1.0 mg/dL (Ref: 0.4-1.2)	Protein, total 8.3 g/dL (Ref: 6.1-8.0)	Toxoplasma IgM less than 3.0 AU/mL (Ref: less than 8)	Protein 109 mg/dL (Ref: 12-60)
	Glucose 120 mg/dL (Ref: 70-108)		CMV negative	Lactate 2.3 mmol/L (Ref: 0.5-2.2)

µL, microliter; *U/L,* units per liter; *thou/mm^3,* thousand per cubic millimeters; *mmol,* millimoles; *mg/dL,* milligrams per deciliter; *g,* grams; *mEq,* milliequivalents; *cpy/mL,* copies per milliliter; *ALP,* alkaline phosphatase; *ALT,* alanine aminotransferase; *AST,* aspartate aminotransferase; *AU*, antibody units; *BUN,* blood urea nitrogen; *CD4+,* cluster of differentiation 4; *CMV,* cytomegalovirus; *EBV,* Epstein-Barr virus; *RNA,* ribonucleotide; *IgM,* immunoglobulin M; *PCR,* polymerase chain reaction; *RBC,* red blood cells; *Ref,* reference range; *RPR,* rapid plasma reagin; *TB Quant,* tuberculosis QuantiFERON; *VDRL,* venereal disease research laboratory; *WBC,* white blood count.

tomography of the head was unremarkable for acute findings. Magnetic resonance imaging of the brain, requested by the referring consultant, demonstrated small filling defects within the left transverse sinus.

The patient was admitted for treatment of neurosyphilis with ocular and otic involvements and started on penicillin G and antiretrovirals. The infectious disease specialist recommended penicillin G intravenous (IV) treatment at a dosage of 24 million units per day for 14 days, administered every four hours during hospitalization and a continuous infusion once the patient was discharged. The patient's home dose of bictegravir-emtricitabine-tenofovir (50-200-25 mg per tablet once daily) was continued, and erythromycin ophthalmic ointment was initiated. Magnetic resonance imaging findings were interpreted by the interventional neurologist as either arachnoid granulation or non-obstructive thrombus, neither of which would explain the papilledema. Consequently, a daily, low-dose aspirin with subsequent outpatient follow-up in six months was recommended.

After three days of treatment, the patient reported improvement in his visual symptoms, indicating that the black line and floater were no longer affecting his vision. Additionally, the patient noted a reduction in his tinnitus and reported overall improvement. A follow-up appointment four days after hospitalization revealed that the patient had begun to experience hair loss, but he reported continued improvement in his vision, particularly in the left eye. On day 14, the patient visited his infectious disease physician after completing his IV benzylpenicillin treatment. The patient was advised to continue with antiretroviral therapy and to follow up with his primary care physician and neurologist as scheduled.

DISCUSSION

Treponema pallidum, the causative agent of syphilis, is an adept infiltrator, capable of eluding the body's immune defenses, sequestering itself in a multitude of tissues, and even breaching the blood-brain barrier prior to the onset of initial symptoms.[4] It can be transmitted through intimate physical contact with an individual who harbors a syphilis-related skin lesion, which often arises through sexual intercourse. Although the patient suspected that he acquired the infection through an accidental exposure of fecal-ocular material during plumbing

work prior to the onset of symptoms, this mode of transmission seems unlikely due to the limited survival of *T pallidum* outside the human host. Attempts to culture this organism in vitro have been mostly unsuccessful, with few documented success cultures performed within tissue culture systems.[5]

If untreated, the insidious pathogen can affect multiple systems including cardiovascular, neurological, otic, ocular, and immunological systems. Timely treatment, however, has shown the potential to reverse certain debilitating injuries inflicted by the infection, emphasizing the importance of prompt recognition and intervention.[6] This patient's experience underscores the significance of early detection and treatment in the battle against syphilis, which has been on the rise in the United States since 2001. According to the US Centers for Disease Control and Prevention (CDC), there has been a surge in syphilis infections among specific demographic groups between 2018–2022, with a disproportionate increase of 146.7% in incidence among men who have sex with women, compared to a comparatively lower 6.6% increase among men who have sex with men.[7] These findings highlight the vulnerability of individuals engaging in high-risk sexual behaviors, including those with HIV, emphasizing the need for targeted interventions and tailored public health strategies to curb the spread of syphilis within the population.

To avoid premature closure, papilledema and uveitis reported by the ophthalmologist, in conjunction with the patient's reported symptoms, required further investigation. Differential diagnoses were extensive, including HIV-associated meningitis or encephalitis with pathogens including cytomegalovirus, Epstein-Barr virus, tuberculosis, syphilis, toxoplasmosis, Cryptococcus, herpes simplex virus, aspergillus; cerebral venous sinus thrombosis; lymphoma; HIV-associated encephalopathy; HIV-associated neurocognitive disorder; idiopathic intracranial hypertension; typical and atypical causes of conjunctivitis and uveitis including allergic, bacterial, and viral.

While regular screenings are recommended for high-risk populations,[8] this is often challenging to execute due to a combination of factors, including worsening difficulty in access to primary care with the COVID-19 outbreak, difficulty of notifying partners particularly in an era of online dating and sexual encounters,[9] and insufficient resources to tackle the crisis. Interestingly, the patient's last documented syphilis screening was 19 months prior to this hospital visit, supporting the challenges in screening this vulnerable population.

Laboratory diagnostics of syphilis remain challenging. Treponemal and non-treponemal testing, both implied in traditional and reverse-sequence screening algorithms, are used to aid in the diagnosis of syphilis. Serological treponeme-specific tests such as fluorescent treponemal antibody absorption (FTA-ABS) used to rule out neurosyphilis are reliably reactive in patients with neurosyphilis including late-stage neurosyphilis.[3] Although CSF VDRL is the gold standard for the diagnosis of neurosyphilis, its low sensitivity and high false negative rate of up to 70% makes it an unreliable measure to rule out the disease. Similarly, CSF fluid FTA-ABS test has limitations and may not be a definitive diagnostic tool.

Treatment of syphilis depends on the stage of the disease and the organs involved per CDC guidelines. Early syphilis (primary, secondary, or early latent) is treated with a single dose of penicillin. Late latent or syphilis of unknown duration requires three doses of penicillin. Otosyphilis, ocular syphilis, or neurosyphilis are treated with aqueous crystalline penicillin G 18-24 million units per day, administered as 3-4 million units by IV every four hours, or by continuous infusion, for 10-14 days.[7]

CONCLUSION

A high suspicion of neurosyphilis should be maintained in immunocompromised patients who present with neurological symptoms, irrespective of recent, non-treponemal screening findings. A full workup, using the appropriate screening algorithm, is imperative to rule out this organism in symptomatic patients. Accurate staging and adherence to the treatment recommendations provided by the CDC have demonstrated favorable treatment outcomes and improved prognosis.

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

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A Case Report of a Rare, but Important, Cause of Delerium Presenting to an Emergency Department

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Introduction: Delayed post-hypoxic leukencephalopathy is a rare cause of acute neuropsychiatric decline diagnosable in the emergency department (ED), but it has not been described in the emergency medical literature. We present a case report of a pathognomonic presentation.

Case Report: A 53-year-old man developed akinetic mutism 14 days after being discharged from a hospitalization for fentanyl overdose. Magnetic resonance imaging demonstrated symmetric frontoparietal white matter demyelination.

Conclusion: Delayed post-hypoxic leukencephalopathy can present to the ED as altered mental status days to weeks after apparent full recovery from an initial episode of cerebral hypoxia. This report will help emergency physicians avoid missing this diagnosis. [Clin Pract Cases Emerg Med. 2025;19(2):178-181.]

Keywords: *delayed post-hypoxic leukoencephalopathy; delayed neurologic sequelae; neuroimaging; opiate overdose; opioid overdose.*

INTRODUCTION

Delayed post-hypoxic leukencephalopathy (DPHL) is a demyelinating brain disorder that causes an abrupt onset of neuropsychiatric dysfunction (either acute onset of parkinsonism or akinetic mutism) days or weeks after apparent full recovery from coma following an episode of profound cerebral hypo-oxygenation. This biphasic presentation distinguishes DPHL from acute anoxic encephalopathy.[1] The lucid interval (median 22 days) makes the emergency department (ED) a likely venue for presentation.[2] Characteristic findings on magnetic resonance imaging (MRI) aid in diagnosis. The increasing use of MRI in EDs will give emergency physicians (EP) increasing opportunities to make (or to miss) the diagnosis of DPHL.[3,4] We present a case report of a patient presenting with acute akinetic mutism three weeks after appearing to fully recover from a fentanyl overdose. His presentation demonstrates characteristic historical, physical, and imaging features of DPHL.

CASE REPORT

A 53-year-old man with no known neurologic or psychiatric history was brought to the ED after being found unresponsive after accidental fentanyl overdose. Emergency medical services initially noted apnea and percentage oxygen saturation in the 40s. After administration of naloxone and oxygen he was observed to be moving all four extremities but remained unresponsive to verbal stimuli in the ED; so, he was intubated for airway protection. Fever and hypotension prompted initiation of sepsis care. Head computed tomography (CT) showed no acute findings. A urine toxicology panel was positive for fentanyl, amphetamines, and benzodiazepines, but negative for opiates. Over the following day his vital signs stabilized, and on day three an electroencephalogram (EEG) showed no signs of epileptiform activity. He tolerated extubation and had no focal neurologic deficits. Seven days after admission he was discharged to home and resumed work.

Nineteen days after the anoxic event, family members found the patient "spaced out and drowsy" and brought him to

the ED. He had normal vital signs, but speech was very slow, nearly mute, and he answered orientation questions with "I am here." Movement was markedly slow. He would squeeze fingers placed in his palms, but he did not follow any complex commands and was incontinent of urine. Illicit drug toxicity, hypercarbia, electrolyte abnormalities, traumatic brain injury, and meningitis were all considered and were evaluated with the following studies: toxicology screening was negative, venous blood gas and chemistry profiles were normal, CT brain showed signal hyperintensity in the basal ganglia but no other acute finding, and lumbar puncture yielded clear fluid with normal cell counts and no organisms or viruses detected. An MRI (Image 1) showed hyperintensity of basal ganglia and hyperintensity of white matter tracks. This raised concern for an autoimmune encephalopathy and for opioid-related conditions that cause encephalopathy persisting long after the offending drug has been metabolized: DPHL and opioid-induced leukencephalopathy (OIL).

During inpatient care methylprednisolone 1gram was administered daily for potential autoimmune process, and plasmapheresis was initiated. He later became tremulous and diaphoretic with tachycardia, hypertension, and muscle stiffening and was intubated for airway protection and given valproate for possible seizure disorder. Subsequent EEG showed no ictal changes yet did show diffuse delta slowing consistent with encephalopathy, but not seizure. We considered neuroleptic malignant syndrome (NMS) and serotonin syndrome (SS), as well as tetanus and strychnine poisoning as causes of this change. The patient had received haloperidol early in his course, although only 2 milligrams total. As he was without fever at that time, and he had no clonus or hypereflexia, NMS and SS were unlikely. Additionally, his neurocognitive symptoms continued for several days after resolution of this acute change and without the administration of any further serotonergic or anti-dopaminergic agents, ruling out NMS and SS as causes of his ongoing inability to follow commands. Strychnine poisoning and tetanus cause muscular hyperactivity in awake patients, but our patient had depressed mental status throughout his hospital stay. While his hypertonicity began several days into his hospital course and resolved within minutes, strychnine poisoning is rapid in onset, and tetanus causes more prolonged symptoms.

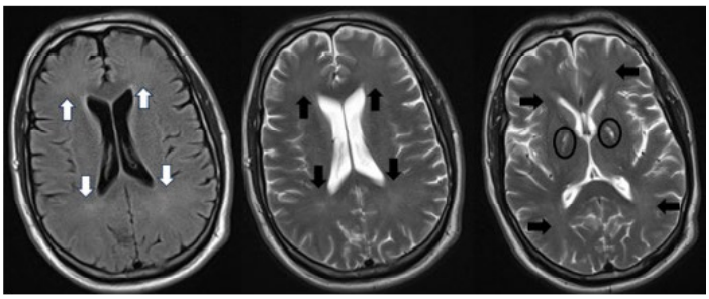


Image 1. Magnetic resonance imaging at onset of neuropsychiatric decline (19 days after initial visit) showing symmetric subtle hyperintensity in frontal and parietal lobes (axial FLAIR sequence on far left with white arrows, axial T2 sequence in middle and far right with black arrows) and hyperintensity of the bilateral globi palidi (ovals) with normal-appearing gray matter.

A repeat MRI (Image 2) showed progression of symmetric hyperintensity in frontal and parietal lobes and bilateral globi palidi hyperintensity otherwise sparing the gray matter; posterior circulation areas were spared.

We considered posterior reversible encephalopathy syndrome (PRESS); however, his blood pressure was within normal limits upon presentation of encephalopathy, and his condition did not improve with supportive care. Additionally, while PRESS tends to affect the cortical regions and predominates in the posterior areas of the brain, our patient's abnormal MRI findings were seen in the subcortical regions of the frontal and parietal areas.

Both OIL and DPHL cause mutism, incontinence, confusion, and extrapyramidal symptoms. And both cause

CPC-EM Capsule

What do we already know about this clinical entity?
Cerebral hypoxia can cause lasting neurologic sequelae, but emergency physicians (EP) may be unaware of delayed post-hypoxic leukencephalopathy (DPHL).

What makes this presentation of disease reportable?
This demonstrative emergency department (ED) case of DPHL elucidates this lesser-known cause of acutely altered mental status.

What is the major learning point?
When a patient presents with new-onset parkinsonism or akinetic mutism days to weeks after recovering from a profound hypoxic episode, DPHL should be considered.

How might this improve emergency medicine practice?
This case report will help EPs recognize DPHL, a condition that is likely to become increasingly diagnosable in the ED.

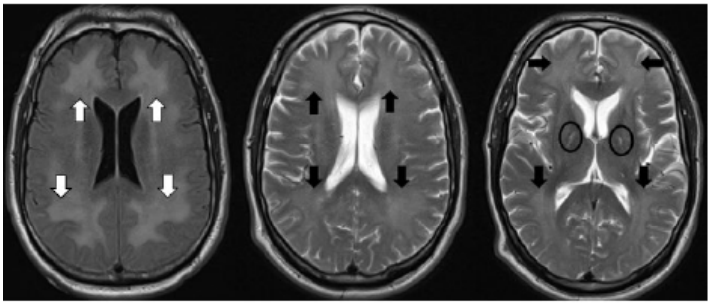


Image 2. Magnetic resonance imaging two days after onset of neuropsychiatric decline (20 days after initial visit) showing more apparent symmetric hyperintensity in frontal and parietal lobes (axial FLAIR sequence on far left with white arrows, axial T2 sequence in middle and far right with black arrows) and hyperintensity of the bilateral globi palidi (ovals) with otherwise normal-appearing gray matter.

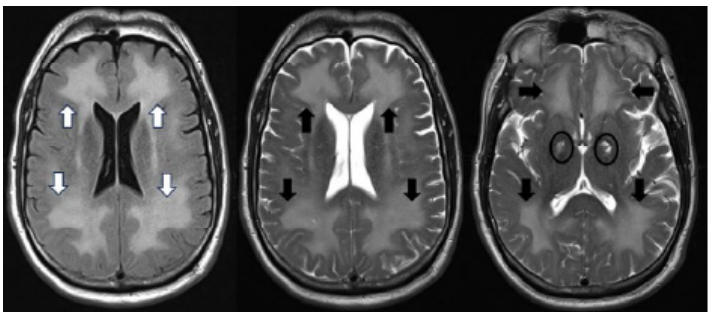


Image 3. Magnetic resonance imaging 20 days after onset of neuropsychiatric decline (30 days after initial visit) showing marked progression of symmetric hyperintensity in frontal and parietal lobes (axial FLAIR sequence on far left with white arrows, axial T2 sequence in middle and far right with black arrows) and hyperintensity of the bilateral globi palidi (ovals) with normal-appearing gray matter.

symmetric changes that predominantly affect white rather than gray matter, suggesting that myelinotoxicity is a unifying feature. In our case the biphasic presentation distinguished DPHL from OIL, in which a lucid interval after the inciting hypoxic event is not observed.[5] Thirty days after his hypoxic episode an MRI (Image 3) demonstrated marked symmetric white matter signal hyperintensity in the frontal and parietal lobes with necrosis of the medial globi pallidi.

It was at this time that we considered the diagnosis DPHL to be conclusive and, given his progressive symptoms, the likelihood of recovery to be extremely low. Palliative care met with the family, and after discussions of prognosis and the patient's known values his care we transitioned to comfort-focused care. After extubation he had spontaneous eye-opening during which time he appeared to stare at nothing and tracked neither examiners nor family members. He did not speak, follow commands, or eat when offered food. He died peacefully four days later.

DISCUSSION

Both OIL and DPHL cause abrupt neuropsychiatric decline and appear to be due to myelin disruption. Opioid-induced leukencephalopathy has commonly been described after inhalation of vaporized heroin ("chasing the dragon") but has also been described after other methods of heroin use and after use of oxycodone. In DPHL an acute neuropsychiatric decline follows an apparent full recovery from a period of profound cerebral hypoxia. It was first described in 1936 as a sequelae of carbon monoxide (CO) toxicity and has been observed after cardiac arrest, asphyxia, drug overdose, and strangulation.[6-8] Common to all causes of DPHL is a period of profound cerebral anoxia. Delayed post-hypoxic leukencephalopathy and OIL have significant clinical overlap, but there are some features to differentiate the two. As mentioned above, DPHL is characteristically biphasic while OIL is not. Whereas the neuroimaging findings in DPHL typically involve the white matter of the frontal and parietal regions, those in OIL most commonly affect the cerebellum, the posterior cerebrum, and the posterior limbs of the internal capsule.[9]

The decline in DPHL is characterized by symptoms that fit one or both of two categories: parkinsonism and akinetic mutism.[10] Common parkinsonian symptoms are rigidity, tremor, and hallucinations. Case reports are mixed on the role of levodopa to treat DPHL-related parkinsonism; however, caution around use of antidopaminergic agents seems prudent, as these could worsen symptoms. Akinetic mutism symptoms include apathy, staring, markedly slowed speech, and functional incontinence. Presentations can overlap with both types, and common physical exam findings include frontal release signs, upper motor neuron signs, and primitive responses to noxious stimuli. This is consistent with our patient's grasping reflexes, inanition, and periods of increased tone. Several features are suggestive that myelinotoxicity suffered during the hypoxic event leads to DPHL: The cerebral white matter is affected, while gray matter is spared, and elevation of myelin basic protein in cerebral spinal fluid is strongly associated with DPHL.[11] The delayed onset of symptoms correlates with the pattern of myelin secretion occurring on average every 20 days.[12]

In most cases, CT will not be diagnostic, and MRI will be required for diagnosis. The following findings are pathognomonic: cerebral white matter hyperintensities seen on T2-weighted images that symmetrically affect the dorsal frontal and parietal lobes while sparing the cortex, cerebellum, and brainstem.[13] On diffusion-weighted sequences a diffusion restriction involving the globi pallidi, which are traversed by numerous myelinated axons, can show high diffusion restriction, indicating cytotoxic edema.[14] All these features were present in our case. These findings help distinguish

DPHL from other diagnoses in the differential.

Steroids and plasmapheresis have been attempted for treatment of DPHL but have not been reported to be of benefit. Zolipidem has been reported to transiently improve alertness and orientation in patients after anoxic brain injury who are no longer comatose.[15] We did not observe any improvement when we administered it to our patient. Care for DPHL is focused on support and rehabilitation if the patient can participate. Among patients with CO-associated DPHL the likelihood of recovery has an inverse relationship to age at onset. The majority of those who survived hospitalization recovered over three to six (median four) months and suffered continued difficulties related to frontal lobe function.[9] For patients who progress to coma during the second phase, recovery from DPHL has not been reported.

CONCLUSION

Delayed post-hypoxic leukencephalopathy is a cause of altered mental status that EPs should consider in patients who present with acute neuropsychiatric decline days to weeks after recovering from a severe episode of cerebral hypoxia. Characteristic symptoms are new-onset parkinsonism or akinetic mutism. If an MRI is performed, specifically look at the T2 sequences for the pathognomonic findings of symmetric, white matter hyperintensity involving the anterior portion of the cerebrum, while sparing the areas supplied by posterior circulation.

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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Physician-Assisted Dying Witnessed by Emergency Medical Services: A Case Report

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Introduction: Physician-assisted dying (PAD) is a practice that allows terminally ill patients to self-administer prescribed lethal medication. In the 11 states in the United States where PAD is legal, the incidence of PAD cases is rapidly rising. Despite most of these cases occurring in the out-of-hospital setting, states lack specific emergency medical services (EMS) protocols to guide prehospital responders who may encounter PAD in the field. We report a case in which a patient called 9-1-1 for a medical emergency and requested to ingest her prescribed lethal medication while in EMS care.

Case Presentation: Emergency medical services was dispatched for a 56-year-old female bleeding from her tracheostomy stoma. Despite the EMS responders' recommendation, the patient refused transport and instead requested to ingest her PAD medication. The crew, unfamiliar with PAD laws, were unsure whether they could legally honor the patient's refusal. Clinicians consulted with online medical control, who were also unaware of PAD. After extensive deliberation, the crew decided to honor the patient's refusal and thoroughly document the situation. The patient self-administered her medication as EMS cleared the scene.

Conclusion: This case highlights logistical challenges and ethical dilemmas faced by EMS responders and underscores the complexity of balancing patient autonomy with legal and medical responsibilities in prehospital situations involving PAD. As PAD becomes increasingly prevalent, equipping EMS responders with clear protocols and providing ongoing education about prehospital PAD cases are vital for preserving patient rights while protecting the responders from legal and ethical uncertainty. [Clin Pract Cases Emerg Med. 2025;19(2):182-187.]

Keywords: *physician-assisted dying; emergency medical services; refusal of care; protocols; case report.*

INTRODUCTION

Physician-assisted dying (PAD)—also referred to as medical aid in dying (MAiD), physician-assisted suicide, and death with dignity—is a medical practice involving the prescription of lethal medication to a requesting patient who intends to end their life.[1] It is important to distinguish PAD

from euthanasia, which is illegal in the United States. Euthanasia refers to a physician directly administering lethal medication to a patient, whereas PAD refers to a prescription of lethal medication to be voluntarily self-administered by a patient.[2] Since the legalization of PAD in Oregon in 1997, the practice has become increasingly sanctioned across the US.[3] Currently, 20% of US residents live in the 11 states where PAD services are legal, and an additional 12.5% live in the five states where PAD is on the legislative agenda.[4] Since its initial legalization in 1997, the incidence of PAD cases across participating states has rapidly increased each year.[5]

While existing PAD literature largely focuses on PAD in the emergency department setting, a vast majority of PAD deaths occur in out-of-hospital settings, such as the patient's home or other hospice and comfort-care environments.[5] In cases where the dying process is interrupted by incomplete ingestion of medication, unanticipated medical emergencies, or other variables, emergency medical services (EMS) may be called to assist. Currently, these cases are seemingly few. In Oregon, which has the most longitudinal and comprehensive PAD data to date, EMS is involved in fewer than 1% of PAD deaths every year.[6] Consequently, there is a paucity of literature surrounding the involvement of EMS responders in prehospital PAD situations. Although EMS involvement may be rare, as the incidence of PAD cases rises the frequency of EMS encounters with PAD will likely increase.

Situations involving the self-administration of lethal medication can occur in diverse settings and in circumstances that can complicate EMS responsibilities on scene. To guide first responders in PAD situations, which often require quick decision-making with limited information and heightened emotions from patients and bystanders, clear protocols are imperative. As is described in the following case report, prehospital PAD cases pose unique ethical and legal challenges to EMS responders that should be considered and integrated into EMS protocols and training.

CPC-EM Capsule

What do we already know about this clinical entity?
Physician-assisted dying (PAD), a legal medical practice in 11 states, enables terminally ill patients to end their lives with prescribed lethal medication.

What makes this presentation of disease reportable?
Patients enrolled in PAD programs have official documentation verifying their enrollment and may have do-not-resuscitate orders and/or hospice paperwork.

What is the major learning point?
To protect emergency medical service responders from legal and ethical uncertainty, agencies must equip them with clear protocols and education about PAD practices.

How might this improve emergency medicine practice?
By knowing how to navigate PAD in the field, first responders can better manage complex scenarios, including refusal of care and complications such as failed ingestion.

CASE REPORT

A Basic Life Support and Advanced Life Support crew was dispatched with township police to a residence for the report of a 56-year-old female hemorrhaging from a surgical site. Upon EMS arrival, the patient was found lying supine on a bathroom floor. Family members present on scene reported they were changing the dressings around the patient's tracheostomy stoma when the site began to bleed. Bleeding was controlled using gauze prior to EMS arrival; the first responders estimated 500 milliliters of blood loss. The crew ensured a patent airway and completed an initial assessment. The patient was fully alert and oriented. She had a history of stage IV laryngeal cancer and communicated nonverbally using a notepad. Initial vital signs were pulse rate of 125 beats per minute, respiratory rate of 16 breaths per minute, and an oxygen saturation of 94%. A blood pressure reading could not be obtained due to limited access to the patient's upper extremities. Given the patient's blood loss and potential for further hemorrhage, urgent transport to the emergency department was recommended. However, the patient indicated that she did not want to be transported and wrote on the notepad: "I want MAiD now." The family members presented the crew with MAiD paperwork, hospice care paperwork, and a Physician Orders for Life-Sustaining Treatment (POLST) form that indicated the patient had a do-not-resuscitate (DNR) order. While familiar with hospice care and POLST, none of the EMS professionals or law enforcement officers had previously heard of MAiD.

Although the patient was alert and oriented, and understood the severity of her illness, the EMS crew questioned whether they could complete a refusal of care if the patient intended to end her life. Despite the crew's recommendation, she remained firm in her decision to not want to be transported and to ingest her medication instead.

On-line medical control (OLMC) was contacted for guidance. The OLMC physician indicated that they were also unaware of MAiD but directed the on-scene EMS crew to continue following their refusal protocols. During this time, the family members were actively preparing the patient's MAiD medication while the EMS responders continued to be uncertain about whether it was appropriate for the patient to proceed with medication ingestion.

Law enforcement officers, who were concerned about foul play and potential legal wrongdoing, requested that their supervisor respond to the scene and ensured that their body cameras were recording. Seeking further clarity, EMS requested to speak via telephone to a representative from the medical practice that had prescribed the patient's lethal medication. The representative, an advanced practice provider, explained the legal basis for MAiD and clarified that the patient was able to ingest the medication at home at any time of her choosing. After nearly an hour of discussion with supervisors, the representative from the prescribing medical practice, and family members, the EMS crew decided to document the patient's refusal of care and obtain witness signatures from law enforcement and the family. Then the family members, holding a pharmacy bag of compounded medication and a glass of water with sugar, entered the bathroom and closed the door. The EMS crew cleared the scene and returned to service while law enforcement remained on scene to complete their report and await the arrival of a hospice nurse who could pronounce the patient deceased.

