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Clinical Practice and Cases in Emergency Medicine (CPC-EM) is a MEDLINE-indexed internationally recognized journal affiliated with the *Western Journal of Emergency Medicine* (*West*JEM). It offers the latest in patient care case reports, images in the field of emergency medicine and state of the art clinicopathological and medicolegal cases. CPC-EM is fully open-access, peer reviewed, well indexed and available anywhere with an internet connection. CPC-EM encourages submissions from junior authors, established faculty, and residents of established and developing emergency medicine programs throughout the world.

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50-year-old Male With Chest Pain

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A 50-year-old male presented to the emergency department with four days of intermittent chest pain and shortness of breath, which progressively worsened in severity. Testing revealed a troponin I greater than 100 times the upper limit of normal and an electrocardiogram with non-specific findings. This case takes the reader through the differential diagnosis and systematic work-up of the deadly causes of chest pain, ultimately leading to this patient's diagnosis. [Clin Pract Cases Emerg Med. 2019;3(4):321–326.]

CASE PRESENTATION (William Fernandez, MD)

A 50-year-old male presented to the emergency department (ED) clutching his chest. He complained of severe generalized chest pain, shortness of breath, left leg pain, and feeling as if he were going to pass out. Over the preceding four days, he had felt a generalized, non-radiating, non-specific chest pain with mild shortness of breath both at rest and with minimal exertion. The discomfort resolved at times spontaneously and at other times with marijuana use. Over these four days the discomfort intermittently recurred, worsening with each occurrence. The morning of his presentation to the ED he woke at 3 AM with sudden worsening of the generalized chest discomfort, which at that time had become pressure-like and was associated with an acute worsening of his shortness of breath. Later that morning, he developed severe nausea/vomiting and had a sudden episode of pre-syncope after which he noted an ache-like pain throughout his left leg. At home he tried smoking marijuana to improve his symptoms, but because his symptoms continued to worsen, he presented to the ED for evaluation.

He had a past medical history significant for non-ischemic cardiomyopathy with a left ventricular ejection fraction (LVEF) of 50-55%, remote cerebral vascular accident with residual mild slurred speech, polycystic kidney disease, chronic obstructive pulmonary disease, hypertension, hyperlipidemia, bipolar disorder, and schizoaffective disorder. He did not take any medications and had no known drug allergies. His family history was notable for a myocardial infarction (MI) in his mother at age 56 years, and a cerebral aneurysm rupture in his mother at age 57 years resulting in her death; there was no known paternal history. He related daily tobacco use with a 12.5 pack-year smoking history and daily marijuana use. He denied alcohol or other substance use. He lived in an abandoned apartment with 4-6 roommates, all of whom were active polysubstance abusers.

On arrival to the ED, he was alert and oriented, ill appearing, and in moderate distress due to chest pain. He was afebrile (36.5 degrees Celsius) with a heart rate of 78 beats per minute, blood pressure of 116/69 millimeters of mercury, oxygen saturation of 100% on room air and a respiratory rate of 26 breaths per minute. He weighed 54.4 kilograms (kg) and was 1.65 meters (m) in height with a body mass index of 20.0 kg/m^2. He was well developed and well nourished. His head was normocephalic and atraumatic with dry mucus membranes. Pupils were equal, round, and reactive to light and accommodation bilaterally with normal extra-ocular movements. Sclera were anicteric, and fundi were without papilledema. The neck was supple and without lymphadenopathy or carotid bruits. His lungs were clear to auscultation bilaterally without wheezes, crackles, or rhonchi. He had no retractions but was tachypneic. His heart was regular rate and rhythm without murmurs, rubs, or gallops. The abdomen was soft with normal bowel sounds and without distension, rebound, guarding, or hernias; however, he had tenderness in the epigastric and periumbilical regions. There was no costovertebral angle tenderness. The extremities had no edema, tenderness or deformity. There were 2+ radial and dorsalis pedis pulses bilaterally.

Neurologic examination showed intact cranial nerves II-XII, 5 out of 5 strength throughout all extremities, normal muscle bulk and tone, and intact sensation. His speech was slightly slurred but unchanged from baseline, according to him. His affect and behavior were normal, and he was answering all questions appropriately. He was fully oriented to self, place, and time.