DISCUSSION

This case illustrates the complexities of PAD situations in the setting of prehospital emergency care. Despite familiarity with hospice and POLST protocols, the EMS crew's unfamiliarity with PAD led to significant confusion on how to proceed. Given the knowledge that the patient intended to end her life, a key challenge for the crew was in deciding whether to allow the patient to ingest her PAD medication while in their care or document the refusal of care and clear the scene prior to medication ingestion. Another challenge was in deciding whether the patient even met criteria for refusal of care. The EMS crew was uncertain whether the patient's plan for PAD could be interpreted as suicidal ideation, which would disqualify her from refusing care.

Medical psychologists and proponents of PAD argue that suicide and PAD are distinct.[7-9] While suicide is associated with individuals experiencing psychological suffering or emotional distress who wish to end their lives, PAD specifically involves terminally ill patients who, after thorough consultation and contemplation, make a medically sanctioned decision to end their lives. This distinction implies that intending to end one's life by means of PAD may not necessarily prevent a patient from refusing care. First responders of all levels receive foundational education on ethical principles, including patient autonomy, informed consent, and handling of advanced directives. However, in complex or unclear situations, as with PAD, EMS first responders should not be left to navigate decisions without clear protocol guidance.

In situations where the decision-making process is protracted by an absence of defined protocols, clear communication between all parties and involvement of additional resources is paramount. Delays in responders' actions stemming from uncertainty on how to proceed can provoke frustration from the patient and their family, who may perceive them as obstacles to the patient's final wishes. Additionally, EMS responders should acknowledge gaps in their familiarity with PAD and be prepared to seek additional guidance from supervising staff or OLMC. In this case, where supervisors and OLMC were also unfamiliar with PAD, additional guidance was sought by contacting the prescribing medical practice. This step clarified the legal basis for the patient's request, helping the responders navigate the situation.

In contrast to this case, there could be instances in which EMS is requested for complications that arise from a patient's ingestion of PAD medication. Commonly reported complications include difficulty swallowing, regurgitation, seizures, and regaining consciousness after ingestion.[10,11] Additionally, delays in death due to prolonged time to effect of lethal medication can lead to patient and family distress, prompting them to contact EMS. In the US, the current medication regimen for PAD commonly includes a combination of digoxin, diazepam, morphine, amitriptyline and, in some cases, phenobarbital.[11,12] Variations in medications, dosages, and individual patient metabolism may contribute to the wide variability in the time from ingestion to death, which can range from minutes to over 100 hours.[6,10] All EMS responders who might encounter PAD patients should be familiar with the medications commonly prescribed, their mechanism of action, and the wide variability in their time to effect.

The ingestion of lethal medication not only introduces medical complexities but also raises situational challenges. Responders may have difficulty differentiating a patient's intent of PAD from suicide, particularly if the patient ingested their lethal medication alone or without informing others. This can be further complicated when PAD documentation, often unfamiliar to EMS, is present, but no DNR or POLST forms are available. Given a lack of clear documentation, EMS responders may be forced to resuscitate the patient against their wishes. In PAD-participating states, there is minimal regulation surrounding the ingestion of PAD medication at home: healthcare clinicians do not need to be present, and there is no requirement either for informing family members or for an accompanying DNR order.[13] Thus, EMS responders

need to be well-informed and properly equipped with standardized protocols to navigate uncertain situations and ensure consistency in care.

None of the 11 states where PAD is currently legalized include provisions in their legislation regarding the possibility that a PAD patient may require care from EMS. While one state, Maine, mentions PAD in its EMS protocols, none provide specific guidelines for EMS management of such cases, and few discuss the management of patients with terminal illness (Table).

With the rising incidence of PAD nationwide, the lack of PAD-specific guidance in state EMS protocols highlights a significant gap in emergency response preparedness. Emergency medical services responders should be able to rely upon guidelines that outline procedures for proper identification of PAD intent, refusal of care, withholding of resuscitation, and continuity of care. Accordingly, we present a sample EMS protocol that outlines basic procedures for clinicians when responding to a call involving PAD (Figure). While this protocol was independently developed, it expands upon a sample EMS protocol published in 2007 in the Oregon Death with Dignity Act Guidebook for Health Care Professionals.[14] We encourage state EMS programs and regulatory bodies to consider adopting similar PAD protocols within their practice guidelines.

CONCLUSION

This case report outlines relevant logistical challenges and ethical considerations for prehospital professionals who may encounter PAD situations. As more states adopt PAD legislation, millions more individuals will be able to obtain prescriptions for lethal medication. It is crucial for state regulatory bodies to ensure that emergency responders, including EMS professionals, medical control, and law enforcement, are equipped with the knowledge, protocols, and ongoing education to appropriately handle PAD situations. Moreover, PAD patients and their families should be provided with resources to help them understand what to expect if they request an EMS response. By working toward these provisions, states can foster an emergency medical system that is better prepared for the challenges of PAD in the prehospital environment.

Table. Physician-assisted dying (PAD) legislation mentioning emergency medical services (EMS), and EMS protocols mentioning PAD or terminal illness in the 11 PAD-legalized states. Data was collected directly from PAD legislative texts and published statewide EMS protocols. States without statewide EMS protocols are denoted with N/A.

Jurisdiction	Authorization Year	Legislation	EMS mentioned in PAD Law?	PAD mentioned in EMS protocol?	Terminal illness mentioned in EMS protocol?
Oregon	1997	Death with Dignity Act	No	N/A	N/A
Washington	2008	Death with Dignity Act	No	No	Yes
Montana	2009	Rights of the Terminally Ill Act	No	No	No
Vermont	2013	Patient Choice and Control at the End-of-Life Act	No	No	No
California	2015 (reauthorized in 2021)	End-of-Life Options Act	No	N/A	N/A
Colorado	2016	End-of-Life Options Act	No	N/A	N/A
District of Columbia	2017	Death with Dignity Act	No	No	Yes
Hawaii	2018	Our Care, Our Choice Act	No	No	No
New Jersey	2019	Medical Aid in Dying for the Terminally Ill Act	No	No	No
Maine	2019	Death with Dignity Act	No	Yes	No
New Mexico	2021	Elizabeth Whitefield End-of-Life Options Act	No	No	No

EMS, emergency medical services; *PAD*, patient-assisted dying; *N/A*, not applicable.

Physician-Assisted Dying EMS Protocol

Patient Care Goals:

To acknowledge the ability of terminally ill patients with decision-making capacity to end their lives by self-administration of an ingested lethal medication via this state's physician-assisted dying law.

Patient Presentation:

Inclusion Criteria

1. Patient must have a physician-assisted dying (PAD) form, presented to EMS by family/caregiver/patient, and:
   - The patient has conveyed to EMS their wish to ingest their lethal medication **OR**
   - The patient is known or suspected to have ingested lethal medication prior to EMS arrival
2. The PAD form is verified to have the patient's name, the patient's signature, and a physician's signature.

Assessment

1. Determine the reason 9-1-1 was called (eg, complication from ingestion, the patient no longer wishes to die, etc).
2. Request physical POLST and/or DNR orders, if present.

Treatment and Interventions

1. A patient with decision-making capacity can change their intent to end their life even after ingesting lethal medication. If ingestion occurred prior to EMS arrival, document the medication used and the time of ingestion.
2. If the patient is exhibiting symptoms of distress (eg, pain, dyspnea, vomiting), treat symptoms per existing protocols to improve patient comfort.
3. Follow all POLST and/or DNR orders. If these documents are not available, contact on-line medical control for guidance.
4. If the patient is transported, ensure all relevant documentation is brought and provide early notification to the receiving facility of the patient's ingestion of lethal medication.

Refusal of Care

1. Ensure decision-making capacity as per standard refusal of care protocol.
2. If patient verbalizes intent to ingest lethal medication after EMS leaves the scene, contact on-line medical control for further guidance.
3. Document patient's intent in their own words and obtain witness signatures from family, caregivers, or bystanders.

Notes/Key Considerations

1. Death after lethal ingestion can take several minutes to hours.
2. Vomiting, seizures, or regaining consciousness may indicate a failed ingestion of lethal medication.

Figure. A sample emergency medical services protocol to guide the prehospital management of patients participating in physician-assisted dying.
EMS, emergency medical services; *POLST*, physician orders for life-sustaining treatment; *DNR,* do not resuscitate; *ED*, emergency

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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Rapid Titration of Methadone for Opioid Use Disorder in the Emergency Department: A Case Report

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Introduction: The prevalence of high-potency synthetic opioids (HPSO), such as fentanyl and its analogs, presents significant treatment challenges to current strategies for emergency department (ED) initiation of medication for opioid use disorder (MOUD). While most EDs traditionally use buprenorphine for MOUD, its effectiveness can be limited in patients exposed to HPSOs due to risk of precipitated withdrawal or inadequate control of withdrawal symptoms. Methadone, a full agonist, is another MOUD agent that addresses severe withdrawal symptoms and cravings associated with HPSO dependence and will not cause precipitated withdrawal. Traditional methadone protocols often fail to provide sufficient doses to alleviate withdrawal symptoms, but new federal guidelines allow higher initial doses and rapid titration to therapeutic levels.

Case Report: We report a case of rapid methadone titration in the ED for a patient with a history of high HPSO utilization. The patient received an initial dose of methadone 50 milligrams (mg) orally, followed by titration of additional 10 mg doses hourly to a cumulative dose of 70 mg at discharge. Vital signs, mental status, and Clinical Opiate Withdrawal Scale scores were monitored to guide dosing.

Conclusion: The protocol allowed for safe, individualized care, achieving therapeutic dosing levels that alleviated withdrawal symptoms and enabled the patient to transition to outpatient follow-up treatment. This approach addresses the need for rapid, effective methadone initiation in an era in which high-potency synthetic opioids pose challenges to traditional opioid use disorder treatment. [Clin Pract Cases Emerg Med. 2025;19(2):188-192.]

Keywords: *case report; methadone; rapid titration; medications for opioid use disorder; emergency department.*

INTRODUCTION

The rise in high-potency synthetic opioids (HPSO), including fentanyl and its analogs, is the leading cause of drug overdose deaths in the United States.[1] Emergency department (ED) initiation of buprenorphine for patients with opioid use disorder (OUD) has proven effective in enhancing medication for opioid use disorder (MOUD) treatment engagement and reducing illicit opioid use.[2,3] However, HPSOs present new treatment challenges due to their lipophilic nature, which leads to accumulation in adipose tissue and heightens the risk of

precipitated withdrawal when initiating buprenorphine, a high-affinity partial μ-opioid receptor agonist that displaces other opioids including fentanyl.[4] Methadone, a full μ-opioid receptor agonist, is advantageous in patients using HPSOs as it does not precipitate withdrawal.[4]

Patients who struggle to stop using HPSOs due to withdrawal intolerance or who have experienced precipitated withdrawal with buprenorphine often show poor MOUD retention, hesitancy to retry buprenorphine, and a preference for treatment with methadone.[5] However, methadone has been underused in the ED due to concerns for respiratory depression and restrictive federal regulations.[6] Until recently, the US Substance Abuse and Mental Health Services Administration guidelines for methadone recommended a maximum initial dose of 30 milligrams (mg). Subsequent dose increases by 5 to 10 mg every 3-5 days often required weeks of titration to achieve therapeutic levels.[7] This dosing regimen was established before the era of HPSOs. Patients often require maintenance doses of 120 mg/day or greater.[4,8] In the current HPSO era, there is a critical need to develop methadone titration protocols that achieve a therapeutic dose more quickly, increasing treatment retention and reducing the risk of fatal overdose.[9]

Recent changes in 42 Code of Federal Regulations Part 8 allow for higher initial dosing of methadone (up to 50 mg) with additional dosing at the physician's discretion if a clinical indication (eg, persistent withdrawal symptoms) is documented.[10] We previously described the feasibility of methadone initiation in the ED for patients with OUD through our Start Treatment and Recovery (STAR) program.[11] In this case report, we present modifications to our protocol that enabled the safe and rapid titration of methadone to therapeutic levels in the ED for a patient with OUD.

CPC-EM Capsule

What do we already know about this clinical entity?
Buprenorphine may precipitate withdrawal. Methadone is an alternative, but traditional dosing may not adequately control symptoms.

What makes this presentation of disease reportable?
We describe rapid methadone titration in the emergency department (ED), safely initiating treatment at 50 mg and titrating to a cumulative 70 mg dose to manage withdrawal symptoms.

What is the major learning point?
New federal guidelines permit an increase in the initial methadone dose from 30 to 50 mg, with further titration to higher doses when clinically indicated.

How might this improve emergency medicine practice?
Given the prevalence of high-potency synthetic opioids, increasing methadone doses can improve withdrawal management and retention in treatment for opioid use disorder.

CASE REPORT

With written patient consent, we report on a 34-year-old male who presented to the ED seeking inpatient treatment for his OUD. The patient reported a history of severe OUD using fentanyl six times daily via intravenous (IV) injection. Upon arriving at the ED in the early morning, the patient showed no signs of withdrawal, had normal vital signs and no other complaints, but he appeared sedated, consistent with recent opioid utilization. A peer recovery coach (PRC) was paged to assist in identifying residential treatment programs. The patient slept most of the day while the PRC explored placement options. Upon awakening, the patient reported withdrawal symptoms, including restlessness, sweating, and agitation, and expressed interest in MOUD.

Using shared decision-making, the emergency physician and STAR coordinator had a detailed conversation with the patient regarding his choice of MOUD with either buprenorphine/naloxone or methadone. Given the patient's use of HPSOs and prior precipitated withdrawal with buprenorphine, methadone was chosen. The patient recognized that his chances of success in a residential treatment program would improve if he were first stabilized on an effective dose of MOUD. He agreed to initiate methadone in the ED, titrate the dose as much as possible per protocol, and follow up with our local, affiliated outpatient treatment program (OTP) for further dose adjustments before enrolling in a residential program.

The patient was treated using our rapid methadone titration pathway, which includes an initial dose of 40-50 mg orally based on clinician discretion, followed by 10 mg every hour, as clinically indicated for persistent withdrawal symptoms, until symptomatic relief is achieved, or a maximum dose of 70 mg is reached. Titrated dosing is only provided if the patient's Clinical Opiate Withdrawal Scale (COWS) score is greater than five, and there are no signs of sedation including hypoventilation (SpO_2 less than 94% on room air or respiratory rate less than 12 respirations per minute) or hypotension (systolic blood pressure less than 100 millimeters of mercury). Exclusion criteria are considered prior to rapid methadone titration. Contraindications include known QTc prolongation and medical comorbidities such as

pulmonary disease, cirrhosis, end-stage renal disease, congestive heart failure, or ventricular arrhythmia. Relative contraindications include the concurrent prescription of benzodiazepines, tricyclic antidepressants, alcohol or other sedatives, and medications affecting methadone metabolism (eg, CYP inhibitors and inducers) (Figure).[9]

At the time of MOUD initiation the patient had a COWS score of 14, and 50 mg of methadone was administered. The patient was reassessed after one hour. He had no signs of sedation or respiratory depression, and the recalculated COWS score was 10. An additional 10 mg of methadone was given. Another 10 mg methadone dose was administered 90 minutes later for persistent withdrawal symptoms and a COWS score of 6, for a total dose of 70 mg. Five hours after the initial dose, the patient again had a COWS score of 6 but stated that he felt improved. At no point were any complications observed. No additional medications were administered during the ED visit. The patient was observed for one hour after the final methadone dose, prior to discharge (Table). The patient successfully followed up with the outpatient treatment program (OTP) the next morning at which time he was given an 80 mg dose of methadone. This was followed by an additional 100 mg dose the following day, both administered without any adverse effects.

DISCUSSION

We present a case report demonstrating the feasibility and safety of rapid methadone titration in the ED to initiate MOUD. This case highlights the importance of methadone as a treatment option, particularly in the era of HPSOs, which have complicated OUD treatment. Methadone offers a crucial alternative to buprenorphine that mitigates the risk of precipitated withdrawal caused by buprenorphine and is associated with a lower risk of treatment discontinuation, particularly at the beginning of the treatment episode. In addition, the use of shared decision-making and clinical judgment to choose between buprenorphine and methadone, with careful consideration of the patient's level of exposure to HPSOs, use of other substances, and co-morbidities, leads to improved MOUD adherence and outcomes.[5,12,13]

Traditional methadone protocols recommend titrating to a maximum dose of 30-40 mg on the first day, increasing by 10 mg daily to reach 60 mg by day three, and maintaining this dose for five days before making further adjustments.[7] Such a conservative approach may not be feasible for patients using HPSOs, as reaching a therapeutic dose may take over two weeks, during which time withdrawal and cravings may be inadequately controlled, potentiating relapse and overdose.[9] New federal guidelines allow for more flexible dosing protocols, including higher initial doses that enable rapid titration to therapeutic levels. These regulations state that the initial dose of methadone shall be individually determined with consideration of the types of opioids and other substances the patient is using, medical history, and severity of opioid withdrawal.[10]

Under these new guidelines, patients must be assessed for opioid exposure. We suggest that patients using HPSOs daily be considered to have high opioid exposure, with a treatment goal of titrating the methadone dose to 40-70 mg before ED discharge. In contrast, patients with lower opioid tolerance, such as those using only prescription opioids, only occasional opioids, or who have been exposed via contaminated stimulants (eg, cocaine, methamphetamine), need lower doses of methadone and should be initiated at 20-30 mg. Our case demonstrates how, with careful patient selection, rapid titration of methadone can be safely accomplished in the ED, an ideal setting for methadone initiation due to availability of cardiopulmonary monitoring, immediate access to medical interventions, and streamlined referrals to OTPs. Inpatient case reports have reported the successful induction of patients with severe OUD who were rapidly titrated on methadone starting at 30 mg with an additional 10 mg every three hours to a total of 60-70 mg on day one.[14]

Higher induction doses of methadone are not without risk. Methadone's long half-life increases the risk of overdose, especially during the first two weeks of treatment. The most common complication associated with methadone administration is over-sedation, which typically does not require naloxone administration and can be effectively monitored in the ED.[15] Given its complexity compared to buprenorphine, it is essential that emergency physicians are thoroughly trained in the proper administration and titration of methadone.

Table. Patient vital signs and Clinical Opiate Withdrawal Scale scores during methadone titration.

Time Elapsed	RR (rpm)	HR (bpm)	BP (mm Hg)	SpO_2% (Room air)	Clinical Opiate Withdrawal Scale	Amount of Methadone Administered
0:00	12	89	110/79	97	14	50 mg
1:25	16	77	115/84	100	10	10 mg
2:59	-	83	109/80	98	6	10 mg
4:48	15	71	107/65	98	6	-

BP, blood pressure; *bpm*, beats per minute; *HR*, heart rate; *mg*, milligram; *mm Hg*, millimeters of mercury; *rpm*, respirations per minute; *RR*, respiratory rate; *SpO_2*, oxygen saturation.

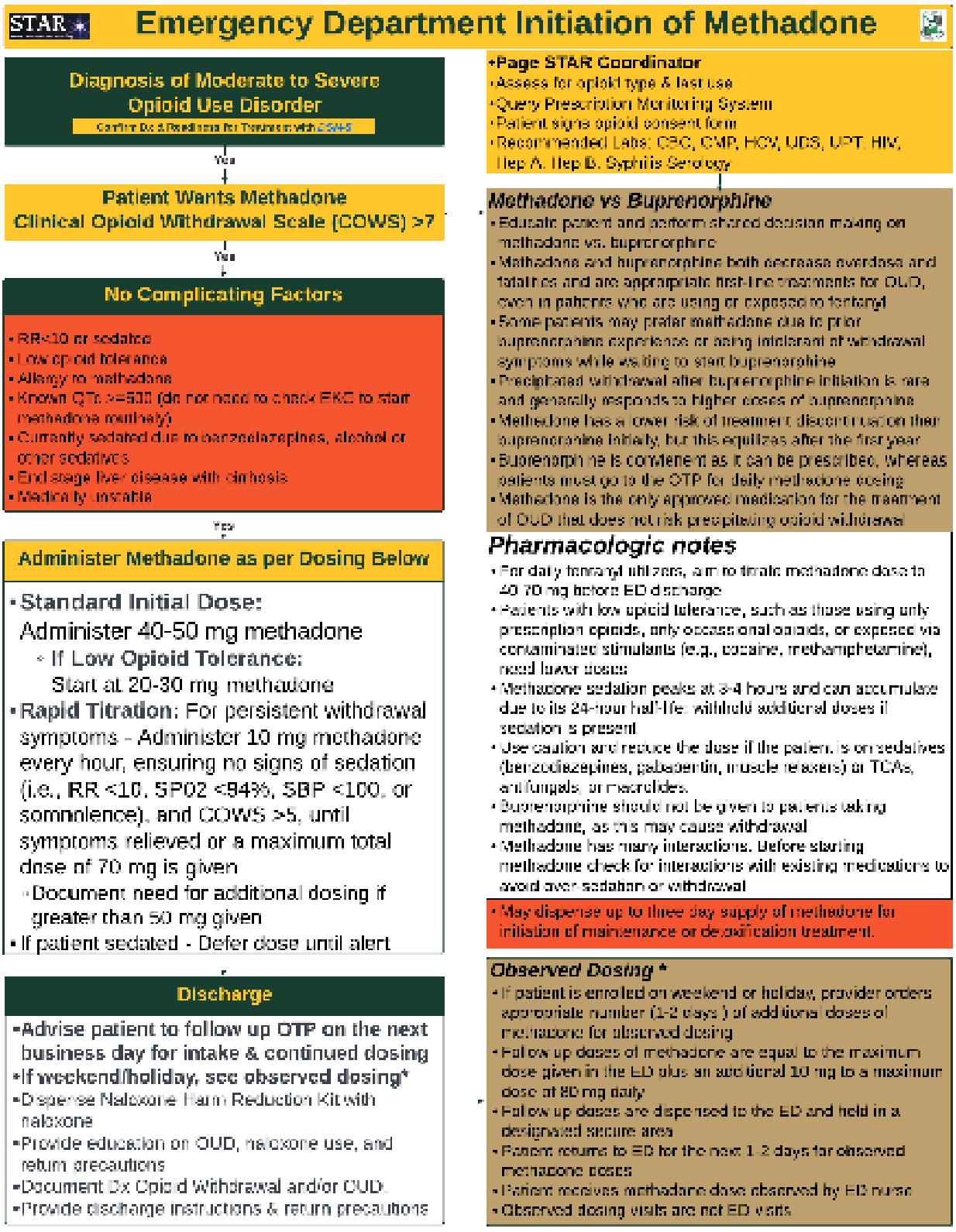


Figure. Emergency department rapid titration of methadone Start Treatment and Recovery pathway.
CBC, complete blood count*; CMP*, comprehensive metabolic panel*; COWS*, Clinical Opiate Withdrawal Scale; *ED,* emergency department; *EKG*, electrocardiogram; *mg*, milligram; *HCV*, hepatitis C; *OTP*, outpatient treatment program; *OUD*, opioid use disorder; *RR*, respiratory rate; *SpO_2*, oxygen saturation; *STAR*, Start Treatment and Recovery; TCA, tricyclic antidepressant; *UDS*, urine drug screening; *UPT*, urine pregnancy test.

A limitation of our rapid methadone titration protocol is treatment time. Our patient was in the ED for 17 hours, although once the rapid methadone titration started, the patient was able to receive the maximum 70 mg dose and be discharged after a period of observation in under five hours. Additionally, it is imperative to have pre-existing agreements for rapid follow-up at a local OTP and a plan for methadone bridge dosing if the patient presents to the ED on a weekend or holiday.

CONCLUSION

While buprenorphine initiation has become the ED standard for medication for opioid use disorder, the prevalence of high-potency synthetic opioids and the risk of precipitated withdrawal often necessitate treatment with methadone. In the era of HPSOs, effective methadone dosing requires rapid titration to achieve therapeutic levels quickly. We recommend considering rapid methadone titration in the ED to initiate MOUD for carefully selected patients with OUD and high opioid exposure, following shared decision-making and thorough assessment.

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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Unusual Complications in Cocaine Stuffers: A Case Report

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Introduction: Body stuffing is defined as ingesting small quantities of drugs in poorly sealed packets, often to avoid repercussions from law enforcement. Cocaine is one of the drugs most commonly involved. Complications reported with stuffing include aspiration, esophageal obstruction, and fatal toxicity. Survival from mechanical airway obstruction due to drug stuffing has not been reported.

Case Report: We present a case of a 32-year-old male who was a cocaine body stuffer, complicated by agitated delirium, cardiotoxicity, and airway obstruction requiring resuscitation, followed by a surgical tracheostomy to retrieve the obstructing cocaine bag. The patient's hospital course was further complicated by rhabdomyolysis and acute kidney injury requiring dialysis. He was discharged in stable condition after a four-week hospital stay.

Conclusion: This case highlights the severe risks of cocaine body stuffing, including airway obstruction and cocaine-induced arrhythmias. Endotracheal intubation in such cases warrants a careful airway assessment to mitigate the risks of obstructive complications. [Clin Pract Cases Emerg Med. 2025;19(2):193-195.]

Keywords: *body packing; body stuffing; cocaine; toxicity.*

INTRODUCTION

Body stuffing is defined as ingesting small quantities of drugs in poorly sealed packets, often to avoid repercussions from law enforcement.[1] Cocaine is one of the drugs most commonly involved in stuffing cases. Other drugs include amphetamines and opioids. Complications reported with stuffing include aspiration, esophageal obstruction, and fatal toxicity due to the leakage of the products into the gastrointestinal tract.[2] Mechanical airway obstruction secondary to drug stuffing is rare, but it carries lethal consequences if not recognized and managed appropriately in a timely manner.

A case of airway obstruction secondary to heroin stuffing was reported by Sullivan et al in 2017.[3] However, that patient expired in the emergency department (ED) due to asphyxiation from an obstructing bag in the right main bronchus. Prompt identification of airway obstruction by medical personnel may limit further complications in body stuffers. We present a case of a cocaine stuffer who presented with systemic manifestations of cocaine toxicity followed by a mechanical airway obstruction.

CASE REPORT

A 32-year-old male was brought in by law enforcement to the ED after reportedly stuffing a suspected illicit drug packet on the street. During initial ED assessment he refused to open his mouth or cooperate. He then had an episode of emesis followed by agitation. Despite administration of midazolam

10 milligrams (mg) and haloperidol 20 mg intramuscularly, he ultimately required intubation via direct laryngoscopy for airway protection and agitated delirium. Once intubated, he was noted to have mydriasis, hypertension, and electrocardiography changes of regular wide complex tachycardia (QRS: 240 milliseconds), right bundle-branch block pattern and a terminal R-wave in AVR (Image 1). He was given a total of 380 milliequivalents of intravenous sodium bicarbonate until the QRS-interval normalized (Image 2). Over the following hours, he was noted to have a high peak airway pressure in the mechanical ventilator and was difficult to ventilate with a bag-valve mask.

He was given albuterol, rocuronium, and ketamine without improvement of his airway pressure. Chest radiograph was unremarkable. An emergent bronchoscopy was performed at the bedside that revealed a plastic bag at the distal end of the endotracheal tube. The patient then developed total airway obstruction and cardiac arrest while the physician was attempting to retrieve the bag by bronchoscope. Return of spontaneous circulation was achieved after a left main bronchial stem intubation was performed with standard resuscitation. Emergency tracheostomy was performed in the operating room, and a plastic bag containing a white powder was removed (Image 3). The patient was admitted to the intensive care unit for further stabilization and supportive care. His hospital course was complicated by rhabdomyolysis, elevated liver enzymes, and acute kidney injury requiring hemodialysis. He was discharged in stable condition after four weeks.

CPC-EM Capsule

What do we already know about this clinical entity?
Body stuffing is the ingestion of poorly sealed drug packets (commonly cocaine) to evade law enforcement, carrying risks of substance toxicity, esophageal obstruction, and airway obstruction.

What makes this presentation of disease reportable?
Airway obstruction is a rare complication of body stuffing; this case highlights both cocaine poisoning and mechanical airway obstruction, emphasizing its severity.

What is the major learning point?
Physicians should recognize the risk of airway obstruction in suspected body stuffers and take extra precautions during intubation to prevent obstructive complications.

How might this improve emergency medicine practice?
It highlights the need for heightened awareness and careful airway management in body stuffing cases, improving patient outcomes and reducing complications.

DISCUSSION

We present a case of a cocaine body stuffer who presented with both sympathomimetic toxidrome and airway obstruction. His course was complicated by a cardiac arrest, attributed to possible mechanical airway obstruction. Wide complex tachycardia has a wide range of causes including structural cardiac disease, electrolyte and metabolic derangements, and ischemic heart disease. However, in the context of body stuffing, specifically with cocaine, it should raise the concern about its mechanism as a sodium channel blocker. This clinical effect should be managed aggressively with sodium bicarbonates to avoid further progression to ventricular dysrhythmias and cardiac arrest.

Aspiration and subsequent airway obstruction are rare but

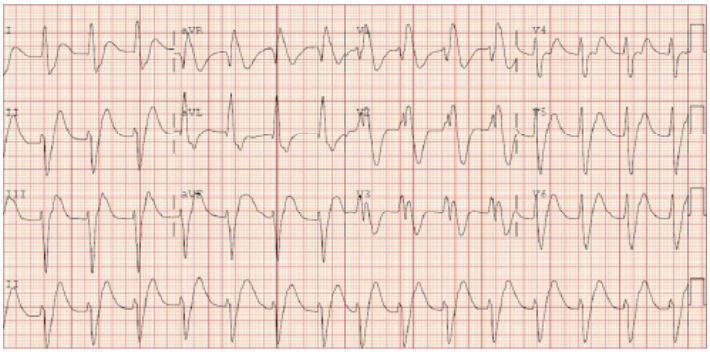


Image 1. Electrocardiography prior to the administration of sodium bicarbonates in a cocaine body stuffer, demonstrating widening of the QRS-interval.

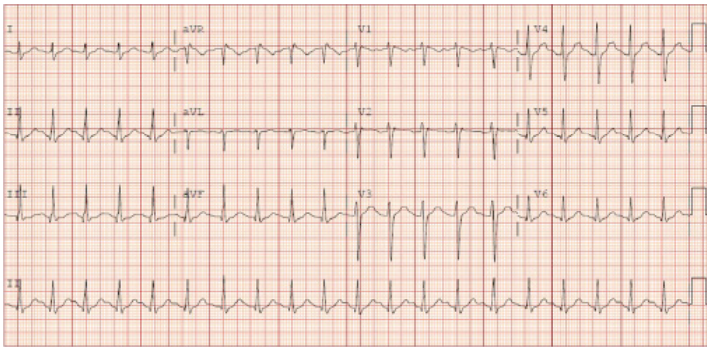


Image 2. Electrocardiography post-sodium bicarbonate administration in a cocaine body stuffer, demonstrating narrowing of the QRS-interval.



Image 3. Plastic bag extraction from the trachea in a cocaine body stuffer through tracheostomy.

life-threatening complications of body stuffing that need to be considered. We suspect that our patient aspirated the plastic bag while vomiting when he developed altered mental status and agitation that led to subsequent upper airway obstruction. It is also possible that performance of direct laryngoscopy intubation might have unintentionally pushed the stuffed packet distally in the airway. It has been described in the literature that body stuffers may not actually ingest drug packets but keep them in the oropharynx.[4]

Our case shares similar findings with a report by Narula et al, which described a 23-year-old man who developed airway obstruction and prehospital cardiac arrest after ingesting a small bag containing white powder while attempting to flee from the police. Similar to our case, the diagnosis was made by bronchoscopy after airway obstruction was suspected due to difficulty in bagging the patient, failure to ventilate, and elevated peak pressures on the ventilator.[5]

The risk of toxicity in body stuffing is strongly associated with the type of packaging and the amount of the drug that it contains. An analysis of 683 packages in body stuffers showed that 74% of them used filter paper and 11% single-layer plastic wraps or pouches,[6] which makes it easy to leak and cause significant toxicity as presented in our case.

CONCLUSION

We present a case of a cocaine body stuffer who suffered a sympathomimetic toxidrome, cardiac arrest, and airway obstruction by the ingested packet. In addition to benzodiazepines and sodium bicarbonate for the treatment of severe cocaine toxicity, careful consideration should be made when performing endotracheal intubation to assess for foreign bodies and airway obstruction in body stuffers.

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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Inferior Vena Cava Tumor Thrombus in the Emergency Department: A Case Report

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Introduction: The inferior vena cava (IVC) drains a significant portion of the lower body. Pathologies associated with the IVC can present significant diagnostic and therapeutic challenges. We present a case of IVC tumor thrombus in the emergency department.

Case Report: A 76-year-old male with symptoms of volume overload was evaluated, leading to the diagnosis of IVC mass likely from tumor thrombus.

Conclusion: Patients with volume overload should be evaluated for both heart failure and presence of a potential thrombus. Point-of-care ultrasound and other imaging modalities play crucial roles in early diagnosis. Prompt identification and differentiation between bland and tumor thrombi are vital for appropriate management, potentially improving patient outcomes. [Clin Pract Cases Emerg Med. 2025;19(2):196-199.]

Keywords: *inferior vena cava; tumor thrombus; congestive heart failure; thromboembolism.*

INTRODUCTION

The inferior vena cava (IVC) is the largest vein in the body, responsible for draining a significant portion of the lower body.[1] Pathologies associated with the IVC, including thrombosis and neoplasms, can present significant diagnostic and therapeutic challenges, especially when first identified in the emergency department (ED).[2]

Inferior vena cava thrombosis is a rare process that usually stems from a congenital abnormality, but it is found even more rarely from an acquired cause.[2] The most common acquired cause of IVC thrombosis is from an unretrieved IVC filter.[2] Other than this, patients with a combination of a predisposing hypercoagulable state and prothrombotic conditions and/or abdominal pathologies are at risk for IVC thrombosis as well.[2,3] For these reasons, IVC abnormalities should be assessed carefully to guide clinical-decision making and improve patient care.

Acquired causes of IVC thrombus can be further stratified into bland thrombus, pseudo-thrombus, and primary and secondary malignancies.[1] A tumor thrombus is the presence of a tumor extending into blood vessel walls such as the portal vein or the IVC.[4] Malignancies like this can have intravascular extensions in underlying leiomyosarcoma and renal cell carcinoma. Differentiation between a bland thrombus and tumor thrombus is vital for determining therapeutic approach and can be done with ultrasound, magnetic resonance imaging (MRI), and computed tomography (CT).[5] In the ED, ultrasound has revolutionized point-of-care imaging, providing faster diagnoses, especially in thrombus identification.[5] Treatments vary greatly depending on clinical identification of the thrombus but may include anticoagulation, chemotherapy, thrombolysis, and surgical resection.[6] There is limited research available on the topic of IVC tumor thrombus, which warrants further research.

In this report, we discuss the case of a man who sought emergent care for worsening dyspnea and fatigue as well as noticeable leg swelling, secondary to IVC obstruction.

CASE REPORT

A 76-year-old male with a history of atrial fibrillation, diabetes mellitus, and hepatitis presented to the emergency department with progressive shortness of breath and generalized weakness over five days. Physical examination revealed bilateral lower extremity edema, decreased breath sounds in the right lower lung field, 2+ bilateral pitting edema to mid-lower legs, and motor weakness with chronic, right-foot drop. Electrocardiogram showed atrial fibrillation with rapid ventricular response, and laboratory tests revealed normocytic anemia, hyperglycemia, and elevated blood urea nitrogen/creatinine levels. Troponin levels were elevated, with values of 382 nanograms per liter (ng/L) at two hours and 429 ng/L at four hours (reference range <40 ng/L). However, the B-type natriuretic peptide (BNP) was normal at 96 picograms per milliliter (pg/mL) (<100 pg/mL).

A point-of-care cardiac ultrasound to evaluate for volume overload and cardiac function revealed a well-circumscribed mass in the IVC near the right atrium (Images 1 and 2). Chest radiograph showed mild pulmonary vascular congestion. A subsequent CT angiogram of the chest, abdomen, and pelvis demonstrated an ill-defined hypodense area at the intrahepatic and suprahepatic portions of the IVC, raising concern for a mass. Additional findings included mild hepatomegaly, a nodule in the left urinary bladder, and lymphadenopathy in the mediastinal, aortocaval, and periportal regions, suggesting neoplasm and metastases.

The on-call vascular surgeon recommended transfer to a higher level of care for oncological and surgical evaluation. However, the patient declined surgical intervention and opted for hospice care.

CPC-EM Capsule

What do we already know about this clinical entity?
Inferior vena cava (IVC) obstruction can have multiple etiologies and lead to severe systemic consequences, thus crucially requiring an early and accurate diagnosis.

What makes this presentation of disease reportable?
A potential IVC tumor thrombus can present clinically similar to Congestive heart failure (CHF), making it easy to miss if not correctly identified early.

What is the major learning point?
Point-of-care ultrasound (POCUS) can be useful to quickly differentiate between IVC occlusions/ tumor thrombus and typical CHF secondary to right-sided heart failure.

How might this improve emergency medicine practice?
Rapid differentiation of an IVC occlusion using POCUS can expedite diagnosis of IVC tumor thrombus and initiate proper treatment and management.

DISCUSSION

The impairment and/or obstruction of the IVC can lead to severe, systemic consequences, making early detection and accurate diagnosis crucial. Abnormalities of the IVC can stem from congenital malformations, trauma, or acquired diseases that result in compression and hinder venous return.[1,2] Among the risk factors for acquired IVC-related illnesses are

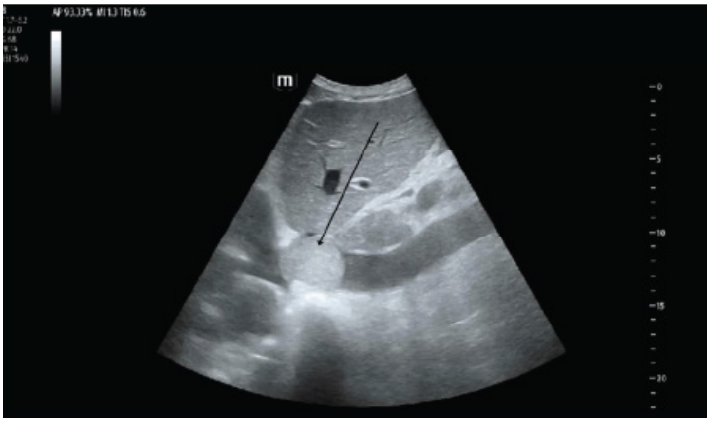


Image 1. Point-of-care ultrasound of the inferior vena cava (IVC) showing a hyperechoic circular mass in the proximal IVC (arrow) adjacent to the right atrium. Distal IVC is seen plethoric.

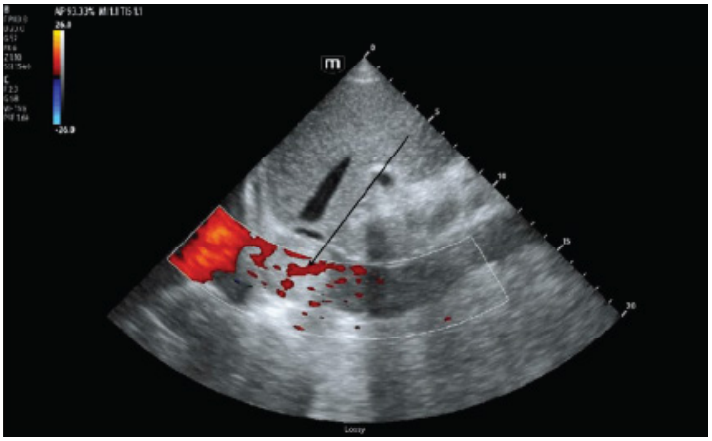


Image 2. Point-of-care ultrasound of the inferior vena cava (IVC) with color Doppler applied showing poor venous return (arrow) to the right atrium secondary to IVC mass/tumor thrombosis.

infection, obesity, vascular diseases, pregnancy, and malignancy.[3,7,8] Tumor thrombus of the IVC, while rare, is a critical finding that can present with varying symptom severity depending on its size and location. The Mayo Clinic's staging system for tumor thrombus classification, ranging from level 0 (thrombus extending into the renal vein) to level 4 (thrombus extending into the supradiaphragmatic IVC or the right atrium), provides a structured approach for assessment.[9] The presented case, with a level 4 thrombus, underscores the complexity and severity of near-complete occlusion of the IVC neighboring the right atrium.[9]

Differentiating between a bland thrombus and a tumor thrombus is vital due to their differing treatment approaches. A bland thrombus carries the risk of embolization and dissemination, necessitating early identification to implement strategies such as IVC filters, anticoagulation, and thrombolytics.[5] In contrast, surgical resection remains the primary curative approach for a tumor thrombus, as pharmacological interventions are typically ineffective and the risk of metastases is significant. Historically, conventional venography was the gold standard for diagnosing venous thrombosis. However, CT and MRI now provide reliable readings, with ultrasound, particularly color Doppler ultrasound, serving as a valuable first-line modality.[5] Computed tomography is often used initially to identify IVC pathologies due to its effectiveness in detecting abdominal abnormalities. Notably, some studies have identified a "streak and thread" sign as indicative of tumor thrombus on CT.[10] For visualizing an IVC tumor thrombus, certain types of MRI are considered more reliable than CT and have the advantage of not using ionizing radiation.[11] Doppler ultrasound is beneficial in demonstrating abnormal or reduced blood flow caused by an IVC mass, although its effectiveness can be limited by operator experience and artifacts from adjacent structures.[11,12]

Emerging imaging modalities such as contrast-enhanced ultrasound (CEUS) offer a comprehensive view, as tumor thrombi typically include small vessels that can be distinctly visualized due to blood-pooling contrast enhancement.[13] Both CEUS and Doppler ultrasound provide expedited, cost-effective imaging compared to MRI and CT, maintaining high sensitivity and specificity. This case aligns with these findings, as both Doppler ultrasound and CT demonstrated IVC occlusion.

Diagnosing an IVC tumor thrombus can be particularly challenging, as it can mimic the clinical presentation of congestive heart failure (CHF). This mimicry is due to shared symptoms and hemodynamic consequences. Congestive heart failure, both left-sided and right-sided, typically arises from the heart being unable to maintain adequate cardiac output. This leads to compensatory responses that, while initially adaptive, exacerbate fluid retention, vascular resistance, and myocardial remodeling, ultimately increasing systemic congestion and cardiac dysfunction. The patient in this case report, with a normal BNP of 96 pg/mL and no prior history of heart failure, presented similarly to a CHF patient. Both IVC thrombus and CHF may manifest with symptoms of systemic venous congestion, including dyspnea, orthopnea, and lower extremity edema.[1-3] The obstruction of venous return by an IVC tumor thrombus can elevate venous pressures, leading to fluid extravasation into the interstitial space and increasing systemic venous pressure. Over time, if the left heart is unable to compensate for this increase in systemic venous pressure, there will be an elevation in pulmonary venous pressure. This will reduce lung compliance and impair gas exchange, resulting in dyspnea and subsequent pulmonary congestion. This mirrors the pathophysiological cascade of CHF. Additionally, an IVC tumor thrombus can exacerbate pre-existing cardiac dysfunction or predispose individuals to decompensated heart failure.

In the context of this patient, it may be valuable to compare the effects of an IVC tumor thrombus to those of CHF, particularly secondary to right-sided heart failure. As in CHF, obstruction of the IVC impairs venous return, leading to decreased right ventricular preload and subsequent overcompensation by the right heart. Over time, this compensatory mechanism may progress to contractile dysfunction, ultimately impairing the cardiac output of the right ventricle and resulting in right-sided heart failure, characterized by hepatomegaly, ascites, and peripheral edema, which can be indistinguishable from advanced CHF manifestations. Distinguishing between an IVC tumor thrombus and CHF is crucial for guiding appropriate therapeutic interventions.[14]

While diuretics and vasodilators are cornerstone treatments for CHF, managing an IVC mass may require interventions ranging from anticoagulation to surgical resection. Surgical options for IVC tumor thrombus may include the need for sternotomy, cardiopulmonary bypass, and coronary artery bypass graft, with the choice of surgery depending on the size and location of the thrombus.[15] Swift and accurate recognition is essential for determining the appropriate surgical route.

Had the patient not elected hospice care, further evaluation of the symptoms and mass through advanced imaging and diagnostic modalities, such as echocardiography, chest CT, positron-emission tomography (PET) or biopsy, would have been essential. An echocardiogram could have provided an estimate of ejection fraction and offered detailed visualization of hemodynamic changes, enhancing the overall clinical assessment. A chest CT would have supported the findings of pulmonary congestion observed on the chest radiograph and provided a more comprehensive view, potentially identifying smaller or early-stage pathologies that might have been missed on the radiograph.[5] Additionally, PET demonstrating vessel expansion and a biopsy confirming neoplastic cell distribution would have established a definitive diagnosis of tumor thrombus.[10]

The absence of these diagnostic steps represents a

significant limitation of this case report. A further limitation is the absence of CEUS, a modality particularly effective in visualizing thrombosis. Although Doppler ultrasound was chosen for its speed and efficacy, CEUS could have provided additional comparative insights, enhancing the diagnostic accuracy. Despite these limitations, this case underscores the critical importance of recognizing and diagnosing IVC tumor thrombus to guide appropriate clinical management.

CONCLUSION

Inferior vena cava tumor thrombosis can present with a wide range of symptoms and subsequently cause significant morbidity. This case underscores the pivotal role of ultrasound in the prompt identification of an IVC tumor thrombus, enabling timely and diagnostic therapeutic interventions. This rare case did not have a correspondingly obvious presentation; however, with thorough preliminary workup, a level four IVC tumor thrombus was identified.

Given the overlap in clinical presentation between IVC tumor thrombus and CHF secondary to right-sided heart failure, ultrasound (Doppler/CEUS) can be used as a rapid and cost-effective frontline imaging modality, allowing clinicians to tailor treatment plans and investigate accordingly. It is important to keep this pathology in mind as part of the differential diagnosis. Therefore, integrating ultrasound into the diagnostic algorithm for patients presenting with symptoms suggestive of venous congestion is imperative for timely and accurate diagnosis of IVC occlusion. With continued testing, the identification of IVC tumor thrombus can be confirmed, which gives better direction to proper treatments and outcomes.

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

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Case Report: Refractory Ventricular Fibrillation Resolved by Double External Defibrillation and Beta Blockade

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Introduction: The mortality rate for refractory ventricular fibrillation (RVF) can be up to 97%. There is no widely accepted treatment plan for this stage of ventricular fibrillation besides the standard combination of defibrillation, amiodarone, and epinephrine. One novel approach that has been documented in a select few cases since 2015 is the combination of double external defibrillation (DED) and esmolol-induced beta blockade.

Case Report: We report the case of a 65-year-old man who presented with RVF after collapsing at work. Upon the simultaneous administration of two defibrillators with a combined shock of 400 joules and 35 milligrams of the beta blocker esmolol, the patient regained pulse and began blinking. He was discharged from the hospital after seven days and walked out of the clinic.

Conclusion: This case continues the trend of several case reports since 2015 that have featured beta blockade and double external defibrillation as a viable solution to refractory ventricular fibrillation. Since there is limited quantifiable data on the efficacy of this treatment, future studies should aim to evaluate whether the combination of DED and beta blockade has the potential to become the new standard in treating RVF over a broader patient population. [Clin Pract Cases Emerg Med. 2025;19(2):200-202.]

Key Words: *refractory ventricular fibrillation; double external defibrillation; beta blockade; esmolol; defibrillation.*

INTRODUCTION

Ventricular fibrillation (VF) is the leading cause of sudden cardiac death in patients with myocardial infarction, accounting for about 70% of mortalities.[1] Ventricular fibrillation is characterized by a period of sporadic electrical output interfering with the process of ventricular excitation, which physically manifests in the heart rate becoming too high to competently pump blood. The patient often dies in minutes.[2] With early intervention, typically consisting of cardiopulmonary resuscitation (CPR) and external defibrillation, many patients can have a prognosis comparable to those with myocardial infarction who have not experienced VF.[3] Patients may also experience refractory ventricular fibrillation (RVF), where a return of spontaneous circulation (ROSC) cannot be established within 10 minutes despite three attempts at defibrillation and the administration of 300 milligrams (mg) of amiodarone and 3 mg of epinephrine. In these cases, the mortality rate can be up to 97%.[4] At this stage of cardiac arrest, the use of double external defibrillation (DED) and beta blockade may be considered, although the research to quantitatively support both treatments in RVF is limited.[5,6] The following is a case of RVF that responded to the use of DED in combination with beta-blocker therapy.

CASE REPORT

A 65-year-old man presented to the emergency department (ED) for a cardiac arrest witnessed outside the hospital. The patient was at work with his colleagues when he collapsed and was noted to be pulseless. Bystander CPR was initiated until emergency medical services arrived. During transportation to the hospital via ambulance, an initial VF rhythm was found;

subsequently, the patient was defibrillated a total of five times and given six doses of epinephrine 1 mg. In addition, he received bicarbonate, calcium, and 450 mg of amiodarone prior to arrival. During transport to the ED, he had a brief, five-minute period of ROSC but lost his pulse again prior to ED arrival. During ROSC, his blood pressure was 132/112 millimeters of mercury, his respiration rate was 20 breaths per minute on pulse oximetry with a saturation of 89%, and his heart rate was 69 beats per minute. Total time between the onset of cardiac arrest and arrival at the ED was 40 minutes.

Upon arrival, the patient had a Lund University Cardiopulmonary Assist System (LUCAS) in place and was placed onto a stretcher. For two minutes CPR was performed, during which he was given 1 mg of epinephrine. He was intubated during the first pulse check, and the rhythm check showed VF on the monitor. Cardiopulmonary resuscitation was restarted, and two Zoll defibrillators were attached to the patient and charged to 200 joules (J) each. The two defibrillations were delivered simultaneously, with a combined output of 400 J. Then CPR was resumed, and 35 mg of esmolol (approximately 0.5 mg/kilogram) was administered. Shortly after, the patient began blinking, and during the subsequent pulse check he was noted to have a strong carotid pulse.

The patient was placed on continued mechanical ventilation and brought to the cardiac catheterization lab. He was found to have significant occlusions in both the proximal left anterior descending artery (LAD) and the proximal left circumflex artery (LCA), requiring four stents to be placed in the LAD and two in the LCA. He then was put under targeted temperature management and started on vasopressors from which he was weaned over two days. On day four he was extubated. On day seven he was discharged and able to walk out of the hospital. During his one-week follow-up visit, the patient stated that he felt great and was back to his normal, active lifestyle and did not experience any of his original symptoms. He also did not need or want cardiac rehabilitation services, and even quit smoking.

CPC-EM Capsule

What do we already know about this clinical entity?
Ventricular fibrillation is a common cause of cardiac death; current advanced cardiovascular life support guidelines recommend cardiopulmonary resuscitation, defibrillation, epinephrine, and antiarrhythmics

What makes this presentation of disease reportable?
This patient's refractory ventricular fibrillation (RVF) was resolved by a combination of double external defibrillation and beta blockade.

What is the major learning point?
The novel combination of double defibrillation and esmolol can be effective in treating RVF, leading to recovery.

How might this improve emergency medicine practice?
These findings could help redefine RVF management, improving survival and outcomes by integrating novel defibrillation techniques and pharmacotherapy.

DISCUSSION

Current guidelines for managing VF emphasize the significance of early intervention with the use of CPR and an automated external defibrillator as being crucial to increasing chances of survival. In fact, in cases of out-of-hospital cardiac arrest where the victim receives early bystander CPR, defibrillation, or both, the incidence of brain damage and death from any cause has been found to be significantly lower.[7] Given the rapid and early care given to the patient in our case, it is likely that this intervention prevented him from garnering significant neurological injury and was certainly a contributing factor to his full recovery.

The management of RVF is less clear. One of the approaches to treating RVF is to use novel defibrillation strategies. A 2022 study found that in RVF patients, both double sequential external defibrillation (DSED) and vector-change (VC) defibrillation were associated with significantly higher rates of survival to hospital discharge than standard defibrillation. Moreover, DSED but not VC defibrillation was associated with a higher percentage of patients with a favorable neurological outcome.[8]

There are several theories that may explain the relative success of DSED in RVF patients: the power theory, the setting up theory, and the multiple vector theory. According to the power theory, the use of more energy allows for more complete recruitment and conversion of the patient's myocytes out of RVF rhythm. If using this approach, the two shocks must be delivered simultaneously, as performed in this particular case. According to the setting up theory, the two shocks must be performed with a deliberate pause between. The first current lowers the defibrillation threshold, allowing for a higher chance at success converting all the remaining myocytes with the second shock. Finally, the multiple vector theory works in tandem with both the two aforementioned theories, as it simply entails the application of multiple defibrillation pads on a patient, thereby increasing the number of vectors that can be used to conduct current to the myocardium.[5]

CONCLUSION

The administration of the beta blocker esmolol is useful in high-acuity cases such as those of RVF patients, as the drug

exhibits a very rapid onset with a nine-minute half-life.[9] In a 2016 study by Lee et al, sustained ROSC was far more common in RVF patients treated with esmolol than in controls; additionally, short- and long-term survival and neurological outcomes were more than twice as good in the esmolol group.[10] Despite the documented benefits of both esmolol and double external defibrillation as separate treatments for RVF patients, there is limited literature available on the integration of both of these strategies in clinical cases. Since this combination of treatment was first proposed in a 2015 paper by McGovern and McNamee, there have been several case reports documenting the success of using double sequential external defibrillation and esmolol-induced beta blockade in RVF patients.[9,11,12] Future studies should aim to bring quantifiable data to the table regarding this combined intervention, with the end goal of creating a more proven, standardized approach to recognizing and treating RVF in patients with myocardial infarction.