We obtained an electrocardiogram (ECG) (Image 1). Initial laboratory results are shown in Table. Point-of-care ultrasound showed a trace pericardial effusion and a significantly dilated left ventricle with poor systolic function. Computed tomography angiogram (CTA) of the chest, abdomen, and pelvis showed no evidence for acute aortic dissection or injury (Image 2). Cardiomegaly with left ventricular dilation was shown, as well as evidence of advanced atherosclerosis of the pelvic vasculature and polycystic kidneys (previously known). After the CTA of the chest was performed, the patient was taken to the cardiac catheterization laboratory for angiography, which showed an LVEF of 10-20% with akinesis of the inferior wall and hypokinesis of the anterolateral wall of the heart, 2+ mitral regurgitation, severe pulmonary hypertension, and mild diffuse atherosclerotic disease without occlusion of any major vessels. A diagnostic test was then performed, which confirmed the diagnosis.

CASE DISCUSSION (Zachary Dezman, MD, MS, MS)

This case illustrates a common challenge for emergency physicians: the simultaneous institution of diagnostics and empiric treatment, while stabilizing an acutely ill patient. I found myself agreeing with every action taken by the treating doctors. The initial history is representative of many patients I see: a middle-aged man with an outsized burden of disease, including polysubstance abuse and psychiatric illness. When I see "chest pain" on a patient's triage sheet, I think of six "deadly" causes: acute coronary syndrome (ACS), pulmonary embolism (PE), pneumothorax, Boerhaave's syndrome, aortic dissection, and pericarditis. The patient's presentation and risk factors could support any of these diagnoses. The challenge is to determine which features of the presentation to focus upon.

The *Journal of the American Medical Association* has endeavored to quantitate physician gestalt through the Rational Clinical Exam series.[1] For the patient in this case, we see that his prior history of cerebrovascular accident, male sex, and hypertension each slightly raise his risk of ACS.[2] Tobacco use and a family history of early cardiac disease are classic risk factors for cardiac disease,[3] but these have variable value for diagnosing ACS in the ED setting.[4] The patient's chest pain, including the recent change in the pattern – the exertional component and its association with dyspnea – all increase the probability of ACS. Note that vomiting is associated with MI specifically.[5] The patient's ECG is also suggestive of ischemia, with T-wave inversions, and S-T segment depressions. Given these patient features and a pre-test probability of 13% used by Fanaroff et al.,[2] this patient's post-test probability of ACS is greater than 80%.[6] Surveys of physicians have shown that a miss rate of approximately 1% is considered "acceptable" for ACS,[7] so the physicians in this