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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Case Report: 2-PAM or not 2-PAM

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Introduction: Organophosphates (OP) are used as pest control agents worldwide and have been seen in accidental and intentional poisonings.

Case Report: A patient presented after intentional ingestion of the OP Orthene (50% acephate). Due to copious secretions, the patient was intubated and given atropine by the paramedic before transport. In the emergency department he displayed both muscarinic and nicotinic effects from OP ingestion. The patient was given multiple doses of atropine and a pralidoxime bolus. He was extubated and transferred to psychiatry two days later.

Conclusion: Acute OP exposure is a rare but complex presentation in the United States. In the United States there are bans on several organophosphate varieties, which have reduced the number and severity of OP toxicities. Acephate is generally considered a safer OP by United States regulators and the World Health Organization. In this case report, we describe an OP exposure with marked symptoms requiring intubation and successful treatment with atropine and pralidoxime. We also discuss the role of oximes in acephate toxicity. [Clin Pract Cases Emerg Med. 2025;19(2):203-206.]

Keywords*: acephate; organophosphate toxicity; pralidoxime; case report.*

INTRODUCTION

Organophosphates (OP) are chemicals commonly used as pest control agents throughout the world. Organophosphate exposures have declined rapidly in the United States since the implementation of the Food Quality Protection Act in 1996.[1] Although a rare occurrence in the US, emergency physicians should be aware of how to recognize and treat OP poisonings.

Orthene is a "fire ant killer" comprised of 50% acephate and a proprietary mixture. It is a fine powder that can easily be bought without license or regulation in the US at various stores and online. Acephate is a dimethyl OP with weak activity on the acetylcholinesterase enzyme.[2] Acephate is generally considered a less toxic OP by the World Health Organization (Class II: Moderately Hazardous) and the US Environmental Protection Agency, which recently has proposed loosening environmental limits for the substance.[3,4] A small number of case reports have been published describing acute acephate ingestions with severe cholinergic symptoms requiring atropine and oxime administration.[5]

CASE REPORT

A 33-year-old, Burmese-speaking male patient presented to the emergency department (ED) by emergency medical services (EMS) after a neighbor called 9-1-1 upon finding a suicide note. His medical history was notable for a past suicide attempt. A witness noted that the patient had drunk an undetermined quantity of acephate in the form of the fire ant killer, Orthene. The EMS responders reported that the patient was fully oriented upon their arrival. However, he appeared diaphoretic, tachypneic, and was complaining of abdominal pain. Soon after their arrival, the patient's mental status declined. He also developed copious secretions, vomiting, diarrhea, lacrimation, incontinence of urine, and spasms.

His blood glucose was 126 milligrams per deciliter (mg/

dL) (reference range: 70-110 mg/dL). His initial vital signs on scene were as follows: blood pressure, 152/97 millimeters of mercury (mm Hg); heart rate, 93 beats per minute (bpm); respiratory rate, 22 breaths per minute; and oxygen saturation, 89% on room air. His temperature was not recorded. Intravenous (IV) access was obtained. Attempts to administer oxygen via nasal cannula and to suction secretions yielded no improvement in the patient's respiratory status. The EMS responders progressed to administering 2 milligrams (mg) of IV atropine and intubated the patient. En route the patient received a total dosage of 4 mg of midazolam for agitation.

Upon arrival to the ED, the patient was increasingly agitated, necessitating 4 mg more of midazolam. His first vital signs in the ED were blood pressure 118/82 mm Hg, heart rate 101 bpm, and oxygen saturation 98% on the ventilator. His initial exam was notable for dry skin, clear lung fields bilaterally without secretions, sinus tachycardia, and no further diarrhea or lacrimation. However, he did have miosis and fasciculations. In conjunction with poison control the decision was made to infuse 2 grams of pralidoxime. He was also given an additional 2 mg of atropine IV. The hospital did not have additional pralidoxime, and the decision was made not to start an infusion as the patient had begun clinically improving rapidly. He was admitted to the intensive care unit and did not require any additional pralidoxime.

The patient's serum laboratory evaluation was notable for glucose160 mg/dL; serum osmolality, 355 milliosmoles per kilogram (mOsm/kg) (reference range: 280-300 mOsm/kg); anion gap, 18 (2-11); serum bicarbonate, 18 millimoles per liter (mmol/L) (22-30 mmol/L); potassium, 2.3 mmol/L (3.7-5.0 mmol/L); lactate, 4.2 mmol/L (0.5-1.9 mmol/L); and creatine kinase, 274 units/L (U/L) (25-257 U/L). The serum drug screen resulted with an ethanol level of 169 mg/dL (0-10 mg/dL). The urine drug screen was positive for benzodiazepines, cocaine, and acetone. Red blood cell cholinesterase level was 8,273 international units/L (IU/L) (9,572-15,031 IU/L) shortly after presentation. Plasma cholinesterase level was 252 IU/L (3,334-7,031 IU/L).

On hospital day two, the patient was extubated after it was deemed that he had no evidence of continued central weakness and had not required additional atropine. He was observed in the intensive care unit for another 48 hours without incident and then transitioned to an inpatient psychiatric service.

CPC-EM Capsule

What do we already know about this clinical entity?
Organophosphates (OP), like acephate, are used as pest control agents worldwide and have been seen in accidental and intentional poisonings.

What makes this presentation of disease reportable?
This case describing the successful management of an acephate ingestion is important as human toxicokinetic and toxicodynamic data for acephate are lacking.

What is the major learning point?
Despite its reported safety profile, acephate, an OP insecticide, can still result in severe presentations.

How might this improve emergency medicine practice?
In the United States, an OP toxidrome is a rare but complex presentation. This case describes the successful treatment of acephate toxicity with atropine and pralidoxime.

DISCUSSION

Organophosphate ingestion can be accidental or intentional. Exposure to OPs is far more common in developing countries and nations, particularly those with robust agricultural industries. Ingestion of OPs has long been a well-known and commonly used method of suicide in several Asian countries including Sri Lanka, India, and China.[6] A combination of changing farming practices and regulations have led to decreased availability of OPs in the US; therefore, exposures presenting to the ED are rare.

Organophosphates competitively inhibit acetylcholinesterase (AChE) throughout the body. This prevents the hydrolysis and inactivation of acetylcholine (ACh), which leads to accumulation of ACh at synapses.[7] Clinical signs from excess ACh are linked to the receptors involved, muscarinic and nicotinic.[7] These symptoms include diarrhea, urination, miosis, bradycardia, bronchorrhea, bronchospasm, emesis, lacrimation, and salivation. The nicotinic symptoms involve mydriasis, tachycardia, hypertension, and seizures. Additional effects include fasciculations or weakness, with severe poisoning causing paralysis.[8] This specific patient had many of the above-mentioned symptoms and fasciculations noted in the ED. Once OPs are bound to the active site of AChE, one of two processes occurs. Either the OP and AChE dissociate and the AChE recovers, or the OP forms a covalent bond with the AChE, permanently inactivating it. Recovery of AChE in OP-poisoned patients may take days to weeks.[7]

There is no laboratory test that will significantly alter management of OP toxicity from the ED. Red blood cell (RBC) and plasma cholinesterase levels can be obtained but will generally take days to return results. Plasma cholinesterase may drop faster in acute toxicity, but other

disease processes can affect plasma cholinesterase results. A drop-in RBC cholinesterase activity is more specific for OP toxicity and correlates better with clinical weakness but may take longer to become apparent following acute poisoning. Our patient had a relatively normal RBC cholinesterase with a significantly low plasma cholinesterase (less than one-tenth reference range), which was likely due to early timing of labs in the setting of an acute OP toxicity.

Management of OP toxicity involves treating both the muscarinic and nicotinic effects. Decontamination is critical. Clothes with OP on them should be removed immediately, and patients should be washed with soap and water. Emesis from OP-poisoned patients should be contained, covered, and separated from healthcare workers as ingested OP can "off gas" from the emesis.[10] Antidotal therapy includes atropine and an oxime.[8] In the US, the typical oxime used is pralidoxime (2-PAM). Pralidoxime works by reactivating inhibited AChE that has not completed the aging process and should be initiated as soon as there is suspicion of OP ingestion.[11] The rate of aging for each OP is different and varies widely. The time to aging for acephate is not known.

Pralidoxime given after the aging process will be ineffective.[11] A typical loading dose is one to two grams intravenously (20-50 mg/kg in pediatrics) bolus over 15-30 minutes with various strategies of intermittent or continuous dosing for continued weakness. Pediatric continuous infusion recommended at 10-20mg/kg/hour per the FDA however controversy exists regarding optimal adult infusion dosing.[12] Oximes may provide alternate benefits beyond preventing the aging process. They also may help reduce nicotinic symptoms and reduce the total dose of atropine required for stabilization. Controversy exists about the utility of 2-PAM and oximes in general in the treatment of OP toxicity. Our patient received only a loading dose at two grams with no infusion. His weakness and altered mental status improved on day two without additional atropine or oxime. He also had no delayed weakness, which is sometimes attributed to underdosing oximes. We suspect that pralidoxime did not make a significant impact on his recovery time since the dose was too low based on prior studies.[13] That would mean the acephate probably dissociated from the AChE, and he recovered spontaneously. It is also possible that the aging for acephate is so weak that even an inadequate dose was able to interrupt the process. Regardless, not enough is known about acephate to determine whether oximes play a major role in acephate ingestions.

CONCLUSION

Human toxicokinetic and toxicodynamic data for acephate is lacking. The overall chemical profile of acephate suggests a safer product, but we report a severe presentation of acephate toxicity treated successfully with atropine and pralidoxime. It is important for physicians to know that acephate, despite its safety profile, can still result in severe presentations. Emergency physicians are rarely exposed to OP-toxicity patients in the US and should be vigilant for patients presenting with symptoms of a cholinergic toxidrome. The need for oximes in the management of acephate toxicity is questionable and needs further study.

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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Quincke Triad and Hepatic Artery Pseudoaneurysm Presenting to the Emergency Department: A Case Report

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Introduction: Hepatic artery aneurysms are exceedingly rare, often asymptomatic, and usually diagnosed when patients present with complications such as rupture or bile duct obstruction.

Case Report: This report describes a 70-year-old female who presented to the emergency department with Quincke triad (epigastric pain, obstructive jaundice, and gastrointestinal bleeding) and was diagnosed with multiple hepatic artery pseudoaneurysms with a thrombosed fistulous connection to the biliary system. She was treated effectively with extensive embolization and biliary stenting.

Conclusion: This case underscores the importance of early diagnosis and highlights the role of multidisciplinary intervention in preventing life-threatening complications from hepatic artery aneurysms. [Clin Pract Cases Emerg Med. 2025;19(2):207-210.]

Key Words: *hepatic artery pseudoaneurysm; endovascular repair.*

INTRODUCTION

Hepatic artery aneurysms (HAA) occur in 0.002-0.4% of the population and account for 14-20% of all visceral artery aneurysms.[1,2] Most HAAs are asymptomatic, with 60-80% of patients not diagnosed until complications such as rupture, bleeding, or obstructive jaundice from bile duct compression arise.[3] The most common presentation is Quincke triad (epigastric pain, obstructive jaundice, and gastrointestinal [GI] bleeding), which is present in approximately 56% of cases.[4] Here we report a rare case of an elderly female patient presenting with Quincke triad who was found to have complex common, proper, and right hepatic artery pseudoaneurysms with a thrombosed fistulous connection to the biliary tree. Pseudoaneurysms are collections of blood that form outside the blood vessel but are contained by connective tissue, whereas true aneurysms are permanent bulges that form in all three layers of the vessel. This pseudoaneurysm was managed effectively with extensive embolization and biliary stenting.

CASE REPORT

A 70-year-old female with a remote history of a cholecystectomy (performed over 14 years prior) presented to the emergency department (ED) via ambulance for hematemesis. She had experienced a week of intermittent nausea and vomiting, culminating in a single episode of frank red blood in her emesis. Emergency medical services reported hypotension in the field, with a systolic blood pressure of 60 millimeters of mercury (mm Hg), and bradycardia ranging between 50-60 beats per minute. Following administration of one liter of normal saline in the prehospital setting, her systolic blood pressure improved to 100 mm Hg.

Upon arrival at the ED, the patient had blood pressure of 102/69 mm Hg, a heart rate of 59 beats per minute, a respiratory rate of 18 breaths per minute, a temperature of 36.3 °Celsius, and room air oxygen saturation of 97%.

Examination revealed dried blood in the oropharynx, scleral icterus, jaundice, and tenderness in the epigastric region. Initial laboratory results showed a mild leukocytosis of 11,490/milliliter (mL) (reference range: 4,000-11,100/mL), a hemoglobin level within normal limits at 14.0 gram (g) per deciliter (dL) (12.1-16.3 g/dL), an elevated serum creatinine of 1.14 milligrams (mg) per dL (0.60-1.20 mg/dL) with a baseline of 0.8 mg/dL from two months prior, and significantly increased liver enzymes with a total bilirubin of 5.4 mg/dL (0.1-1.3 mg/dL), an alkaline phosphatase of 464 international units (IU)/liter (L) (39-117 IU/L), an alanine aminotransferase of 1,152 IU/L (7-52 IU/L), and an aspartate aminotransferase of 507 IU/L (12-39 IU/L). Computed tomography with angiography (CTA) of the abdomen and pelvis identified a 3.1-centimeter saccular pseudoaneurysm of the extrahepatic artery with intraluminal thrombus and severe stenosis of the hepatic artery (Image 1). Mild intrahepatic biliary ductal dilation suggested biliary obstruction, likely due to compression. The CTA did not identify a clear source of the GI bleeding.

A transfer was requested to a facility with endovascular capabilities for definitive management. Before transfer, the patient had one episode of hematochezia but remained hemodynamically stable. Prior to departure, she received 4 mg of intravenous (IV) ondansetron, 80 mg of IV pantoprazole, and a liter of lactated Ringer. Upon arrival at the receiving facility, her clinical condition remained unchanged. General surgery, interventional radiology (IR), and gastroenterology were consulted, and she was promptly taken to the IR suite for embolization.

Gastroenterology was consulted regarding the administration of ceftriaxone and octreotide given biliary pathology with concern for GI bleeding but ultimately recommended against these medications, as there was low concern for esophageal varices given abrupt onset of jaundice and absence of risk factors such as cirrhosis. The general surgery team recommended IR and GI intervention and, if unsuccessful, they suggested hepatobiliary surgical consultation to evaluate for liver transplant.

Interventional radiology review of CTA imaging revealed extensive aneurysm extending from the common hepatic artery to the right hepatic artery deep into the hepatic hilum with the proper and right hepatic artery components partially thrombosed. Additionally, the patient was noted to have some intrahepatic biliary dilation, and the common bile duct appeared to be of higher density than usual, suggesting presence of blood within it (Image 1).

The patient's GI bleeding was attributed to a suspected erosion of a HAA into the biliary system, forming a fistulous connection. Due to the presence of a large aneurysm, the patient was taken directly for angiography and embolization. Selective angiography of the vessels revealed an irregular aneurysmal celiac trunk with high-grade irregular stenosis of the common hepatic artery ostium (Image 2).

CPC-EM Capsule

What do we already know about this clinical entity?
Hepatic artery aneurysms are rare and often asymptomatic, but they can present with life-threatening complications such as rupture, bleeding, or biliary obstruction.

What makes this presentation of disease reportable?
This case highlights a rare case of hepatic artery pseudoaneurysms with a thrombosed arterial-biliary fistula that was managed effectively with embolization and stenting.

What is the major learning point?
Early recognition of hepatic artery aneurysm is critical and often lifesaving and requires prompt identification and multidisciplinary intervention.

How might this improve emergency medicine practice?
Maintain high suspicion for hepatic aneurysms in gastrointestinal bleeding with painful jaundice, especially with a history of prior cholecystectomy.

There was also extensive aneurysmal common hepatic, proper hepatic, and right hepatic arteries with intervening areas of stenosis. Additionally, there was irregular outpouching of the distal right hepatic artery adjacent to the

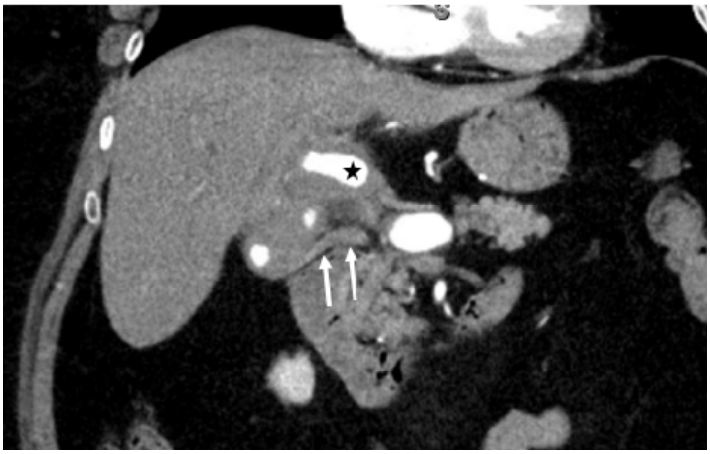


Image 1. Coronal image of abdominal computed tomography with angiography showing a hepatic artery aneurysm (star) with an associated high-density common bile duct (arrows), indicating thrombosis in the biliary system.

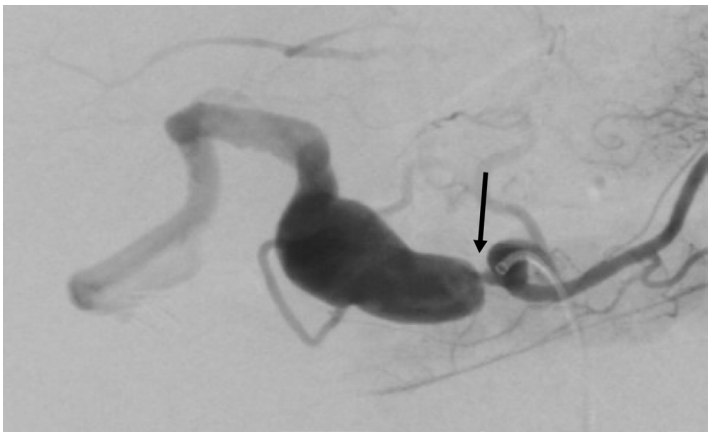


Image 2. Complete angiography demonstrating tight stenosis of the proximal common hepatic artery (arrow).

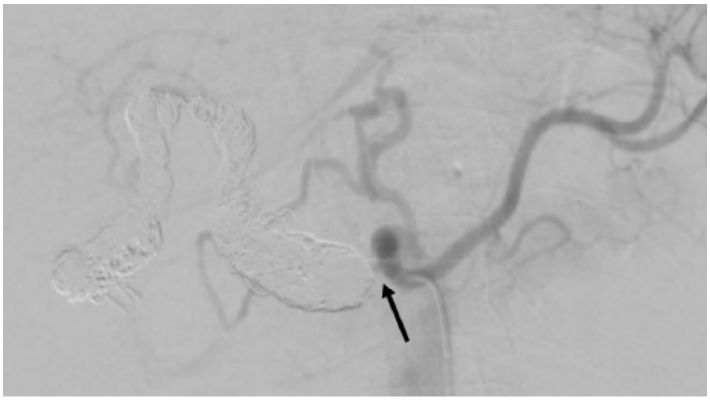


Image 3. Complete angiography demonstrating complete thrombosis of the extensive aneurysmal hepatic artery (arrow indicating inception point) with preserved arterial flow in the splenic artery and gastrohepatic artery.

cholecystectomy clips, possibly representing thrombosed fistula to the biliary tree (Image 3).

During embolization, over 60 coils were deployed to achieve hemostasis. At the conclusion of the procedure, there was complete thrombosis of the extensive aneurysmal common hepatic, proper hepatic, and right hepatic arteries. Notably, arterial flow to the left hepatic lobe was preserved due to variant origin from the left gastrohepatic trunk and, after embolization, supply to the right hepatic lobe was also preserved through left-to-right intrahepatic shunting. There were no immediate operative complications.

On hospital day two, the patient underwent an endoscopic retrograde cholangiopancreatography (ERCP). Contrast revealed diffuse dilation of the hepatic and intrahepatic ducts upstream of a stenosis. Filling defects in the bile ducts, believed to be sludge, were identified, and a balloon was used to sweep clots and old blood from the ducts, confirming the original diagnosis. A biliary sphincterotomy was performed, and a temporary, covered metal biliary stent was placed to address the narrowing in the common hepatic duct. Excellent flow was observed after stent placement.

The patient was discharged on hospital day three with down-trending liver enzymes, improving serum creatinine, and plans for a repeat ERCP in two months.

DISCUSSION

Hepatic artery aneurysms and pseudoaneurysms are infrequent, accounting for a fraction of all visceral artery aneurysms, and are often asymptomatic until they present with complications such as rupture or bile duct obstruction.[1,2] The reported mortality of a ruptured HAA is 80-100%, making its prompt recognition and treatment critical to patient survival.[1] This case highlights the importance of considering an arterial-biliary fistula from a HAA when patients present with GI bleeding in the absence of other clear causes.

True aneurysms are usually found as a single extrahepatic lesion, whereas pseudoaneurysms are normally found as multiple intrahepatic lesions, such as in this case.[4] While the terms aneurysm and pseudoaneurysm were used interchangeably and variably among specialists on the case, the patient's remote history of a laparoscopic cholecystectomy was her strongest risk factor for the development of pseudoaneurysms. Albeit incredibly rare with the incidence of vascular complications after a cholecystectomy at 0.3%, a recent review of 135 cases of hepatic pseudoaneurysms found that 28% of all cases were associated with prior laparoscopic cholecystectomies.[5] This makes hepatic artery pseudoaneurysm a diagnosis to consider on the painful jaundice differential list, especially in a patient with a history of a laparoscopic cholecystectomy. This same study found that 31% of hepatic pseudoaneurysms were secondary to a history of percutaneous transhepatic biliary drainage, and 13% were associated with a history of an open cholecystectomy.[5] The presence of these risk factors in a patient's history should add hepatic artery pseudoaneurysm to the differential list, although the rarity of the condition puts it lower.

Although an appropriate initial imaging evaluation of painful jaundice is an abdominal ultrasound per the American College of Radiology, the rare nature of this condition has not provided enough data for calculation of specificity and sensitivity of this modality in the diagnosis of HAA.[6] The gold standard imaging to identify a HAA is digital subtraction angiography, as determined in a subset of liver transplant patients for whom hepatic aneurysms are more common postoperatively.[7] However, this is not likely to be a viable option for diagnosis in most EDs. Another study found that hepatic arterial complications in a subset of liver transplantation patients diagnosed by ultrasound are often nonspecific and need further categorization with computed tomography imaging.[8] However, due to the infrequency of HAAs, specific guidelines are not present. Notably, CTA has

excellent diagnostic accuracy for detecting HAAs with a sensitivity and specificity of 100%. Given the availability of this imaging modality the ED, it should be prioritized in patients with concern for abdominal aneurysms.[3] While a right upper quadrant ultrasound in the stable patient presenting with obstructive jaundice may be an appropriate starting point, clinicians should have a low threshold to escalate to CTA if the cause of the jaundice is not explained by ultrasound findings and especially if it is accompanied by any GI bleeding.

Once an HAA is identified, there is a lot of variability in the treatment modalities due to the infrequency of the condition and the publication of only single-case studies or metanalyses of case studies to guide treatment. Guidelines from the Society of Vascular Surgery recommend an endovascular-first approach to all hepatic aneurysms and pseudoaneurysms, and endovascular embolization is the most reported approach. Roughly 37% of all reported cases of HAA used coil embolization for treatment.[9] Alternative treatment options are higher risk up to the need for liver transplantation.

While this patient remained hemodynamically stable during her ED and inpatient courses, the potential for instability underscores the importance of identification of this vascular pathology and proper consultation and transfer to a facility with IR. Case reports of patients who do present with hemorrhagic shock due to HAA rupture offer additional stabilization modalities, including one successful case using a resuscitative endovascular balloon occlusion of the aorta.[10] Other treatment options include exploratory laparotomy with excision, ligation, and repair with arterial grafting and hepatic resections.[12] Getting surgical and interventional radiological specialists on board early is paramount to patient survival in the event of HAA rupture.

CONCLUSION

This case highlights the rarity and clinical complexity of hepatic artery pseudoaneurysms, particularly in the context of biliary complications. The patient's presentation with Quincke triad and the subsequent discovery of multiple aneurysms and a thrombosed fistula underscore the importance of early recognition and multidisciplinary intervention in preventing life-threatening outcomes. The successful management through extensive embolization and biliary stenting, preserving hepatic function, emphasizes the critical role of getting interventional radiology and gastroenterology on board early. This report further advocates for heightened clinical suspicion of hepatic artery pseudoaneurysms, especially in patients with a history of laparoscopic cholecystectomy.

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

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Retroperitoneal Necrotizing Fasciitis Following Prolonged Physical Activity: A Case Report

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Introduction: Retroperitoneal necrotizing fasciitis is a rare, rapidly progressive, and often fatal infection of the retroperitoneum. In many cases the source of infection is unclear, and cutaneous signs of necrotizing fasciitis may be absent.

Case Report: We present the case of a 64-year-old female with a history of hypertension, hyperlipidemia, and breast cancer who developed acute kidney injury (AKI) and retroperitoneal necrotizing fasciitis following a 20-mile bike ride. The patient's initial symptoms included severe muscle aches, nausea, vomiting, and flank pain. Diagnostic imaging and laboratory results indicated myositis and severe AKI. Despite aggressive treatment with antibiotics, intravenous fluids, and pain management, the patient developed septic shock and multiorgan failure, ultimately leading to her death.

Conclusion: This case highlights the rapid progression and complexity of managing necrotizing fasciitis and AKI in the context of rhabdomyolysis. Early recognition and aggressive management are crucial in cases of suspected necrotizing fasciitis and AKI. Patients may not initially present with cutaneous findings suggestive of necrotizing fasciitis. Early involvement of a multidisciplinary team can improve patient outcomes in complex and rapidly deteriorating patients. [Clin Pract Cases Emerg Med. 2025;19(2):211-214.]

Keywords*: acute kidney injury; retroperitoneal necrotizing fasciitis; necrotizing fasciitis; pyomyositis; rhabdomyolysis.*

INTRODUCTION

Retroperitoneal necrotizing fasciitis is a rare, rapidly progressive and often fatal infection of the retroperitoneum. This infection has a high mortality rate, estimated to be 40-60%.[1] In many cases the source of infection is unclear. A systematic review of peritonitis caused by streptococcus found that 69% of patients developed the infection from an unknown source, 16% of patients' source of infection was from ascending vaginal infection, 9% from a droplet infection, and 6% from pharyngitis.[2]

Cutaneous manifestations of necrotizing fasciitis, such as necrosis and erythema, will often present in patients with a defined infectious source such as a wound or surgical site. However, according to another study, more than half of patients with necrotizing fasciitis had no defined portal of entry and arrived to the emergency department (ED) with the main symptom of increasingly severe pain. This pain can begin at a site of recent trauma such as a joint injury, hematoma, or muscle strain.[3]

CASE REPORT

A 64-year-old female with a history of hypertension managed with lisinopril, hyperlipidemia, and breast cancer status post mastectomy and current letrozole use presented to the ED with severe muscle aches, nausea, vomiting, diarrhea, decreased urine output, frontal headache, and flank pain. She was physically

active most days of the week including walking, running, and occasional bicycle riding. Four days prior to arrival, the patient had completed a challenging 20-mile bike ride on a summer day. Three days prior to arrival, she developed severe muscle aches. One day before arrival, she experienced diarrhea, nausea, vomiting, and headache. The patient came to the ED due to these symptoms, in addition to left flank pain and decreased urine output. (See Figure for a timeline of patient illness.)

The patient had never smoked or used smokeless tobacco. She consumed about 10 alcoholic drinks per week. She had a family history of hypertension. She had recently traveled from Utah to Arizona after her biking trip. On presentation, she appeared anxious with a heart rate of 70 beats per minute and respirations of 20 breaths per minute; she was afebrile at 36.6 °Celsius and hypertensive to 138/99 millimeters of mercury. Her oxygen saturation was 100% without supplemental oxygen use. The patient rated her flank pain a 10/10. On physical examination, the patient had a normal cardiovascular and pulmonary exam. There was moderate pain to palpation of the left middle and left lower flank, with mild right lower quadrant abdominal tenderness.

Initial laboratory results indicated a white blood cell count (WBC) of 6.1 $\times 10^9$ cells per liter (L) (reference range: 4.5-11.0 $\times 10^9$/L) and hemoglobin of 13.4 grams per deciliter (g/dL) (11.6-15.0 g/dL). She had a sodium of 129 millimoles (mmol) per L (135-145 mmol/L), chloride 92 mmol/L (98-107 mmol/L), bicarbonate 18 mmol/L (22-29 mmol/L), and anion gap of 19 (7-15). She had a severe acute kidney injury (AKI) with a creatinine of 4.25 milligrams (mg)/dL) (patient baseline 0.8, reference range 0.59-1.04 mg/dL) and an elevated blood urea nitrogen of 45 mg/dL (6-21 mg/dL). Her lactate was elevated to 4.1 mmol/L (0.5-2.2 mmol/L) and creatine kinase (CK) of 200 units (U)/L (2-192 U/L). Influenza A, influenza B, respiratory syncytial virus, and coronavirus polymerase chain reaction tests were negative.

Diagnostic imaging with computed tomography of the abdomen and pelvis without contrast due to her kidney injury revealed heterogeneous hyperattenuation and asymmetric enlargement of the left quadratus lumborum muscle, external oblique muscle, left psoas major muscle, and iliacus muscle (Image 1).

The patient was admitted to internal medicine for treatment of her AKI, elevated lactic acid, and pain, and she was treated with aggressive antibiotics including cefepime 1

CPC-EM Capsule

What do we already know about this clinical entity?
Retroperitoneal necrotizing fasciitis is a rapidly progressive disease associated with a high morbidity and mortality.

What makes this presentation of disease reportable?
This is a rare case of retroperitoneal necrotizing fasciitis without a clear infectious source and an initially non-specific disease presentation.

What is the major learning point?
Retroperitoneal necrotizing fasciitis does not always have a clear infectious source and should be considered when in the setting of severe pain or organ failure.

How might this improve emergency medicine practice?
Early recognition of retroperitoneal necrotizing fasciitis requires a broad differential and recognition of rapidly progressive multisystem organ failure.

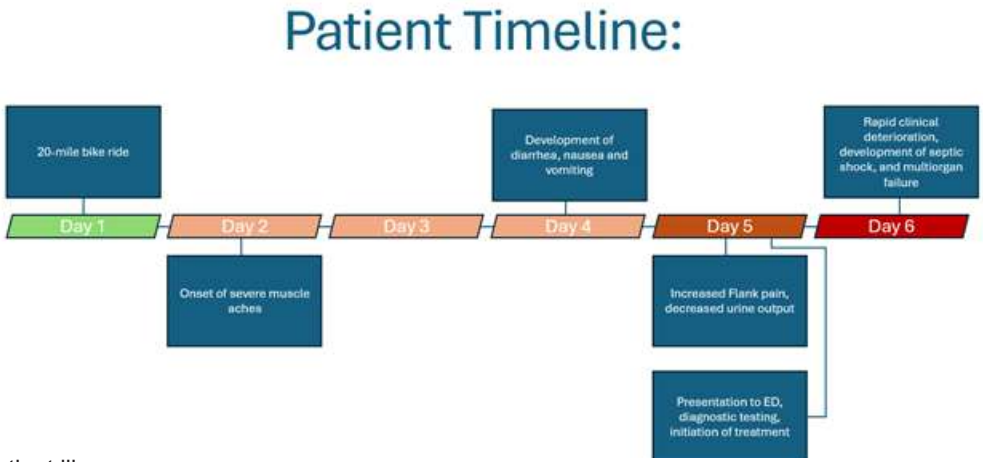


Figure. Timeline of patient illness.
ED, emergency department.

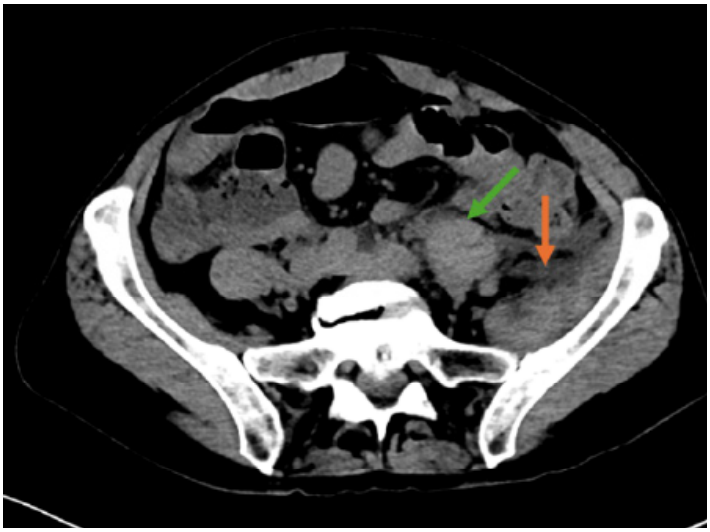


Image 1. Lower sections of computed tomography abdomen and pelvis demonstrates heterogenous hyper-attenuation of the left psoas major muscle (green arrow) and iliacus muscle (orange arrow).

gm intravenously (IV), doxycyline 100 mg IV, linezolid 600 mg IV, and metronidazole 500 mg IV. She was also treated with IV fluid resuscitation and pain management with morphine 2 mg and multiple 1 mg doses of hydromorphone. She was also given hydrocortisone 50 mg and IV immune globulin (human) 10% infusion 20 g. The patient started continuous renal replacement therapy due to her AKI and subsequent severe metabolic acidosis.

Five hours after admission, the patient developed rapidly progressive multiorgan failure that presented as a possible stroke, after which she was intubated, and additional imaging was obtained (Image 2). Her CK increased from 200 to 5,535 U/L, serum creatinine from 4.25 to 4.41 mg/dL, blood urea nitrogen from 45 to 51 mmol/L, and WBC decreased from 6.9 to 3.7 × 10^9 cells/L.

Surgical consultation was obtained due to suspicion of necrotizing fasciitis, for which the patient was deemed inoperable due to the extensive organ involvement of the infection. A muscle biopsy was obtained and confirmed to be group A *Steptococcus pyogenes*. The patient was transitioned to comfort care after family discussions of these findings. She died shortly thereafter due to multiorgan failure and septic shock.

DISCUSSION

In patients with no defined infectious source, the severe pain associated with necrotizing fasciitis can precede cutaneous evidence of infection by 12-24 hours. In our case, the patient presented with increasingly severe left flank pain followed by cutaneous findings of necrotizing fasciitis after admission to the hospital. She did not present with an increased WBC on initial presentation, possibly due to her use of letrozole for breast cancer.

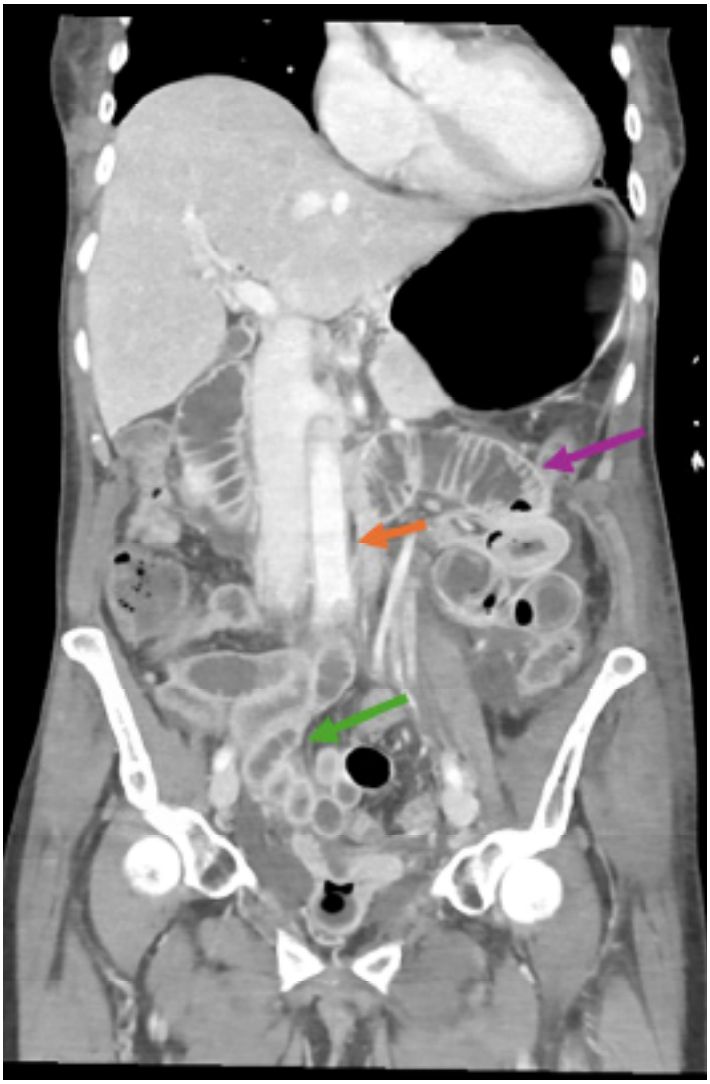


Image 2. Post contrast computed tomography abdomen and pelvis performed one day after the initial presentation demonstrated the muscular findings; in addition, there was new diffuse thickening of the aorta concerning for aortitis (orange arrow), new proximal small bowel dilatation with areas of thickening (purple arrow) and the distal small bowel hyper-enhancing wall (green arrow) concerning for sequelae of active septic shock.

The treatment of necrotizing fasciitis includes early recognition, administration of broad spectrum IV antibiotics, and surgical debridement.[4] Retroperitoneal necrotizing fasciitis is a rare, aggressive infection associated with a high mortality. A case report of a young, healthy, 33-year-old female patient is similar to our case. She presented to the ED with generalized abdominal pain suspected to be due to gastroenteritis, and a CT demonstrated intrabdominal fluid likely secondary to a ruptured corpus luteal cyst. The patient returned with worsening pain, with repeat CT demonstrating worsening free fluid and evidence of peritonitis. Subsequently laparoscopy confirmed peritonitis with no identified infectious source, with cultures positive for *group A S. pyogenes*.[5]

Intra-abdominal necrotizing fasciitis should be part of a broad differential considered in the ED to reduce the mortality and morbidity associated with this condition. One tool is the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score to distinguish necrotizing fasciitis from severe cellulitis or abscess, which takes into account C-reactive protein, WBC, hemoglobin, sodium, creatinine, and glucose.[6]

In another case, a 33-year-old man had a three-week history of back ache due to trauma, followed by fever, vomiting, and severe left flank pain, which was then diagnosed as retroperitoneal necrotizing fasciitis due to *Escherichia coli*. The source for this patient was unclear, similar to our patient.[7] However, our patient had gone mountain biking for 20 miles, which could have been a source of trauma and back pain for her. In cases of trauma and hematoma formation, necrotizing fasciitis has been known to infect these areas. For example, a 26-year-old man presented with severe pain in his right biceps, which he attributed to a muscle tear, and was diagnosed as a muscle belly tear with hematoma formation. The patient returned four days later with necrotic skin and erythema of the upper extremity and grew group F β-hemolytic streptococci and *Bacteroides*.[8]

In our case the source of the patient's retroperitoneal necrotizing fasciitis remains unclear, but contributing factors included her recent strenuous bike ride and use of letrozole for breast cancer. Kejela et al described 14 cases of retroperitoneal necrotizing fasciitis, with infection sources ranging from Fournier gangrene, fistula, and abscess to perforated diverticulitis, renal stone, and "none."[9]

CONCLUSION

This case highlights the rapid progression and complexity of managing necrotizing fasciitis and acute kidney injury in the context of rhabdomyolysis. Early recognition and aggressive management are crucial in cases of suspected necrotizing fasciitis and AKI. Early involvement of a multidisciplinary team can improve patient outcomes in complex and rapidly deteriorating 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.

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Thoracic Outlet Syndrome Case Report: Appropriate Diagnosis Can Expedite Patient Treatment and Prevent Negative Outcomes

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Introduction: Thoracic outlet syndrome (TOS) is a diagnosis classifying upper extremity symptoms caused by compression of the neurogenic and vascular structures between the clavicle and first rib. It is important to promptly decompress these structures to prevent long-term deficits and poor patient outcomes. However, TOS often presents in unique ways with substantial symptom variance, making it difficult to identify, diagnose, and promptly treat. Compounding this, common diagnostic tools such as magnetic resonance imaging are not independently appropriate for a conclusive diagnosis of TOS. Patients with TOS can initially present acutely due to symptom exacerbations or emergent situations, necessitating multimodal diagnostic methods and early TOS recognition to improve patient outcomes, particularly in emergency department (ED) settings.

Case Report: A 22-year-old male presented with chronic symptoms of numbness and weakness in his right hand in addition to chest pain that radiated into his right elbow, along with a diminished right radial pulse. The patient also suffered from acute symptomatic exacerbations of total arm asthenia, paresthesia, and what the patient described as "an intensely cold hand" during football practice. He was eventually treated with a right first-rib resection to decompress the brachial plexus, which resulted in complete symptom resolution and recovery.

Conclusion: Due to the serious long-term complications associated with uncorrected brachial plexus compression and the fact that TOS patients can initially present to ED settings with acute exacerbations, it is important for emergency clinicians to be able to recognize and either treat or appropriately refer patients for treatment. The ED is equipped to enable physicians to perform a comprehensive diagnostic assessment because they often have access to the diagnostic modalities necessary for diagnosing thoracic outlet syndrome. [Clin Pract Cases Emerg Med. 2025;19(2):215-219.]

Keywords: *case report; thoracic outlet syndrome; emergency medicine.*

INTRODUCTION

The development of thoracic outlet syndrome (TOS) can arise from anatomic variation, repetitive motions, trauma, or malignancy.[1] Trauma and bleeding can lead to direct compressive forces on neurovascular structures followed by post-injury fibrosis that can create chronic constrictive force, causing the symptoms of TOS.[1] Consequently, motor-vehicle collisions and midshaft clavicular fractures are common examples of underlying causes of TOS.[1] Repetitive motions can cause muscle hypertrophy that engenders neurovascular

compression particularly in patients who experience overuse injuries, resulting in insidious small hemorrhages and microfibrosis.[1] Anatomic osseous and muscular variation cause direct compressive force on neurovascular structures, with the most notable examples being the presence of a cervical rib and congenital supernumerary scalene muscles.[1] A cervical rib is usually an asymptomatic occurrence, but it can be a predisposition for the development of TOS following a neck trauma.[2] Malignancy is another well-documented etiology of TOS, particularly in patients who develop superior pulmonary sulcus tumors.[1] These examples illustrate the utility of increased awareness of TOS in the emergency department (ED) due to the common nature of these presentations to ED settings, often making emergency physicians the initial point of medical evaluation for these patients.

CPC-EM Capsule

What do we already know about this clinical entity?
Thoracic outlet syndrome (TOS) can be difficult to diagnose due to its diverse presentation, which is dependent on arterial, venous, or neurogenic compression symptoms.

What makes this presentation of disease reportable?
A young male diagnosed with neurogenic and arterial TOS required multiple imaging studies during his diagnostic workup.

What is the major learning point?
The combination of physical exam maneuvers and imaging modalities in symptom-provoking positions could be the key to streamlining the diagnostic process of TOS.

How might this improve emergency medicine practice?
A thorough understanding of TOS and the available diagnostic modalities can expedite future patient care.

CASE REPORT

A 22-year-old male presented with chronic symptoms of numbness in his right hand in addition to chest pain radiating to his right elbow and a diminished right radial pulse. The patient also suffered from acute symptomatic exacerbations of total arm asthenia, paresthesia, and what the patient described as "an intensely cold hand" during football practice. He was a lifelong overhead-throwing athlete with a pertinent surgical history of an uncomplicated superior labrum, anterior to posterior surgical repair of his right shoulder. After plain radiographs and a magnetic resonance imaging (MRI) study of the right shoulder showed an absence of abnormal findings, the patient was left without a diagnosis. His symptoms continued to progress over the course of an additional year, motivating him to seek additional evaluation at a thoracic and vascular clinic.

The additional workup included a Doppler ultrasound vascular study and in-depth physical exam. The results of the Doppler ultrasound vascular studies demonstrated decreased distal blood flow in the right arm vasculature compared to the left, and the physical examination demonstrated a positive Adson maneuver and Roos test, leading to an ultimate diagnosis of TOS. Several weeks later, he was admitted to the hospital and underwent a right first-rib resection performed by thoracic surgery to decompress the brachial plexus, specifically, the medial division leading to the ulnar nerve along with the subclavian artery and the surrounding vasculature. After the procedure, he was discharged on hospital day two and subsequently treated with six weeks of physical therapy. The patient made a full recovery, including complete restoration of range of motion and blood flow, as well as cessation of pain and numbness. Progress was monitored at yearly postoperative visits, where his vascular supply was reassessed with arterial and venous Doppler ultrasounds along with upper extremity vascular studies to ensure complete termination of symptomatology and disease pathology.

DISCUSSION

Although the patient detailed above underwent a diagnostic process that was lengthy and included care from specialist clinics, his diagnosis was made with diagnostic studies and physical exam maneuvers that are readily available to most clinicians. A thorough understanding of TOS and the available diagnostic modalities can expedite future patient care.

Thoracic outlet syndrome was defined in 1956 to describe a spectrum of upper extremity symptoms caused by compression of the neurologic and vascular structures of the thoracic outlet.[3] There are three points of compression in TOS. The first is at the interscalene triangle, which consists of the anterior and middle scalene muscles and the first rib.[4] The symptomology seen from compression in this region is due to impingement of the subclavian artery or all trunks of the brachial plexus.[4] The second point is the costoclavicular space, which is defined by the anterior border of the clavicle, subclavius muscle, the costocoracoid ligament, the posterior border by the first rib and scalene muscles, and the lateral border by the scapula.[4] The symptomology seen from compression in this region resembles the structures described above.[4] The third point is the retropectoral space, which is inferior to the coracoid process adjacent to the second through fourth ribs, and posterior to the pectoralis muscle.[4] Compression in this region causes symptoms due to impingement of the axillary artery, axillary vein, and the

brachial plexus cords.[4]

Thoracic outlet syndrome is subcategorized based on which impinged structures are the primary cause of the patient's symptoms.[4] These categories are neurogenic TOS (nTOS), venous TOS (vTOS), and arterial TOS (aTOS).[4] Neurogenic TOS comprises 90-95% of TOS cases and is more common in women.[3,4] Neurogenic TOS symptomatology is determined by which brachial plexus structures are primarily compressed, with lower plexus compression of the eighth cervical and first thoracic nerves (C8-T1) resulting in neurological symptoms in an ulnar distribution, whereas upper plexus compression of fifth cervical and seventh cervical nerves (C5-C7) resulting in more widespread neurological symptoms that occur in a supraclavicular, upper thoracic, or radial nerve distribution.[4] In addition to these non-specific neurological findings, hand coldness and color changes (Raynaud phenomenon) are often experienced, this is caused by sympathetic overactivation from brachial plexus impingement.[4] The variety of non-specific symptoms makes nTOS diagnosis difficult.[4]

Venous thoracic outlet syndrome comprises 3-5% of TOS cases and affects young adults of both genders who do repetitive upper-arm movements. Venous thoracic outlet syndrome (otherwise known as Paget-Schröetter syndrome) is the most easily identifiable of the TOS classifications because it is characterized by drastic upper extremity swelling, indicative of subclavian vein obstruction. It can also be accompanied by cyanosis, severe pain in the upper extremity, and a feeling of arm heaviness after activity.[2,4] This obstruction is mainly due to thrombotic events that can result from repetitive vessel injury, making it most common in young, physically active individuals.[2-4]

Arterial TOS comprises less than 1% of cases. Arterial TOS is a rare condition caused by subclavian artery compression or thrombi obstruction presenting with unilateral symptoms of digital ischemia, coldness, pallor, non-radicular pain, or paresthesias localized to the hand and rarely accompanied by cervical or upper extremity symptoms.[2,4] Almost all cases of aTOS occur in patients who have a cervical rib or an anatomically unique first rib that presses on the subclavian artery.[4] If aTOS is left untreated, aneurysm formation, embolic events, and life-threatening ischemia can occur.[4]

After obtaining a history, physical exam maneuvers should be performed to diagnose TOS. The Adson maneuver is performed with the patient seated in a chair with their arms resting at their sides. The examiner then palpates the radial pulse on the affected side, while having the patient rotate their head to the same side and take a deep breath.[2,5,6] A positive Adson maneuver would reveal a decreased or absent radial pulse with the onset of paraesthesias.[2,5] The Roos test is arm elevation for three minutes with the arm abducted to 90º and elbow flexed to 90º while the patient opens and closes his or her hand rapidly (Figure).[7,8] The Roos test is positive if symptoms are reproduced.[6] The data on sensitivity and specificity for TOS physical exam maneuvers is variable; therefore, other imaging modalities and clinical suspicion play an important role.[9]

Imaging is the next step in the diagnosis of TOS. Radiographic assessment of gross anatomical abnormalities can easily identify a cervical rib or an anomalous first rib, which raises clinical suspicion for TOS (Image 1).[2] If a rib abnormality is not identified with a radiograph, it does not completely rule out TOS if the patient's symptoms are highly suggestive, but it can decrease the clinical suspicion that the symptoms are being caused by bone compression on the brachial plexus structures and increase the suspicion of another cause.[2]

Following radiograph imaging, a neck and shoulder magnetic resonance imaging (MRI) study is the appropriate next step in diagnosing TOS to evaluate the brachial plexus. Magnetic resonance imaging can have an increased positive predictive value for TOS because brachial plexus compression can be caused by constricting fibrous bands and other compressed elements in the brachial plexus. These fibrous bands and compressed elements have been noted to emit increased signals in MRI images and can increase clinical suspicion.[10] It has been shown that MRI images from TOS-positive patients demonstrate a thicker subclavius muscle and a wider retropectoralis minor space in patients with TOS compared to controls.[11] However, TOS signs and symptoms are often not provoked in the standard anatomical position with arms positioned at the sides. Imaging in a symptom-provoking position could be a key to unlocking more usefulness of MRI in TOS diagnosis.[11]

Next, performing a movement-specific imaging modality is key. A Doppler ultrasound to assess vascular flow in various movements and joint articulations or a high-frequency ultrasound

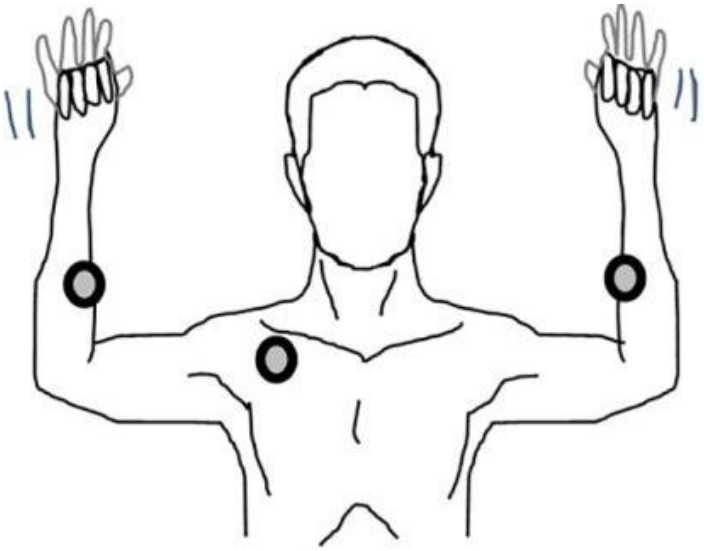


Figure. Representation of the Roos test. Circles on each arm represent regions where a diminished pulse can be felt in a positive Roos test. The circle in the right axillary region represents possible compression of the thoracic outlet. The lines around the hands exemplify rapid opening and closing of hands during the Roos test.Reprinted with permission.[8]

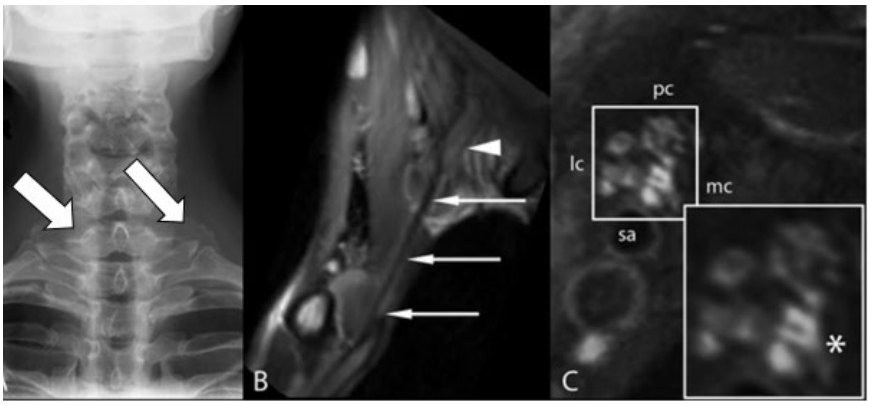


Image 1. Diagnostic imaging findings for thoracic outlet syndrome: A) demonstrates a radiograph displaying bilateral cervical ribs (arrows); B) points out the fibrous band from the cervical rib to the first thoracic rib near the inferior trunk (arrows); and C) is a T2-weighted magnetic resonance image showing increased signal near the inferior trunk (asterisk).
Reprinted with permission, with arrows added to the image in section A.[10]

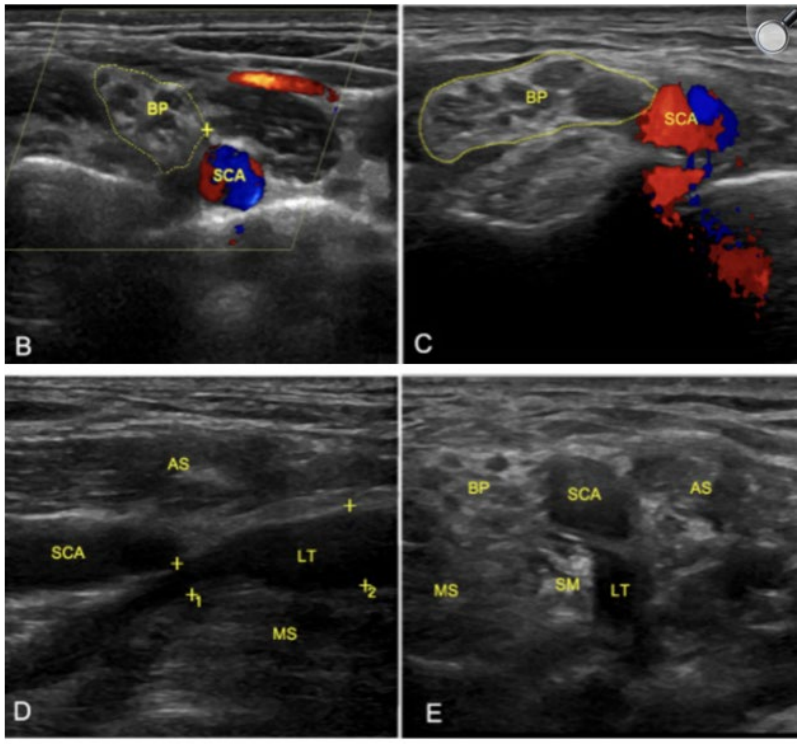


Image 2. Ultrasound of the brachial plexus with and without Doppler: B) normal and C) injured side. Compare the cross-sectional view of the brachial plexus (BP) in these images. The BP is larger on the injured side. D) Longitudinal axis showing compression of the lateral trunk (LT) by the middle scalene (MS) muscle as is denoted by the measuring markers labeled 1; and E) short-axis view of the LT compression by the scalenus minimus (SM).
SCA, subclavian artery; *AS*, anterior scalene.
Reprinted with permission.[12]

to assess nerve edema or compression in exacerbating positional states are the two main options.[12] This allows the clinician to assess positional compressions and possibly identify the specific locations of compression (Image 2).