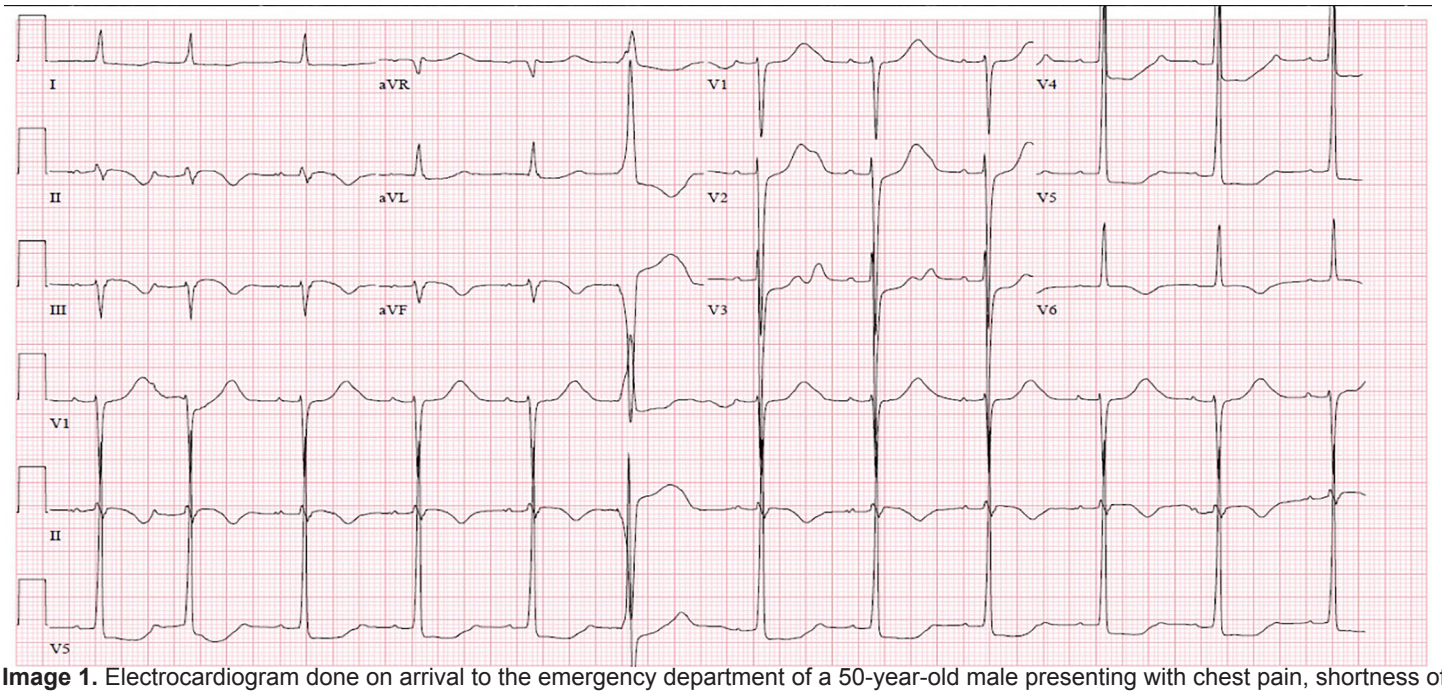

Image 1. Electrocardiogram done on arrival to the emergency department of a 50-year-old male presenting with chest pain, shortness of breath, leg pain, and pre-syncope.

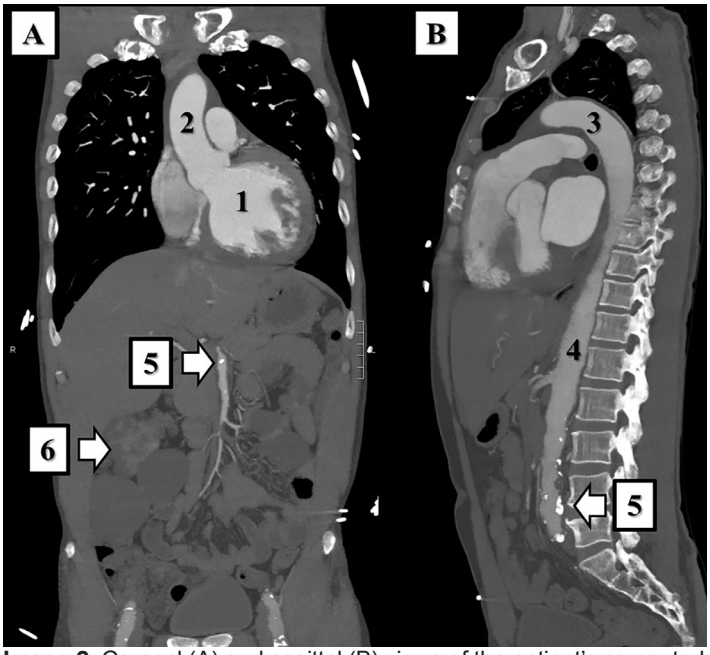

Image 2. Coronal (A) and sagittal (B) views of the patient's computed tomography angiogram of the chest, abdomen, and pelvis. The patient's cardiomegaly can be seen (1), but there is no evidence of dissection at the aortic root (2), arch (3), or descending aorta (4). Calcifications within the abdominal vasculature (5) suggest atherosclerosis. Evidence of the patient's known polycystic kidney disease was seen (6).

case are well over the evidentiary threshold needed to pursue a diagnosis ACS![7] They should immediately activate the cardiac catheterization lab.

Almost; except for the fact that the treating physicians must have also been concerned about aortic dissection and PE. When a patient complains of pain radiating across anatomical borders, in the way this patient's chest pain radiated to his leg, I think of vascular disasters such as aortic dissection. Proximal dissections (i.e., type A) are a rare mimic of ACS, and 4% of all proximal dissections are diagnosed while the patient is undergoing cardiac catheterization for presumed acute MI.[8] Misdiagnosis can be dangerous in these patients as there is a *1-2% cumulative increase in mortality for every hour of delay in treatment.*[8] Similarly, the patient's dyspnea, relative hypotension, insidious presentation, and ECG changes could be caused by a pulmonary embolism (PE). The next reasonable step is to rule out these diagnoses and order a CTA of the chest, abdomen, and pelvis. Thankfully, the patient's imaging did not reveal a dissection or PE.

Once the patient got to the catherization lab, the cardiologist found evidence of heart failure (HF) without an occlusion of the coronaries. The patient's other laboratory tests had returned by this point, showing an elevated erythrocyte sedimentation rate (ESR), a high C-reactive protein, and a really, *really* high troponin.

A number of non-ischemic conditions can can cause an elevation in troponin. Patients with chronic renal or heart failure, or those with an exacerbation of their obstructive lung disease can have an elevated troponin, but these are usually mild. Strenuous exercise, such as running a marathon or a tachydysrhythmia, can elevate one's troponin as well, but these too are usually mild. Takostubo or stress cardiomyopathy can present with very high troponins, but the characteristic "apical ballooning," usually seen on ventriculogram, wasn't mentioned in the presentation. This leaves myocarditis as the most likely cause of the patient's presentation.

Myocarditis can have an insidious onset and then present acutely such as an MI with HF.[9] Troponins in excess of 1,700 nanograms per milliliter (ng/mL) have been recorded in the literature.[10] ESR and C-reactive protein levels have also been seen in patients with myocarditis. About 7.0% of cases recur, and this patient had undergone a prior nuclear medicine scan in 2002, which could have been related to a prior episode.

This patient's history shows he is at risk for myocarditis from several causes. He could have had viral myocarditis due to hepatitis B or C, given his substance use history. There are cases of clozapine-induced myocarditis, which the patient might have been exposed to while being treated for his mental illness. Myocarditis has also been associated with lung cancer, and we discovered a new lung mass on this patient's imaging.

Cardiovascular magnetic resonance (CMR) has become much more common and is now "the primary tool for noninvasive assessment of myocardial inflammation in patients with suspected myocarditis."[11] I believe that a CMR was done, which detected edema at the site of injury, seen as high-intensity enhancement on T2- weighted images. Early and late enhancement with gadolinium, which is indicative of irreversible injury, may also have been seen.[11] These findings would support the final diagnosis of myocarditis.

CASE OUTCOME

The diagnostic study of choice was CMR. The patient was found to have significant edema throughout the myocardium, with a focus over the anterolateral and inferior walls, seen on T2-weighted images.

While in the cardiac catheterization lab, the patient received an intra-aortic balloon pump (IABP) to support his cardiac perfusion. He was then admitted to the cardiac intensive care unit (CICU) and started empirically on broad-spectrum antibiotics. Serial echocardiograms showed an unchanging LVEF of 10-20% during his first week in the CICU. Further testing did not identify a specific infectious etiology. His LVEF improved to 20-25% on hospital day (HD) 8; the IABP was discontinued, and he was extubated successfully. CMR was then performed, demonstrating the edema described above and making the diagnosis. The remainder of his hospital course was complicated by a moderate retroperitoneal hematoma and dysphagia, both of which were managed medically and

Table. Laboratory values of a 50-year-old male presenting with chest pain, shortness of breath, leg pain, and pre-syncope.

		Reference values
Complete blood cell count		
White blood cells	20.2 K/mcL	(3.4-9.6 K/mcL)
Hemoglobin	13.2 g/dL	(13.2-16.6 g/dL)
Hematocrit	38.3%	(38.3-48.6%)
Platelets	239 K/mcL	(135-317 K/mcL)
Serum chemistries		
Sodium	139 mmol/L	(136-145 mmol/L)
Potassium	3.6 mmol/L	(3.5-5.0 mmol/L)
Chloride	97 mmol/L	(95-105 mmol/L)
Bicarbonate	29 mmol/L	(22-28 mmol/L)
Blood urea nitrogen	20 mg/dL	(7-18 mg/dL)
Creatinine	1.59 mg/dL	(0.6-1.2 mg/dL)
Magnesium	1.5 mmol/L	(1.5-2.0 mmol/L)
Total protein	7.1 g/dL	(6.0-7.8 g/dL)
Albumin	3.9 g/dL	(3.5-5.5 g/dL)
Total bilirubin	0.7 mg/dL	(0.1-1.0 mg/dL)
Aspartate aminotransferase	1579 u/L	(8-20 u/L)
Alanine aminotransferase	157 u/L	(8-20 u/L)
Alkaline phosphatase	88 u/L	(20-70 u/L)
Additional Labs		
Troponin I	697.0 ng/mL	(<0.034 ng/mL)
Lactate	3.9 mEq/L	(0.3-2.3 mEq/L)
C-reactive protein	6.6 mg/L	(0.0-3.0 mg/L)
Erythrocyte sedimentation rate	55 mm/hr	(0.0-22 mm/hr)