Lastly, electrodiagnostic tests such as nerve compression and conduction can be one of the most promising diagnostic tools for nTOS. Assessing the medial antebrachial cutaneous nerve in symptomatic patients with suspected TOS showed abnormal conduction velocities in action potentials.[12] When comparing to the unaffected limb, the affected limb displayed differences in amplitudes of approximately 0.3 milliseconds. The combination of appropriate history-taking, physical exam maneuvers, and these diagnostic modalities can allow clinicians to promptly and accurately diagnose TOS,

preventing the development of adverse outcomes associated with uncorrected long-term brachial plexus compression.

CONCLUSION

Thoracic outlet syndrome is a pathology caused by compression of the brachial plexus and can cause neurologic, venous, or arterial symptoms. It can be caused by abnormal anatomic development, chronic hypertrophy, or acute events such as trauma, malignancies, or vascular thrombosis. These types of acute presentations make awareness of TOS specifically relevant to ED settings, because in these acute situations an emergency clinician is the primary point of patient evaluation. Emergency department settings are uniquely equipped to be able to mount an appropriate diagnostic plan for TOS because they often have access to the diagnostic modalities necessary for diagnosing TOS and are, therefore, able to promptly treat or refer patients to treatment, expediting patient care and preventing negative patient outcomes associated with chronic brachial plexus compression. These factors all serve to emphasize the importance of awareness and efficient diagnosis of TOS, particularly in ED settings.

Patient consent has been documented and is on file for publication of this case report.

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Catecholaminergic Polymorphic Ventricular Tachycardia in a 16-year-old: Case Report

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Introduction: Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a rare, inheritable cardiac disorder associated with stress- or exercise-induced syncope or cardiac arrest in children and young adults. Diagnosis of CPVT is often missed or delayed due to variable presentation and normal cardiac imaging and electrocardiogram results, with about 40% of patients dying within 10 years of diagnosis. This case underscores the importance of cross-departmental communication when managing complex pediatric cases, especially when using an interpreter.

Case Report: A 16-year-old male presented to the hospital with cardiac arrest in ventricular fibrillation following collapse despite a history of treatment with flecainide and nadolol. He was resuscitated, stabilized with antiarrhythmic drips, received an implantable cardioverter defibrillator, and was discharged neurologically intact nine days later. It is vital for physicians to consider CPVT in young patients with syncope to prevent errors in diagnosis of this highly fatal disease.

Conclusion: Catecholaminergic polymorphic ventricular tachycardia is a rare genetic disease with significant morbidity and mortality. Treatment decisions for acute CPVT often occur without prior knowledge of the disease; so, in patients diagnosed with CPVT, physicians should implement appropriate therapeutic options to prevent future cardiac events. For patients who remain symptomatic despite compliance with beta blockers and/or other antiarrhythmic therapy, interventions such as placement of an implantable cardioverter defibrillator or sympathetic denervation may be necessary to prevent life-threatening arrhythmias. This case also underscores the importance of obtaining a detailed family history and coordinating care with other physicians in cases where history is limited. [Clin Pract Cases Emerg Med. 2025;19(2):220-222.]

Keywords*: case report; catecholaminergic polymorphic ventricular tachycardia; arrhythmia.*

INTRODUCTION

Here we present the case of a 16-year-old male who experienced sudden cardiac arrest because of catecholaminergic polymorphic ventricular tachycardia (CPVT), an uncommon congenital and life-threatening condition. The patient was successfully resuscitated and stabilized with Advanced Cardiovascular Life Support (ACLS) and multiple cardiac medications, including a transition from amiodarone to esmolol following consultation with his cardiologist. Although CPVT is uncommon, it is crucial for emergency physicians to recognize and manage it promptly. Our case underscores the importance of early identification and appropriate intervention, which led to the patient's complete neurological recovery and successful placement of an implantable cardioverter defibrillator (ICD).

CASE REPORT

A 16-year-old boy presented to the emergency department (ED) via emergency medical services (EMS) in cardiac arrest, receiving active chest compressions. Per EMS, he had

collapsed suddenly and was found pulseless with no spontaneous respiration and cardiopulmonary resuscitation was immediately started. En route, the patient was in ventricular fibrillation (VF), defibrillated five times, and given 300 milligrams of amiodarone intravenously (IV) and epinephrine IV per ACLS protocol. He was intubated in the ED, where ACLS was continued and return of spontaneous circulation was achieved. Vitals showed a temperature 37 °Celsius, heart rate 110 beats per minute, respirations 18 breaths per minute, 100% oxygenation on the ventilator, and blood pressure 71/51 millimeters of mercury. His exam showed no trauma, a temporary airway in the oropharynx, and mechanical breath sounds bilaterally.

The patient was intubated with a 7.5 endotracheal tube and started on an amiodarone drip due to initial ventricular tachycardia. Due to hypotension, he was started on a norepinephrine drip, along with fentanyl and propofol for pain and sedation. The patient's electrocardiogram (ECG) showed wide complex tachycardia but no ST-segment elevation myocardial infarction. His mother, using an interpreter, reported he had been in a normal state of health before collapsing and had a history of "bradycardia," taking flecainide and nadolol for it. He had not been sick and was not exercising when it happened. Although an interpreter was used, history was limited due to a language barrier and limited medical literacy of the family.

His labs showed significantly elevated troponin at 344 picograms per milliliter (pg/mL) (reference range 0-40 pg/mL); aspartate aminotransferase of 341 units per liter (U/L) (8-33 U/L) and alanine transaminase of 404 U/L (7-56 U/L); potassium of 3.5 milliequivalents per liter (mEq/L) (3.4-4.7 mEq/L); magnesium of 2.6 mEq/L (1.3-2.1 mEq/L); bicarbonate of 17 mEq/L (22-29 mEq/L) , an anion gap of 23 millimoles per liter (mmol/L) (4-12 mmol/L); and negative acetaminophen, salicylates, alcohol and urine drug screen. Due to the history of flecainide use, a possible overdose was considered, and poison control recommended a bicarbonate drip due to the wide complex tachycardia. After consulting the patient's cardiologist, who revealed a history of CPVT, the amiodarone drip was changed to esmolol. They recommended strict electrolyte repletion with goal potassium greater than four mEq/L and magnesium of greater than two mEq/L. The patient was given 40 mEq of potassium chloride intravenous.

As recommended by the cardiologist, the patient was transferred to their facility via critical care ambulance. While in the pediatric intensive care unit, he was started on milrinone, lidocaine, and esmolol drips to stabilize his heart rhythm. On hospital day two he was extubated, had an ICD placed, and was weaned off the drips, starting oral nadolol and flecainide. He developed a post-ICD deep vein thrombosis, which was treated with anticoagulation. He was discharged on hospital day 10 with a completely normal neurological exam. His post-ICD stress test was normal with no arrhythmia, and he was eventually taken off anticoagulation. On subsequent follow-up visits the patient was noted to be doing well on his current medications.

CPC-EM Capsule

What do we already know about this clinical entity?
Catecholaminergic polymorphic ventricular tachycardia (CPVT) is an inherited heart condition that causes life-threatening arrhythmias, especially during periods of stress, due to a genetic mutation in calcium regulation.

What makes this presentation of disease reportable?
When CPVT causes cardiac arrest, it is usually undiagnosed. Here, it was already known, but a language barrier prevented the family from disclosing this information.

What is the major learning point?
Detailed family history, a broad differential, and interpreter use is integral in identifying rarer cardiac diagnoses to provide optimal care.

How might this improve emergency medicine practice?
Direct physician-to-physician communication and a broad differential may reduce risk of diagnostic error, improving treatment in acute cardiac settings.

DISCUSSION

Catecholaminergic polymorphic ventricular tachycardia is a rare heart condition characterized by polymorphic or bidirectional tachycardia that is mostly inherited in an autosomal dominant manner. It is often undiagnosed due to normal ECG and postmortem findings. The disease has a heterogeneous genetic basis, typically involving ryanodine or calsequestrin channel mutations. These mutations lead to increased calcium leakage from the sarcoplasmic reticulum during diastole, causing an abnormal elevation in intracellular calcium levels, which can result in VF. Episodes are often precipitated by acute exercise or emotion and can lead to syncope and potentially fatal VF. The age of onset is most frequently between 7-12 years, although there have been cases of onset in the forties. Diagnosis is defined by a structurally normal heart, normal ECG, or exercise- or stress- induced VT or those with certain genetic variants. The differential diagnosis for arrhythmogenic syncope in a pediatric patient includes Brugada syndrome, congenital prolonged QT-

interval, hypertrophic obstructive cardiomyopathy, Wolff-Parkinson-White syndrome, and arrhythmogenic right ventricular cardiomyopathy.

The true prevalence of CPVT is likely higher because these other inherited arrhythmias can at least present with resting ECG abnormalities. Treatment includes resuscitation, beta blockers, flecainide, nadolol and, in refractory cases, calcium channel blockers, ICD implantation, and left cardiac sympathetic denervation.[2] This case highlights the clinical, diagnostic, and management challenges of CPVT in pediatric patients.[3] The patient presented with cardiac arrest due to VF despite treatment with flecainide and nadolol. Physicians must recognize the nuances of this case to be able to recognize and diagnose CPVT in patients with unexplained syncope or cardiac events, even with ongoing antiarrhythmic therapy.

Catecholaminergic polymorphic ventricular tachycardia has a variable clinical presentation and no unique ECG findings. Electrocardiograms are typically normal until an arrhythmic episode occurs, showing polymorphic VT, bidirectional tachycardia, or supraventricular tachycardias.[4,5] Yet these findings can also apply to other cardiac disorders such as nonischemic cardiomyopathies.[6] Our patient had wide complex tachycardia, which suggests many other diagnoses, including Brugada syndrome and drug overdose.[6] In turn, accurate diagnosis and management depend on clinical suspicion and thorough evaluation of the patient›s history and presentation.

Our initial suspicion in this case was a potential overdose on a medication like flecainide that can induce VT, but this was a red herring. The discovery that the patient had CPVT highlights the importance of detailed family history despite communication barriers and knowledge gaps, as well as interdisciplinary collaboration to identify CPVT before it presents as cardiac arrest. We recommend direct physician-to-physician communication when possible to clarify any gaps in knowledge in patients with complex medical histories, as well as always using a licensed interpreter.

CONCLUSION

In the absence of treatment, CPVT is a highly lethal arrhythmic disorder. To maintain hemodynamic stability and minimize the risk of sudden cardiac death, physicians treating patients presenting to the ED with cardiac arrest must recognize this condition quickly. Nevertheless, patients may still experience symptoms even when diagnosed and compliant with maximally tolerated medical therapy. In describing this case, we aimed to contribute to the literature describing these cases and enhance awareness of this condition. It is critical that physicians be aware of the possibility of CPVT in cardiac arrest with apparent arrhythmic control, ask for a detailed history, and collaborate with other emergency clinicians when unable to obtain clear information from the family before arrest. A further aim was to emphasize the consideration of concurrent interventions, such as ICDs or sympathetic denervation, in high-risk patients to reduce the risk of adverse cardiac events.

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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Lidocaine for Sodium Channel Toxicity in Diphenhydramine Overdose: Case Report

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Introduction: Diphenhydramine overdose is a growing concern, particularly among adolescents influenced by online challenges. Traditionally managed with supportive care and sodium bicarbonate, severe cases may exhibit refractory symptoms due to sodium channel toxicity, necessitating alternative treatments.

Case Report: A 28-year-old male with a history of anxiety and depression presented to the emergency department unresponsive, next to an empty bottle of diphenhydramine and wine bottles. Vital signs indicated hypotension and hypoxia. The patient was intubated and administered vasopressors. Initial electrocardiogram (ECG) showed a widened QRS complex and terminal R wave in lead aVR, suggesting sodium channel blockade. Treatment with multiple boluses of sodium bicarbonate was ineffective. Lidocaine (95 milligrams intravenously) was administered, resulting in improved ECG findings and patient stabilization. Subsequent care focused on supportive measures and treatment for aspiration pneumonia. The patient was extubated on day two and discharged on day seven to a behavioral health facility.

Conclusion: This case underscores the effectiveness of lidocaine as a secondary treatment for diphenhydramine-induced sodium channel toxicity when standard sodium bicarbonate therapy fails. Lidocaine's ability to restore myocardial conduction illustrates its potential as a critical intervention in toxicological emergencies. [Clin Pract Cases Emerg Med. 2025;19(2):223-227.]

Keywords: *diphenhydramine overdose; sodium channel toxicity; lidocaine; case report; sodium bicarbonate resistance.*

INTRODUCTION

Diphenhydramine is a commonly misused over-the-counter drug intended for allergies or as a sleep aid, with overdose rates particularly high among children and adolescents due to rising social media challenges like the TikTok "Benadryl Challenge" became popular.[1] Among overdose deaths, diphenhydramine is the most common antihistamine found.[2] Although perceived as an H1 receptor antagonist, it functions as an inverse agonist, affecting histamine and acetylcholine receptor activity.[3] In overdose, effects can range from mild sedation and tachycardia to coma, seizures, and cardiac dysrhythmias.[4] First-generation antihistamines like diphenhydramine cross the blood-brain barrier, leading to central nervous system effects, unlike second-generation antihistamines, which are more selective for

peripheral receptors. Most drugs from both generations are metabolized hepatically via the cytochrome P450 system, but their duration of action differs significantly.[5]

After oral intake at therapeutic doses, diphenhydramine concentration peaks in the bloodstream within two to three hours. After first-pass metabolism, 40-60% of an oral dose enters systemic circulation, with excretion predominantly via urine. The drug's half-life ranges from 4-17 hours, increasing in duration with patient age.[4,6] Toxicity may manifest as antimuscarinic effects such as tachycardia, blurred vision, dry mucous membranes, and urinary retention. Sodium channel blockade from tricyclic antidepressant (TCA) effects can lead to QRS widening and subsequent cardiovascular collapse from dysrhythmias. Pediatric fatalities have been reported at doses below 500 milligrams (mg), with seizures noted at 150 mg. For adults, the fatal dose is estimated at 20-40 mg per kilogram (kg). Overdose treatment is largely supportive. However, antidotal therapy with physostigmine to treat the antimuscarinic effects and treatment of sodium channel blockade with sodium bicarbonate to treat dysrhythmia may be necessary.[7]

CPC-EM Capsule

What do we already know about this clinical entity?
Diphenhydramine overdose can cause severe sodium channel blockade, leading to widened QRS, ventricular arrhythmias, and hemodynamic instability.

What makes this presentation of disease reportable?
Despite multiple sodium bicarbonate boluses, the patient had persistent QRS widening and ventricular tachycardia, which was successfully reversed with lidocaine.

What is the major learning point?
Lidocaine may serve as an effective alternative treatment for refractory sodium channel toxicity when sodium bicarbonate fails in diphenhydramine overdose.

How might this improve emergency medicine practice?
This case highlights the importance of recognizing lidocaine as a secondary therapy in diphenhydramine toxicity and optimizing treatment protocols for sodium channel blockade.

CASE REPORT

A 28-year-old man with a past medical history of anxiety, depression, stiff person syndrome, and Hashimoto thyroiditis was brought to the emergency department (ED) unresponsive after being found next to an empty bottle of diphenhydramine and multiple wine bottles. Upon presentation to the ED, vital signs were noted as follows: blood pressure, 104/27 millimeters mercury (mm Hg); heart rate, 90 beats per minute; respiratory rate, 18 breaths per minute; and oxygen saturation was low at 87% on room air. Due to the patient's altered mental status, critical illness, and concern for airway protection, he was orotracheally intubated with etomidate and rocuronium and placed on mechanical ventilation. Post rapid sequence intubation sedation was done with propofol and dexmedetomidine. Due to the patient's hypotension, norepinephrine was initiated. Initial electrocardiogram (ECG) demonstrated sinus rhythm at a rate of 54 beats per minute, a terminal R wave in lead aVR of 9 mm, and a QRS interval of 162 milliseconds (msec) (reference range: 80-100 msec), findings suggestive of sodium channel blockade like those seen in TCA overdoses[8,9] (Image 1).

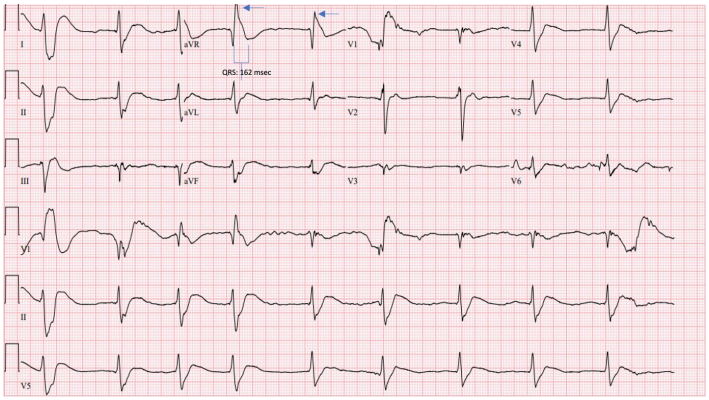


Image 1. Initial electrocardiogram (8:52 AM) notable for widened QRS to 162 milliseconds (blue bracket) with terminal R wave noted in AVR (blue arrow).

Poison control was called at this time; their recommendations were to give sodium bicarbonate and repeat vitals and ECGs on the patient. They did not specify dosing. They suggested that we look for co-ingestions as well, and if there was no improvement to possibly try extracorporeal membrane oxygenation. Venous blood gas (VBG) revealed severe metabolic acidosis with pH less than 7.00 (7.32-7.43), a markedly elevated partial pressure of carbon dioxide over 115 mmHg (40-60 mm Hg), and a lactic acid level exceeding 20 millimoles per liter (mmol/L) (0.5-2.0 mmol/L). To treat sodium channel blockade, the patient received 11 boluses of 8.4% sodium bicarbonate totaling 550 milliequivalents. A repeat ECG showed no improvement, with sinus tachycardia

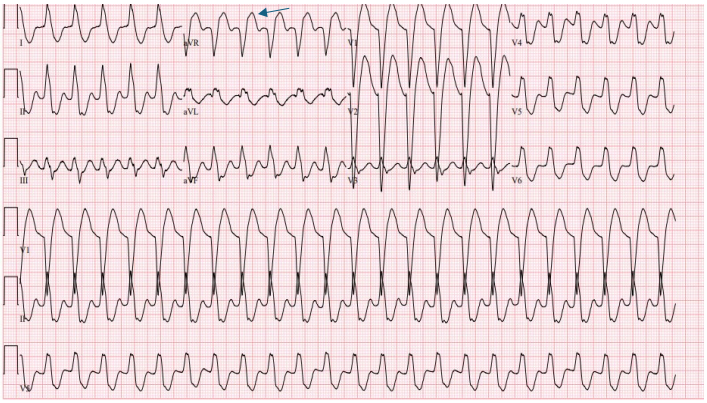


Image 2. Electrocardiogram after sodium bicarbonate and amiodarone administration 10:12 AM (notable for persistently widened QRS to 144 milliseconds with terminal R wave in AVR still present (blue arrow).

at a rate of 156, widened QRS complex of 174 msec, and a terminal R wave in lead aVR of 7 mm (Image 2). The patient's blood pressure did improve to 122/24 after administration of norepinephrine post-intubation.

Initially, this rhythm was interpreted as ventricular tachycardia ventricular tachycardia by the bedside team, who administered a bolus of 150 mg amiodarone and attempted synchronized cardioversion twice, with no appreciable response. Repeat VBG at this time demonstrated a pH of 7.31. Given the lack of appreciable ECG changes despite alkalinization, further sodium bicarbonate was not administered. The attending physician made the decision to administer 95 mg intravenous lidocaine based on the patient's weight (1-1.5 mg/kg) at 10:16 AM due to minimal improvement of V-tach and refractory V-tach. At 10:38 AM the patient's ECG improved, demonstrating sinus tachycardia with a rate of 140 beats per minute, QRS 104 msec, and a decreased terminal R wave amplitude in aVR to 2.5 mm (Image 3).

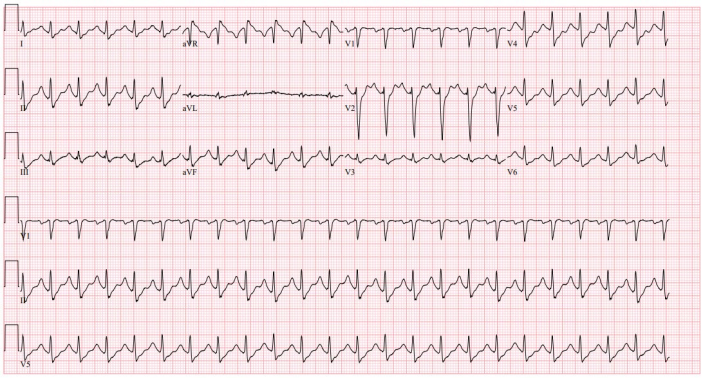


Image 3. Electrocardiogram after lidocaine administration QRS duration decreased to 102 milliseconds 10:38 AM – lidocaine administered at 10:30 AM (per electronic health record).

Activated charcoal, 100 grams, was then administered via nasogastric tube. The urine toxicology screen was negative for any commonly detected drugs of abuse. Serum acetaminophen and salicylate concentrations were undetectable. The patient was admitted to the intensive care unit (ICU), where he continued to receive supportive care and treatment for aspiration pneumonia. Given that sodium bicarbonate has considerable effects on their serum potassium concentration, the initial potassium once the patient was stabilized and then admitted to the ICU was 2.7 mmol/L. An initial potassium was not determined before the boluses of sodium bicarbonate were given. A serum diphenhydramine concentration obtained approximately eight hours after arrival was found to be elevated at 2,500 nanograms/mL. To note, the recommendations for diphenhydaramine dosing are 25–50 mg orally every 4–6 hours, with a maximum dose of 300 mg/day.

The patient was extubated on hospital day two and transferred to the general medical floor on hospital day three, during which time he admitted to psychiatry and hospital staff that the overdose was intentional. He was discharged to a behavioral health facility on hospital day seven where an additional ECG showed normal sinus rhythm (Image 4).

DISCUSSION

This patient exhibited severe cardiovascular toxicity from diphenhydramine overdose as evidenced by his ECG changes. He presented with severe sodium channel toxicity secondary to diphenhydramine overdose, exhibiting hypotension, widened QRS, and acidosis. Despite initial treatment with sodium bicarbonate and amiodarone, the QRS complex remained persistently wide with a terminal R wave in aVR. After a bolus of lidocaine, the QRS narrowed significantly, and the patient stabilized. He was admitted to the ICU on a bicarbonate drip and did not require additional doses of lidocaine. A detailed timeline of this case can be seen in Figure below.

Diphenhydramine, an H1 histamine receptor inverse agonist commonly used for its antiallergic and sedative

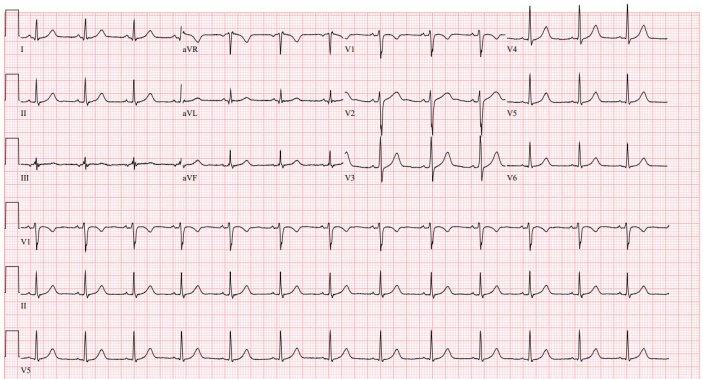


Image 4. Electrocardiogram with normal sinus rhythm on day seven.

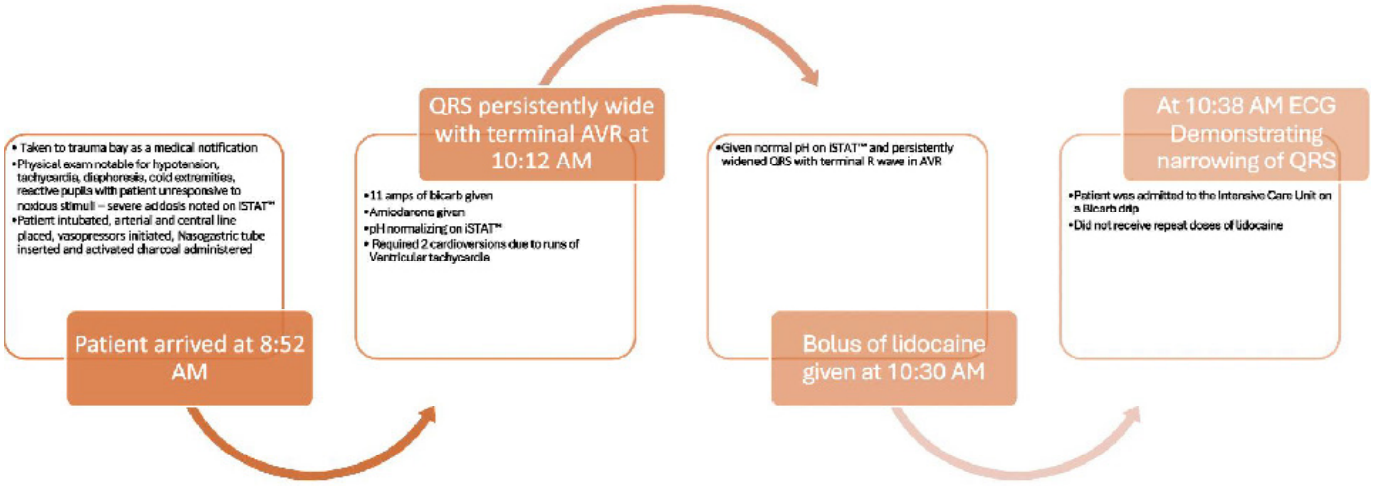


Figure. Case timeline.
bicarb, bicarbonate; *i-STAT*, portable blood analysis system (Abbott Laboratories, Chicago, IL).

properties, works on many different pathways including the histamine and muscarinic receptors as well as sodium channels in the neurologic and cardiovascular systems. The ECG findings in this case demonstrated sodium channel blockade with a prolonged QRS interval (>100 msec) and terminal R wave in lead aVR, similar to ECG findings seen in TCA toxicity. The presence of a terminal R wave in lead aVR (terminal rightward axis deviation) and a widened QRS interval suggested severe toxicity, consistent with the patient's clinical condition.[10] These ECG changes were effectively managed with hypertonic sodium bicarbonate therapy in this scenario.

The literature provides multiple instances where these specific ECG alterations have been observed, such as the case of a 13-year-old female who overdosed on diphenhydramine. Treatment with sodium bicarbonate usually reverses these changes and improves symptoms.[11] Unfortunately, although sodium bicarbonate improved our patient's acidemia, it did not improve the cardiovascular toxicity as reflected by ECG.

The cardiotoxic effects attributed to diphenhydramine, including wide-complex tachycardia, arise from its blockade of fast sodium channels on cardiac membranes. This prolongs phase 0 of the cardiac action potential and delays electrical conduction through the heart, akin to the action of class Ia antiarrhythmics.[12] Sodium bicarbonate counteracts the cardiotoxic effects by increasing the transcellular sodium concentration gradient, decreasing xenobiotic affinity for the sodium channel by increasing pH, and restoring normal myocardial conduction.[12] In this case, a large amount of sodium bicarbonate was given with little, if any, improvement in the cardiac conduction abnormalities, despite effective alkalinization as demonstrated by VBG.

Lidocaine is a class Ib anti-dysrhythmic with affinity for inactivated sodium channels, resulting in decreased action potential duration and decreased effective refractory period.[13] Lidocaine exhibits "fast on, fast off" sodium channel binding kinetics, meaning it rapidly binds to and inhibits the channel before quickly dissociating, restoring normal function. This property allows lidocaine to effectively compete with diphenhydramine, displacing it from the sodium channel. Despite its rapid dissociation, a single dose of lidocaine may lead to sustained improvement by transiently restoring normal sodium channel function, allowing endogenous recovery mechanisms—such as drug redistribution or metabolism—to reduce diphenhydramine's inhibitory effects over time.[13] This property allows lidocaine to "compete" with diphenhydramine and effectively displace it from the sodium channel. This case demonstrates the effective use of lidocaine in refractory sodium channel blockade unresponsive to sodium bicarbonate therapy and its utility for toxicologic Advanced Cardiovascular Life Support (ACLS). Lidocaine has become a second- or third-line antidysrhythmic in ACLS algorithms due to the evolution of the medical literature over the years.[13]

CONCLUSION

This report highlights the successful use of lidocaine for refractory sodium channel toxicity following a diphenhydramine overdose in a 28-year-old male. Despite initial management with sodium bicarbonate, only lidocaine effectively reversed the severe cardiotoxic effects. This case emphasizes lidocaine's potential utility in ACLS for toxicological emergencies. It also underscores the variability in treatment responses, urging further investigation into alternative therapeutic strategies for managing drug-induced cardiac disturbances.

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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Anterior Mitral Leaflet Flutter on M-mode Echocardiography as an Indicator of Atrial Fibrillation: Case Report

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Introduction: M-mode in bedside point-of-care ultrasound (POCUS) transthoracic echocardiography (TTE) remains an important tool for emergency physicians. M-mode of the mitral valve is used to assess ejection fraction (EF) during assessment of E-point septal separation (EPSS). Anterior mitral leaflet fluttering visualized on M-mode echocardiography is a known sequela of aortic regurgitation. Although not previously reported in the emergency medicine (EM) literature, anterior mitral leaflet fluttering also occurs with atrial fibrillation.

Case Report: We present the first case in peer-reviewed EM literature of anterior mitral leaflet fluttering observed on M-mode echocardiography caused by atrial fibrillation. Our patient was a 54-year-old male who had a POCUS TTE that showed anterior mitral leaflet fluttering on EPSS. Subsequent inpatient workup confirmed the diagnosis of symptomatic atrial fibrillation without ischemia or clinically significant aortic regurgitation.

Conclusion: Emergency physicians must rapidly assess and risk-stratify undifferentiated patients presenting with chest pain. Understanding that anterior mitral leaflet fluttering on M-mode during E-point septal separation may signal atrial fibrillation augments efficient and appropriate disposition of these patients. [Clin Pract Cases Emerg Med. 2025;19(2):228-231.]

Keywords: *POCUS, point-of-care ultrasound, transthoracic echocardiogram, TTE, M-mode, aortic regurgitation, atrial fibrillation, anterior mitral leaflet flutter, case report.*

INTRODUCTION

Point-of-care ultrasound (POCUS) transthoracic echocardiography (TTE) is an important and frequently used tool for evaluation of the undifferentiated emergency department (ED) patient with chest pain, dyspnea, palpitations, or a dysrhythmia observed on electrocardiogram (ECG). In assessing ejection fraction (EF), emergency physicians may estimate EF visually. E-point septal separation (EPSS) ascertained using M-mode echocardiography is among the most accessible options for quantifying the EF and supporting the visual estimate.

M-mode echocardiography depicts motion by isolating a single raster line of two-dimensional (2D) echocardiography.[1] While 2D and Doppler ultrasound have overshadowed M-mode in past decades, M-mode remains valuable in a range of contexts, especially where temporal resolution and a high frame rate are necessary for detecting pathology.[2] These qualities carry special weight in assessment of the mitral valve, which is likely "the fastest moving structure within the heart."[2] M-mode facilitates assessment of certain aberrant mitral valve motion, including "the fine fluttering associated with aortic regurgitation."[1, 2]

"[F]ine fluttering" of the mitral valve also appears to be a manifestation of atrial fibrillation.[3, 4] Based on our literature search, this association with atrial fibrillation is not widely known or discussed in the emergency medicine (EM) literature.

We present the first case in peer-reviewed EM literature of anterior mitral leaflet fluttering observed on M-mode echocardiography during EPSS assessment, favored to represent a sequela of the patient's underlying atrial fibrillation.

CPC-EM Capsule

What do we already know about this clinical entity?
Anterior mitral leaflet flutter on M-mode is known to occur with aortic regurgitation but is underrecognized as a sign of atrial fibrillation.

What makes this presentation of disease reportable?
It's the first peer-reviewed emergency medicine case linking anterior mitral leaflet flutter on M-mode to atrial fibrillation.

What is the major learning point?
Mitral leaflet flutter on M-mode during E-point septal separation may indicate atrial fibrillation, even without significant aortic regurgitation.

How might this improve emergency medicine practice?
It enhances rapid bedside identification of atrial fibrillation using point-of-care ultrasound, aiding diagnosis and patient disposition.

CASE REPORT

Our patient was a 54-year-old male, active-duty soldier with a past medical history of paroxysmal atrial fibrillation (onset in the prior year, anti-coagulated on rivaroxaban, rate-controlled on diltiazem, and adherent to his medications), hypertension, and obstructive sleep apnea, presenting with chest pain, shortness of breath, and palpitations that woke him from sleep. He described the chest pain as left-sided, radiating to his left shoulder, 8/10 in intensity, and associated with diaphoresis. He presented to sick call, where he was given aspirin 324 milligrams (mg) and transferred to the ED. On evaluation in the ED, his pain had resolved.

The patient was stable, afebrile, with a heart rate of 88 beats per minute, respiratory rate of 16 breaths per minute, blood pressure 105/70 millimeters of mercury, and pulse oximeter oxygen saturation of 97% on room air. His physical exam was significant for an irregularly irregular cardiac rhythm with no murmurs, rubs, or gallops appreciated. Labs were significant for a negative troponin performed at sick call, a repeat negative troponin five hours later in the ED, negative D-dimer, unremarkable complete blood count, and unremarkable electrolytes. The patient's ECG was significant for atrial fibrillation with a ventricular rate of 72, normal axis, unremarkable intervals, and no ischemic changes, all consistent with his prior ECGs. Chest radiograph was without acute cardiopulmonary process.

Bedside POCUS TTE was performed with a PX ultrasound system (Fujifilm Sonosite, Inc, Bothell, WA) using the cardiac phased array 5-1 MHz transducer and cardiac settings. The patient was in atrial fibrillation at the time of the TTE. The scan was significant for normal EF, no wall motion abnormality, no pericardial effusion or tamponade, and fluttering motion of the mitral valve thought to represent the effect of regurgitant flow from an incompetent aortic valve (Videos 1, 2).

M-mode in the parasternal long-axis view to assess EPSS demonstrated a sawtooth pattern of anterior mitral valve fluttering (Image). The apical four-chamber view was significant for aortic regurgitation.

Given the concern that the sawtooth pattern of anterior mitral valve pattern represented a new-onset valvular pathology, including severe aortic regurgitation, the patient was admitted to the hospital for further workup. The next day, he underwent TTE performed by a radiology technician. He was again in atrial fibrillation. The cardiologist's report largely affirmed the findings of the POCUS TTE. That said, although M-mode EPSS was performed, it did not detect anterior mitral valve leaflet fluttering, and the patient's aortic regurgitation was quantified as "trivial." The patient also underwent a myocardial perfusion study that was negative for ischemia or infarction. He was discharged with a cardiology referral and primary care follow-up.

At his follow-up appointment with cardiology three weeks later, the patient reported that he did not feel that he had converted to sinus rhythm since his admission. He continued to remain symptomatic, with chest pain and dyspnea (for which he had been evaluated again in the ED the day after his discharge from the hospital) and was referred to electrophysiology for ablation.

DISCUSSION

With the advent of 2D imaging and Doppler techniques, present-day echocardiography relies less on M-mode.[1, 2] The peer-reviewed literature on the M-mode appearance of the mitral valve dates largely from the 1980s and early 1990s.[5, 6, 7, 8, 9] This literature established that aortic regurgitation can cause anterior mitral valve leaflet fluttering detectable on M-mode.[1, 2, 7, 9] This anterior mitral valve fluttering has been a proposed etiology of the Austin Flint murmur,[10, 11] but at least one study reported no difference in the frequency of anterior mitral valve fluttering in patients with and without the Austin Flint murmur.[12] These decades-old studies do not discuss POCUS. In the ED, POCUS TTE is commonly performed for

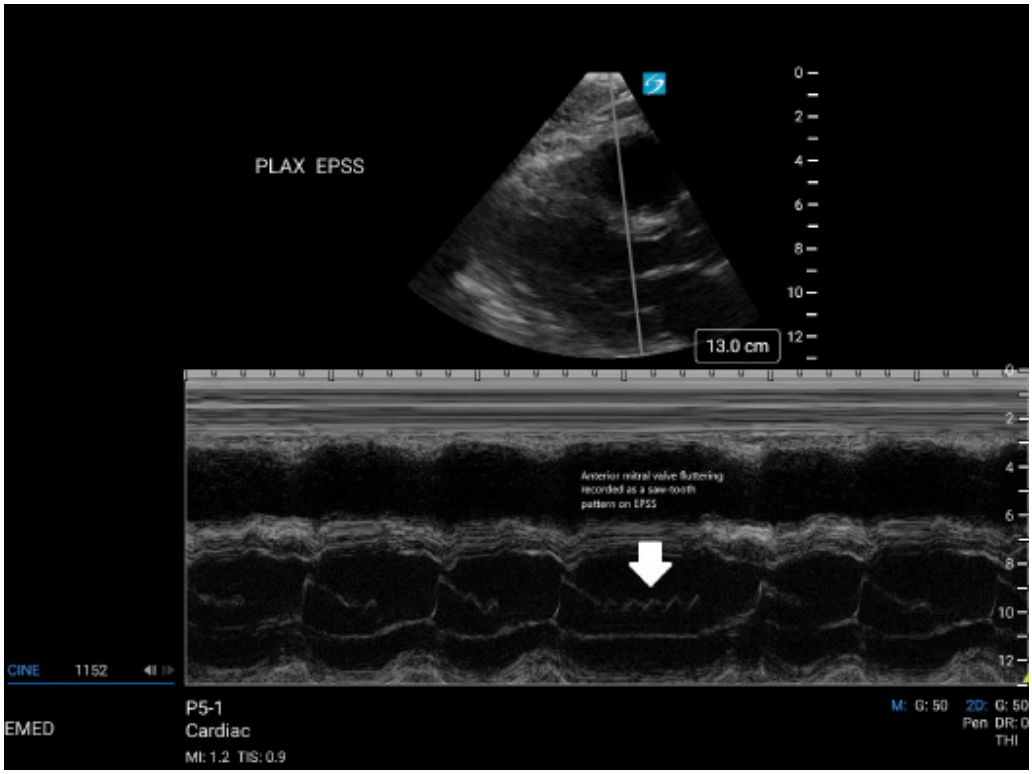


Image. M-mode visualization of anterior mitral valve fluttering in parasternal long axis shows a sawtooth pattern (arrow). *PLAX EPSS*, E-point septal separation measured in the parasternal long-axis view.

patients presenting with chest pain, dyspnea, dysrhythmia, and palpitations. With the less frequent use of Doppler on TTE in the ED, M-mode EPSS may be used to quantify EF and support EF estimation based on visual assessment.[12, 13]

In the present case, the anterior mitral leaflet flutter observed on M-mode echocardiography was interpreted as a sequela of the patient's incompetent aortic valve, consistent with the echocardiography literature. That said, subsequent TTE performed by the radiology technician and read by the cardiologist found only "trivial" aortic regurgitation. "Severe" aortic regurgitation is typically required to produce the mitral valve flutter.[1, 7, 9]

The cardiologist's report did not provide an obvious alternative explanation for the M-mode anterior mitral valve flutter. Our examination of the peer-reviewed literature was likewise unrevealing. But similar M-mode tracings of anterior mitral valve flutter were located on non-peer-reviewed online sources, and these identified the finding as also indicative of atrial fibrillation.[3, 4] As our patient was in atrial fibrillation at the time of the POCUS TTE, it seemed reasonable to attribute the observed anterior mitral leaflet flutter to a function of his atrial fibrillation.

Recognition of the anterior mitral valve flutter on M-mode echocardiography as a manifestation of atrial fibrillation is valuable for emergency physicians performing POCUS TTE on an undifferentiated patient with chest pain, dyspnea, palpitations, or dysrhythmia. In the case at hand, the patient was admitted with concerns for new-onset mitral valve pathology or aortic regurgitation. Had the consistency of the M-mode finding with atrial fibrillation been known to the emergency physicians, the interpretation of the POCUS TTE would have supported a diagnosis of symptomatic atrial fibrillation, which was the patient's ultimate diagnosis on discharge from the hospital.

CONCLUSION

Anterior mitral leaflet fluttering on M-mode echocardiography is consistent with atrial fibrillation in the appropriate clinical context. This association does not appear in the EM literature, and the frequency and quality of atrial fibrillation associated with this aberrant mitral leaflet flutter is poorly described in the available literature. This information is of particular relevance to emergency physicians assessing E-point septal separation as part of point-of-care ultrasound transthoracic echocardiography on the undifferentiated ED patient with chest pain, dyspnea, palpitations, or dysrhythmia. Where a patient is in atrial fibrillation and lacks severe aortic regurgitation, anterior mitral leaflet fluttering on M-mode may confirm symptomatic atrial fibrillation in a patient presenting with chest pain.

Video 1. The anterior mitral valve is fluttering in the parasternal long axis (see arrow and label).

Video 2. The anterior mitral valve is fluttering in the parasternal short axis (see arrow and label).

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

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Case Report: ST-Elevation Myocardial Infarction in Third Trimester Pregnancy

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Introduction: While rare in pregnancy, acute coronary syndrome (ACS) does happen. It has been found to be more common in individuals with risk factors. A case of chest pain in a previously healthy female in her third trimester demonstrates the importance of keeping ACS high on the differential list.

Case Report: A 26-year-old pregnant female gravida five, para three at 37 weeks gestation with a past medical history of diet-controlled gestational diabetes, obesity, and family history of myocardial infarction (MI) presented to an outside hospital for chest pain and was transferred to the closest ST-elevation myocardial infarction (STEMI) receiving emergency department (ED) after she was found to have an electrocardiogram (ECG) concerning for acute STEMI. On arrival to the ED, STEMI protocol was activated based on ST-segment elevations on inferior and antero-lateral leads on the ECG. Bedside assessment of the fetus by obstetrics showed a viable intrauterine pregnancy, and the patient was taken to the cardiac catheterization lab. She was found to have a 100% thrombotic occlusion in the ostium of the right posterolateral artery, and percutaneous coronary intervention was performed. The patient was discharged with plans for cesarean section at 39 weeks.

Conclusion: This case highlights the need for early STEMI activation and consultation with obstetrics when a pregnant patient presents with an ECG suggestive of STEMI. It also emphasizes the importance of maintaining a high level of suspicion for STEMI in pregnant patients presenting with chest pain. Although rare—0.6 in 10,000 pregnancies—mortality rates range from 5.1-37% throughout pregnancy and postpartum. It is important to remember that pregnancy does not preclude a patient from undergoing standard treatment of acute MI. [Clin Pract Cases Emerg Med. 2025;19(2):232-235.]

Keywords: *acute myocardial infarction; spontaneous coronary artery dissection; ST-elevation myocardial infarction; pregnancy; case report.*

INTRODUCTION

Chest pain is a common chief complaint that emergency physicians should be comfortable addressing and risk-stratifying. While the overall risk of myocardial infarction (MI) in the general childbearing-age population is low, a complaint of "chest pain" should heighten the senses of emergency physicians.[2] On the other hand, acute MI during pregnancy is more common when compared to a similar non-pregnant population.[3] Additionally, one should keep in mind that spontaneous coronary artery dissection (SCAD) has been reported as one of the most common causes of acute coronary syndrome (ACS) in pregnant patients.[3,4] However, the risk factors that are commonly associated with acute ST-elevation myocardial infarction (STEMI) during pregnancy differ from those associated with SCAD.[5,6]

From an epidemiological standpoint it is important to

maintain a frame of reference but always keep in mind atypical presentations. There are, however, common risk factors that predispose women to acute MI during different stages of pregnancy. Atherosclerosis plays a bigger role in first trimester MI, atherosclerotic and thrombosis for second trimester, and SCAD during third trimester with SCAD being the most common cause at any point during pregnancy.[5,7,8] As is evidenced by our case report, the patient described falls outside the previously reported pathophysiological causes of MI in the third trimester.

CASE REPORT

A 26-year-old female gravida five, para three at 37 weeks gestation with a past medical history of diet-controlled gestational diabetes, obesity, and a sister who had died from an MI at age 30 presented to an outside hospital labor and delivery, where she reported acute onset of mid-chest pain while grocery shopping. The patient was transferred to a STEMI-receiving ED due to concerns for STEMI on the initial electrocardiogram (ECG) (Image 1). Prior to arrival, the patient received aspirin and morphine at the outside hospital.

Vitals on arrival were blood pressure 131/83 millimeters of mercury, heart rate 99 beats per minute, respiratory rate 25 breaths per minute, and oxygen saturation 99% on room air. The ECG in the ED showed ST elevation in leads II, III, V3, V6, and aVF with reciprocal depressions in V1, V2, I and aVL (Image 2). Laboratory results revealed elevated troponin at 0.07 nanograms per milliliter (ng/mL) and 6.04 (ng/mL) (reference range: ≤0.03 ng/mL) and normal glucose of 114 milligrams per deciliter (mg/dL) (70-140 mg/dL). Point-of-care ultrasound performed by the cardiology team demonstrated a hypokinetic inferior wall. Fetal assessment

CPC-EM Capsule

What do we already know about this clinical entity?
Acute myocardial infarction (MI) is a rare condition in pregnancy, typically caused by spontaneous coronary artery dissection (SCAD) in the third trimester.

What makes this presentation of disease reportable?
This case report highlights a pregnant patient in her third trimester who presents with acute MI secondary to thrombosis and atherosclerosis, conditions typically seen in the first and second trimester.

What is the major learning point?
Acute MI can occur during the third trimester of pregnancy and is not always attributed to SCAD.

How might this improve emergency medicine practice?
It is important to maintain a high level of suspicion for MI in pregnant patients presenting with chest pain.

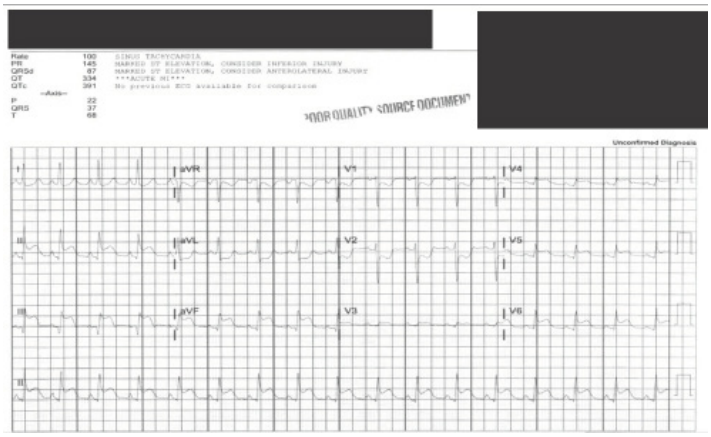


Image 1. Electrocardiogram demonstrating ST-elevation myocardial infarction (STEMI). Leads II, III, aVF, V3-V6 demonstrate marked ST-segment elevation consistent with STEMI, and leads aVL, V1-V2 show marked ST-segment depression consistent with reciprocal ECG changes.

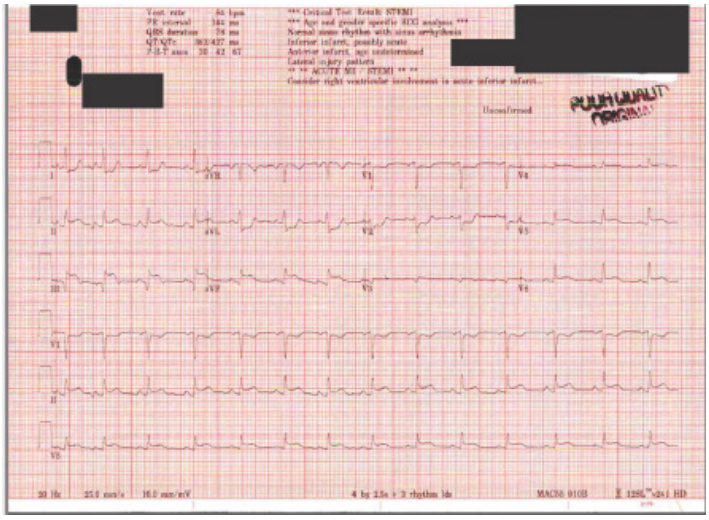


Image 2. Electrocardiogram obtained at time of patient's arrival to cardiac receiving center. Leads II, III, aVF show similar ST-segment elevation compared to initial ECG. Leads I and aVF show progression in ST-segment depression. V1 shows improved ST-segment depression but similar V2 depression. There is improved ST-segment elevation in Leads V3-V6.

showed fetal heart rate of 130 beats per minute (110-160 beats per minute). Ultimately, the patient was emergently taken to the cardiac catheterization lab. She was given clopidogrel 600 milligrams (mg) and 10,000 units of heparin total (3,000 intra-arterial followed by 7,000 units intravenous).

The cardiac catheterization lab report showed 100% thrombotic occlusion in the ostium of the right posterolateral artery (rPL) without other evidence of coronary artery disease. Percutaneous coronary intervention (PCI) was performed to the rPL vessel with a 3.0 x 18 millimeter drug-eluting stent (DES). No coronary artery dissection was evident. During hospitialization, the patient was followed by obstetrics (OB) who initially recommended labor induction at 39 weeks. Obstetrics later recommended elective cesarean section due to suspected fetal macrosomia, gestational diabetes mellitus, and obesity with high risk for shoulder dystocia. The patient did not experience complications and was discharged home from the cardiology service with instructions to continue clopidogrel 75 mg daily, aspirin 81 mg daily, and metoprolol 12.5 mg daily.

Prior to the scheduled C-section, a transthoracic echocardiogram was obtained, which found "mild hypokinesis and preserved thickness of the basal inferior (posterior) wall and mild hypokinesis and preserved thickness of the mid inferior wall, with an ejection fraction of 65%." The patient was later admitted to the OB service; she underwent a C-section under general anesthesia, and a healthy nine-pound, 7.3-ounce male was delivered. There were 700 mL estimated blood loss reported without major complications. On day seven, after an uneventful postoperative course, the patient was discharged home.

DISCUSSION

Acute MI is a rare condition in pregnancy. However, reports indicate that MIs during the third trimester are often associated with SCAD. Acute MI in a pregnant patient is statistically more common in multigravidas, with a prevalence of 66%, but it is particularly higher in patients who are >30 years of age, with incidence of 72%. Additionally, these cases often involve the anterior wall, accounting for 78% of occurrences.[7] The history of gestational diabetes, obesity, and family history of MI put this patient at a higher risk of ACS.

Given that SCAD is most common in the third trimester of pregnancy, it is important to risk-stratify patients whose presentations are concerning for acute MI in the peripartum or postpartum period. Also, without an angiogram, SCAD can only be suspected based on previously described risk factors; for that reason, closed loop communication with cardiology is necessary to guide acute phase treatment. Regarding medication treatment options, there are several working theories. One theory postulates that bleeding from the vasa vasorum creates an intramural hematoma in the coronary arteries leading to myocardial ischemia.[9] For that reason, if SCAD is suspected or confirmed, the continued use of anticoagulation and antiplatelet therapy should be avoided unless there is confirmation of thrombus or there are other systemic indications.[10] If anticoagulation is continued, there is a theoretical risk of worsening intramural hematoma and extension of the dissection.[10] Another theory argues that there is likelihood from an anatomical standpoint, as evidenced by the lack of inflammatory cells in tissue studies, that SCAD might be precipitated by impairment of endothelial repair, which is exacerbated during pregnancy due to low levels of estrogen.[9] It fully advocates for anticoagulation and antiplatelet treatment if no other contraindications exist.[9] This stands in contrast to the mechanical forces theory underlying the intramural hematoma theory.

Ultimately, if diagnostic studies such as coronary angiogram reveal thrombotic occlusion causing MI, then PCI and aggressive medical management are indicated as described in this case report. Glycoprotein blockers have higher binding efficacy to abciximab over eptifibatide and tirofiban, particularly during lactation, and most thrombolytics seem to be compatible with pregnancy; however, human data is still limited.[8]

CONCLUSION

The leading cause of STEMI and non-STEMI during the peripartum and postpartum period in the third trimester is spontaneous coronary artery dissection. As in the general population other causes such as advancing maternal age and history of atherosclerosis and thrombosis are bigger risk factors during the first and second trimesters. While the pathophysiology of SCAD has not been fully elucidated, a low estrogen state seems to play a major role in the impairment of endothelial repair leading to plaque formation and thrombotic events. Interventions such as computed tomography of the coronaries, coronary angiography, and percutaneous coronary intervention should not be withheld if indicated.

Medical management including aspirin, beta blockers, anticoagulation, selective antiplatelets, and thrombolytics if indicated should not be withheld to treat an acute MI in the peripartum or partum period. Finally, the early consultation of OB and cardiology is important if acute coronary syndrome is suspected. If the patient is pregnant and in her third trimester, emergency physicians should discuss the increased likelihood of SCAD. This does not change the initial management in the acute phase of treatment. If SCAD is noted on coronary angiography, cardiology will then decide on optimal medical treatment vs PCI. If interventional cardiology is not available at the time of STEMI diagnosis, the patient should be promptly transferred to a STEMI center preferably with in-person or telephone OB consult services available.

The authors have received Institutional Review Board approval. Patient consent was obtained for publication of this case report. Documentation on file.

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A Case Report of Rattlesnake Musk Exposure Causing Chemical Conjunctivitis

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Introduction: Rattlesnakes are pit vipers belonging to the *Viperidae* family and *Crotalinae* subfamily. They inject venom into their victims via bites from two long, hollow fangs. When disturbed, they can release a liquid musk from cloacal scent glands into the air. This report describes a rare case of non-penetrating rattlesnake ocular exposure with symptoms. We also discuss pathophysiology, evaluation, and treatment recommendations.

Case Report: A 56-year-old male picked up a rattlesnake and was sprayed in both eyes with liquid emanating from the snake. He was not bitten by the snake. He had immediate pain and blurred vision. Despite copious initial irrigation, he continued to have worsening symptoms with subconjunctival hemorrhage and scleral injection. After discussion with poison control, he was given six vials of intravenous (IV) antivenom. After additional irrigation and evaluation by ophthalmology, the patient's symptoms stabilized, but his exam still included blepharitis, subconjunctival hemorrhages, and bilateral, small corneal epithelial defects. He was discharged home with corneal antibiotics and artificial tears. One week later his symptoms were resolved, and his exam was normal.

Conclusion: Non-penetrating musk ocular rattlesnake exposure is rare. In certain conditions, it may be from exposure to snake musk from scent glands and not venom. It should be treated as any other ocular exposure beginning with copious irrigation and then a detailed examination. Current recommendations argue against IV antivenom administration. [Clin Pract Cases Emerg Med. 2025;19(2):236-238.]

Keywords*: rattlesnake; snake musk; ocular exposure; chemical conjunctivitis; case report.*

INTRODUCTION

Venomous snakes use their venom for a variety of purposes including hunting, digestion, and defense.[1] The venom can be released either by biting or spitting. Rattlesnakes are pit vipers belonging to the *Viperidae* family and *Crotalinae* subfamily. This subfamily also includes cottonmouths and copperheads.[2] They evolved to inject venom into their victims via bites from two long, hollow fangs. According to a recent study by Maciulewicz et al, rattlesnake envenomation requires a specific angle and compression of fangs, leading to their conclusion that the likelihood of a rattlesnake spitting venom is close to zero.[3] In contrast, there are numerous reports of a separate family of snakes, the *Elapidae* family, causing non-penetrating ocular envenomation.[4] These snakes are mostly found outside the United States and include mambas, cobras, taipan, kraits, and coral snakes. The *Elapidae* use specialized glands that do not require compression to spit venom onto their prey.[5]

Snakes that are disturbed can forcefully expel an airborne "musk" from their cloacal area via scent glands.[6] This has been observed by herpetologists in many types of snakes including rattlesnakes.[7] Snake musk includes multiple volatile and nonvolatile substances that are not well described.[8] Maciulewicz et al proposes this as the likely mechanism for

ocular irritation when a rattlesnake has been handled and releases liquid but no biting-down force of the mouth or fangs has occurred.[3] Snake musk exposure is an extremely rare topic in the medical literature. This topic is primarily described in animal and zoological texts.

In this report we describe a rare, rattlesnake ocular exposure, likely from snake musk, resulting in a chemical conjunctivitis. We highlight pathophysiology, evaluation, and treatment recommendations.

CASE REPORT

A 56-year-old male with a history of diabetes mellitus and hypertension picked up a rattlesnake with the intent to decapitate it. Instead, he was sprayed in both eyes with liquid from the snake, resulting in immediate pain and blurry vision. The patient was able to confirm that it was a rattlesnake, but no further identifying characteristics were available. At the initial hospital, poison control recommended irrigation, analgesia, ophthalmology evaluation, and topical antibiotics. Labs included complete blood count, complete metabolic panel, and coagulation studies, which were normal. Despite two liters of normal saline irrigation to each eye, his clinical examination worsened with subconjunctival hemorrhages and scleral sloughing. After a secondary discussion with poison control, he was given six vials of the intravenous (IV) antivenom CroFab (BTG International Inc, West Conshohocken, PA). As the initial hospital did not have emergency ophthalmology consultation, he was transferred to our facility.

At our emergency department he reported bilateral ophthalmalgia, subconjunctival injection, watery discharge, and blurry vision. Initial examination was significant for a visual acuity of 20/50 in the left eye, 20/70 in the right eye, mild scleral inflammation, bilateral subconjunctival hemorrhages, and bilateral watery/tan discharge (Image).

Each eye was irrigated with an additional two liters of normal saline prior to formal ophthalmology evaluation. Examination by the ophthalmologist was significant for visual acuity of 20/25 in both eyes, bilateral ocular pH of seven (normal range 7.0-7.4), blepharitis, subconjunctival hemorrhages, and bilateral small corneal epithelial defects. The anterior and posterior segments were normal except for the conjunctival hemorrhages and scleral injection.

He was discharged with seven days of the following medications: ciprofloxacin eye drops four times per day, erythromycin ointment nightly, and preservative-free artificial tears every hour. The medications were chosen by the ophthalmology consultant. One week later, the follow-up examination in the ophthalmology clinic showed complete resolution of his corneal abnormalities.

CPC-EM Capsule

What do we already know about this clinical entity?
Recent literature shows that rattlesnakes are unable to spray venom unless under very particular circumstances. Rattlesnakes can, however, release musk from cloacal glands into the air.

What makes this presentation of disease reportable?
Snake musk exposure is extremely rare in medical literature. We present this case of non-penetrating rattlesnake exposure causing chemical conjunctivitis.

What is the major learning point?
Non-penetrating rattlesnake exposure with conjunctival symptoms may be from cloacal snake musk. It should be treated with copious irrigation and examination.

How might this improve emergency medicine practice?
If any providers have similar patients in the future, they will be able to confidently treat as a chemical exposure and not as a rattlesnake envenomation.

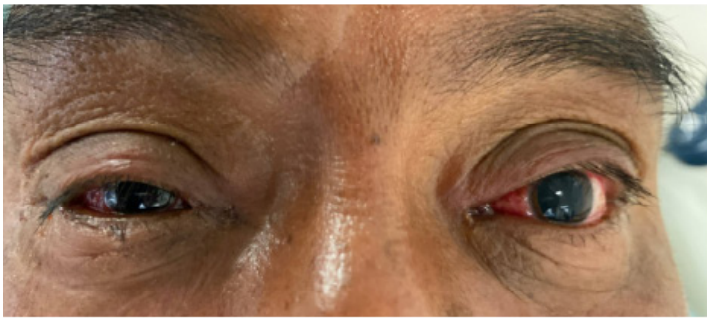


Image. Patient with mild scleral injection and bilateral subconjunctival hemorrhages after rattlesnake musk exposure.

DISCUSSION

This is a rare case of non-penetrating, ocular, rattlesnake exposure, presenting with corneal defects and subconjunctival hemorrhage. Previously, Troutman et al and Cantrell et al both described cases with mild symptoms treated to resolution with irrigation only.[9,10] Johnson described a case treated with irrigation, IV antivenom, and topical antibiotics.[11] In these case reports it was assumed the symptoms were from

rattlesnake envenomation. In our case, the treating clinicians initially considered this to represent another rare case of rattlesnake non-penetrating envenomation. However, given the recent study by Maciulewicz et al demonstrating rattlesnake spitting venom as extremely unlikely in these conditions, we feel this more likely represented a chemical conjunctivitis from exposure to snake musk

Most documented snake ocular exposures are from African and Asian *Elapidae* spitting snakes. All published data demonstrates anterior ocular segment damage including subconjunctival injection, blepharitis, corneal erosions, and uveitis.[4,5] Delayed time to treatment has been shown to result in complications such as corneal opacities and blindness. *Elapidae* venom has effects similar to chemical burns in the eye. Inflammatory cytotoxic compounds such as metalloproteinase and phospholipase are the culprit venom components. There are no reports of systemic toxicity from ocular snake-venom exposure.[4,5]

The treatment of any ocular substance exposure is immediate and prolonged irrigation. Additional options include topical analgesics, topical antibiotics, cycloplegics, and antihistamines.[4,5] Evaluation should include pH testing, visual acuity evaluation, slit lamp examination, and fluorescein staining. Emergency ophthalmology evaluation may be necessary if symptoms worsen or do not resolve. Topical corticosteroids, topical antivenom, and IV antivenom are contraindicated due to potential adverse effects and lack of evidence showing systemic toxicity.[4,5,12]

In our case, IV antivenom was empirically recommended by our local poison center when the patient's symptoms did not initially improve. After reviewing the most recent literature, it is likely that continued supportive care of the patient's chemical conjunctivitis, rather than antivenom treatment, resulted in symptom resolution.

CONCLUSION

Non-penetrating, ocular rattlesnake envenomation is a rare occurrence. Under the right conditions, exposure may be due to musk released from snake scent glands, causing a chemical conjunctivitis. It should be evaluated and treated as any other ocular exposure beginning with copious irrigation and then a detailed examination. Current recommendations advise against intravenous antivenom administration.

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.

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Use of Point-of-care Ultrasound for Placement of a Gastric Tamponade Balloon

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Case Presentation: A 30-year-old female with a history of alcoholic cirrhosis and esophageal varices presented with massive hematemesis. A gastric balloon tamponade device was subsequently placed to temporize variceal hemorrhage, and point-of-care ultrasound (POCUS) was used to confirm the appropriate placement of the gastric balloon before complete inflation. We describe a novel use of ultrasound for use in severely ill patients with gastrointestinal (GI) bleeding.

Discussion: A fluid-filled and distended stomach has long been recognized as a cause of a false-positive focused assessment with sonography in trauma exam but may also be a vital piece of information in the scenario of a patient with suspected upper GI hemorrhage. There is very little description in the literature of using POCUS to confirm the appropriate placement of a gastric tamponade balloon with none by emergency physicians.. Ultrasound may offer advantages over plain radiography in this application given its speed and safety; thus, its utility for this task is worth further investigation. [Clin Pract Cases Emerg Med. 2025;19(2):239-241.]

Keywords: *POCUS; gastric; varices; balloon tamponade device; hematemesis.*

CASE PRESENTATION

A 30-year-old female presented to the emergency department (ED) with a chief complaint of hematemesis. She had a medical history of alcoholic cirrhosis complicated by ascites and esophageal varices. She had recently been admitted to another hospital and had banding of esophageal varices due to bleeding, requiring multiple blood transfusions. On arrival to the ED the patient was noted to be jaundiced and unwell appearing. Her initial vitals were blood pressure of 73/31 millimeters of mercury, heart rate 142 beats per minute, respiratory rate 32 breaths per minute, saturation 97% on room air, and she was afebrile. Her physical exam was notable for a distended, tender abdomen, and jaundice. While being examined, the patient was noted to have a large episode of coffee-ground emesis, approximately 800 milliliters (mL). Intravenous (IV) access was obtained, and one unit of packed red blood cells was infused. She was given octreotide, pantoprazole, and ceftriaxone.

The patient had transient improvement in blood pressure, which dropped again as the initial unit of blood was completed. She had several further episodes of hematemesis. At this point, a focused assessment with sonography in trauma exam was performed due to patient instability and shock, which demonstrated anechoic free-fluid consistent with the patient's known history of cirrhosis and varices. Also noted in the subxiphoid and left upper quadrant windows was a fluid-distended stomach with heterogeneous material concerning for blood products in the clinical scenario (Image 1).

At this time, large-bore central IV access was established, and massive transfusion protocol was started. The patient was intubated uneventfully for airway protection. The gastroenterology team was consulted, and a decision was made to place a Minnesota tube to temporize bleeding prior to transjugular intrahepatic portosystemic shunt (TIPS) procedure.

After confirming the adequate function of Minnesota tube balloons and soaking it in an ice bath, a laryngoscope



Image 1. Subxiphoid/left upper quadrant image obtained with curvilinear 5-1 megahertz probe demonstrating the spleen (hexagon), the gastrosplenic ligament (arrow), and stomach with mixed echogenic contents (star) with anechoic free-fluid between the structures.

CPC-EM Capsule

What do we already know about this clinical entity?
The standard protocol for confirming correct placement of a gastric tamponade balloon is radiographic images.

What is the major impact of the image(s)?
A gastric tamponade balloon can be visualized in the stomach with ultrasound.

How might this improve emergency medicine practice?
Point-of-care ultrasound can be used to rapidly confirm safe placement of a gastric tamponade device in a clinically unstable patient.

blade was placed in the oropharynx to visualize the endotracheal tube through the cords and obtain a view of the esophagus. The Minnesota tube was passed into the esophagus to a depth of 50 centimeters. At this point, the gastric balloon was inflated with 50 mL of air, and an ultrasound machine was used at the bedside to observe the stomach for the appearance of gaseous distension of the balloon (Image 2).

Once confirmed to be in the stomach, the gastric balloon was fully inflated and retracted until resistance was met, and gentle traction was applied by securing the tube with an endotracheal tube holder. After resuscitation, the patient was taken for emergent TIPS; unfortunately, she expired due to hemorrhagic shock.

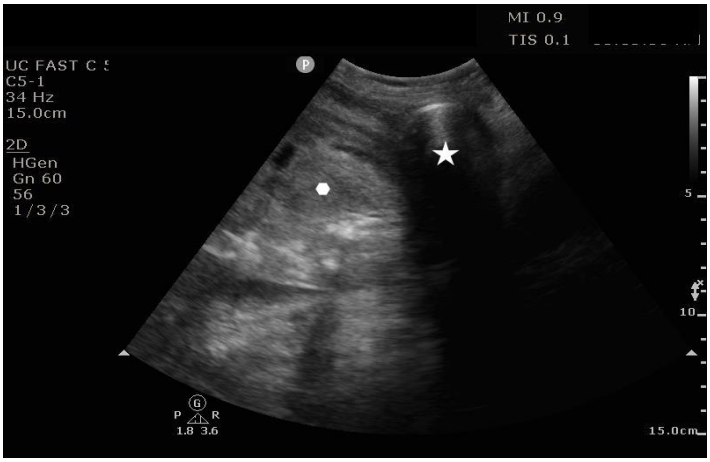


Image 2. Subxiphoid view obtained with curvilinear 5-1 megahertz probe demonstrates upper portion of the stomach with mixed echogenic contents (hexagon) and rounded echogenic structure with posterior shadowing representing inflated gastric balloon of Minnesota tube (star).

DISCUSSION

Literature is sparse on the use of point-of-care ultrasound (POCUS) for guidance or confirmation of gastric tamponade device placement, with no reports of this application by emergency physicians.[1,2] We describe a novel use of POCUS for this purpose. Although plain radiography is the preferred modality for confirmation of correct placement of a gastric balloon tamponade, POCUS offers significant potential advantages to plain radiography. It is more readily available, faster to interpret, and can be performed concurrently with other resuscitative procedures. Using POCUS for placement of gastric tubes is possible when other resources are unavailable, and research demonstrates reasonable accuracy for this purpose.[3,4] Although use of POCUS for evaluation of balloon tamponade devices is not routine, it may be an alternative to plain radiography. More research is warranted.

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

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Pyoderma Grangrenosum

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Case Presentation: We describe a middle-aged female with past medical history of ulcerative colitis presenting to the emergency department with bilateral painful ulcers rapidly growing on her lower legs in the prior four weeks. She was consulted by a dermatologist and after a thorough clinical and pathology assessment (as a diagnosis of exclusion), treatment for pyoderma gangrenosum was started.

Discussion: Pyoderma gangrenosum is a painful, chronic, ulcerative disorder often occurring in association with systemic disease. We review the clinical presentation of pyoderma gangrenosum and its complications. We describe the characteristics of ulcers with pictures from the patient. Our case illustrates the findings of pyoderma gangrenosum both clinically and pathologically. [Clin Pract Cases Emerg Med. 2025;19(2):242-244.]

Keywords: *Pyoderma gangrenosum; ulcerative colitis.*

CASE PRESENTATION

A 51-year-old female presented with bilateral painful shin ulcers. She has a medical history of ulcerative colitis and multiple sclerosis, for which she was being treated with glatiramer acetate and mesalamine, respectively. The patient denied experiencing fever, chills, or night sweats. She also had a brown nodular painful ulcer on her right forearm, which presented four weeks prior. Her exam was significant for bilateral lower leg palm-sized cribriform ulcer, large violaceous bullous plaque on her right lower leg, and red-brown small nodular plaque on the right forearm (Images 1-3). The patient's clinical condition was reviewed by a dermatologist, and after a thorough clinical and pathological assessment, treatment for pyoderma gangrenosum was initiated.

DISCUSSION

Pyoderma gangrenosum (PG) is a reactive, non-infectious, inflammatory dermatosis and is classified as one of the neutrophilic dermatoses, along with Sweets syndrome and Behcets disease. The incidence of PG is approximately 0.63 per 100,000 people, with the median age of onset being 59 years.[1]

Although the lower legs are the most frequently affected, PG can manifest on any part of the body. The condition is often precipitated by minor trauma, a phenomenon known as "pathergy."[2]

A thorough history is key with specific inquiry regarding possible pathergic response to minor or major trauma, as well as a history of pain, rapid progression, symptoms suggestive of infection or systemic disease and a detailed drug history.[3] Indeed, PG lesions are all too often misdiagnosed as simple non-healing ulcers and patients undergo debridement, which can result in catastrophic deterioration of the condition through this pathergic response. The condition predominantly affects adults, but childhood cases are rarely reported.[4] More recently, Mavrakis et al have proposed new criteria based on a consensus of international experts, requiring one major and four minor criteria.[5]



Image 1. The patients left lower leg ulcer characteristic of pyoderma gangrenosum**.**

CPC-EM Capsule

What do we already know about this clinical entity?
Pyoderma gangrenosum is a neutrophilic dermatosis, which presents most commonly in patients with inflammatory bowel disease.

What is the major impact of the image(s)?
We present the classic presentation of pyoderma gangrenosum regarding images, also in a clinical course, which can assist physicians in making a diagnosis.

How might this improve emergency medicine practice?
Recognizing pyoderma gangrenosum classic features and presentation can help physicians increase their index of suspicion and diagnostic accuracy leading to improved outcomes.



Image 2. This image shows both of the patients lower leg ulcers demonstrating different stages of pyoderma gangrenosum.



Image 3. The patients' right forearm pustular lesion, demonstrating another stage of pyoderma gangrenosum**.**

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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A Case of Prehospital Magnesium Sulfate Extravasation

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Case Presentation: A 73-year-old female with chronic obstructive pulmonary disease presented via emergency medical services (EMS) for shortness of breath. She was found to be hypoxic, tachypneic, and in notable distress by EMS. She was treated with inhaled albuterol, oral dexamethasone, and intravenous (IV) magnesium sulfate. Upon arrival to the emergency department her left hand was noted to have significant bleeding, and on further investigation it was determined that the IV catheter had inadvertently become dislodged causing medication extravasation, causing the magnesium to enter the subcutaneous space. The bleeding was significant and noted to be pulsatile; a tourniquet was applied; vascular damage was noted and was ultimately ligated by the trauma surgery service.

Discussion: Intravenous medication administration is ubiquitous with emergency care in both the hospital and prehospital environments. Medication use is paramount to treatment of a vast majority of emergent clinical conditions; furthermore, the route of administration is often via IV in the patient with emergent illness. The placement of IV catheters is a skill that nurses, paramedics, and advanced emergency medical technicians learn early in their training. The care team is tasked with starting IV lines but also in monitoring them and ensuring medication is delivered into the systemic circulation and not elsewhere. Certain medications, notably potassium preparations and vasoactive medications, are known vesicants. We present a case of vascular extravasation of magnesium sulfate, not known for causing tissue damage, which led to significant vascular injury. This case highlights the need for prehospital professionals as well as members of the emergency department care team to be ever vigilant for medication extravasation. [Clin Pract Cases Emerg Med. 2025;19(2):245-247.]

Keywords: *IV infiltration; extravasation; magnesium sulfate; prehospital IV access.*

CASE PRESENTATION

A 73-year-old female with a history of chronic obstructive pulmonary disease (COPD) called emergency medical services (EMS) for worsening shortness of breath. The patient was found to be hypoxic despite home oxygen. Concern for COPD exacerbation prompted bronchodilators via nebulizer as well as oral dexamethasone. A 22-gauge intravenous (IV) catheter was placed with standard tactile technique in the dorsum of the left hand for administration of magnesium sulfate. There were two attempts at IV cannulation, and it was unclear why the dorsal hand was chosen as a site. Of note, no other medications or fluids had been administered through the IV catheter. In the emergency department the patient's hand was edematous with concern for IV infiltration, and the IV was subsequently removed. Significant hemorrhage was noted with pulsatile bright red blood, a tourniquet was applied, and surgery was able to ligate the culprit vessel, which was found to be arterial (Images 1 and 2). During her hospital stay the wound improved, and she was discharged after resolution of a COPD exacerbation with outpatient, wound care follow-up.



Image 1. Dorsal surface of left hand with skin tear and ecchymosis status post extravasation.

CPC-EM Capsule

What do we already know about this clinical entity?
Magnesium sulfate is not known to cause tissue injury when subject to extravasation.

What is the major impact of the image(s)?
Magnesium sulfate was the culprit for acute vascular injury when extravasated in this case; clinicians should be aware to mix and administer this medication appropriately.

How might this improve emergency medicine practice?
Monitoring for intravenous extravasation, even medications previously considered to be benign, is important for clinicians who administer medications via this route.

DISCUSSION

Intravenous catheter infiltration and medication extravasation are well-known complications of IV medication administration. However, extravasation causing vascular injury to point of significant hemorrhage has not been well described in the literature. The extravasation of magnesium sulfate has not been known to cause significant tissue injury; however, in this patient the magnesium sulfate was presumed to be the culprit, as there were no other medications administered through the IV, and the total infusion volume could not have been more than 104 milliliters (mL). Per local EMS protocols, magnesium is stocked in 1 gram (g)/2 mL vials and diluted in 100 mL of normal saline for a total dose of 2 g diluted in 104 mL of normal saline. Infusions are administered by infusion pump over 15 minutes.

The IV was placed in the ambulance while driving to the hospital. There are multiple potential issues with this case that could have led to the vascular and subcutaneous injury. The EMS report indicates that two IV attempts were made and that IV placement was confirmed with blood return; however, this could have certainly been in error. It is



Image 2. Dorsal surface of left hand status post vascular ligation.

possible that the IV was inadvertently placed into an artery and then became dislodged leading to bleeding. The movement of the ambulance as well as the possibility of the successful IV being placed more distal in the same vein as the prior attempt could have led to extravasation as well. While magnesium is directed to be diluted and infused via infusion pump, it is possible that the medication was given undiluted via IV push. This concentrated form has the potential to be much more cytotoxic. Magnesium sulfate has not been known to be an active vesicant and has been considered relatively safe in the setting of inadvertent, soft-tissue exposure. However, if given in a concentrated form it would be more caustic. Taogoshi et al showed that higher concentrations of magnesium sulfate, greater than 8%, were cytotoxic in a rat model.[1]

Treatment for medication extravasation is dependent on the medication being used. In the case of magnesium sulfate, which does not have an antidote, the accepted treatment is to stop the infusion, withdraw as much medication out of the catheter with a syringe as possible, and then remove the catheter.[2] Saline or other irrigation should not be infused into the catheter prior to removal. The affected extremity should then be elevated, bandaged, and a warm pack applied. The affected area should be checked at regular intervals for tension, bleeding, and vascular and neurologic changes.

This case demonstrates the need for heightened awareness when administering all medications, not just vesicants. This is of particular concern for EMS agencies placing IVs and monitoring them enroute. The anatomical location (dorsum of the hand) and non-ideal conditions (patient's home and in a moving ambulance), as well as medication characteristics, all lead to significant injury.

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

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