K/mcL, thousands per microliter; *mg,* milligrams; *dL,* deciliter; *g,* gram; *mmol,* millimoles; *L,* liter; *u,* units; *ng,* nanogram; *mL,* milliliter; *mEq,* milliequivalents; *mm,* millimeter; *hr,* hour.

improved without further complication. His last echocardiogram before discharge on HD 19 showed an LVEF of 30-35%. He was discharged to a subacute rehabilitation center.

RESIDENT DISCUSSION

Myocarditis is an inflammatory disease involving the cardiac muscle. It affects 22 per 100,000 persons or around 1.5 million individuals worldwide, annually.[12] The time course for the disease ranges from acute to chronic, with clinical severity depending on the degree of tissue damage, the underlying etiology, and patient comorbidities.

The most common infectious causes of myocarditis are viral, with over 20 different viruses implicated. Coxsackievirus was the most common up until the 1990s, but more recently parvovirus B-19 and human herpes virus 6 have become more common. Bacteria, fungi, protozoa, and even helminths have been identified as causes of myocarditis. Autoimmune disorders such as systemic lupus erythematosus, giant cell arteritis, and granulomatosis with polyangiitis can cause myocarditis as well. Less common, non-infectious etiologies include various cardiotoxins such as alcohol, cocaine, cyclophosphamide and heavy metals, and hypersensitivity reactions from antibiotics, clozapine, insect bites, and snake bites.[13]

Patients with acute myocarditis will often present with chest pain, dyspnea with or without exertion, unexplained sinus tachycardia, tachypnea, and signs of HF. Physicians evaluating patients with these complaints will often also consider ACS, PE, new-onset HF, or aortic dissection. Items in the patient's history that are more supportive of myocarditis include a history of a recent mild illness, medication change, illicit drug use, or a lack of cardiovascular risk factors.[13]

Initial testing should include an ECG, chest radiograph (CXR), cardiac biomarkers, and point-of-care cardiac ultrasound if available.[9] ECG findings can vary dramatically, from sinus tachycardia to diffuse ST-segment elevation with PR-segment depression suggesting pericarditis. The CXR is

helpful in identifying cardiomegaly, which is concerning if new, and may show evidence of pulmonary edema. Cardiac biomarkers can be markedly elevated, especially if the disease has involved all four chambers of the heart. Cardiac ultrasound can help confirm the presence of cardiomegaly as well as estimate the LVEF, identify wall motion abnormalities, and evaluate for other possible causes of the patient's presentation.

CMR can be used to definitively identify myocardial inflammation and make the diagnosis if the patient is clinically stable and the resources are available.[11] Endomyocardial biopsy is the classic method of diagnosing myocarditis, although it is rarely performed today. The affected tissue may be missed due to sampling error during the biopsy, and myocardial rupture and tamponade are rare but potentially life-threatening complications. Biopsy does have the advantage of being able to provide both a diagnosis and an etiology.

Management of acute myocarditis depends greatly on the patient's presentation. Severe cases can present with cardiogenic shock secondary to acute HF and may require emergent intubation, ventilatory support and stabilization with the early administration of diuretics. Inotropic support with vasopressors, IABP, or extracorporeal circulatory membrane oxygenation (ECMO) can be used if the patient is hemodynamically unstable with evidence of shock.[14] Targeted therapies range from appropriate antibiotics for infectious etiologies to intravenous immunoglobulin for autoimmune etiologies. The time-course of therapy depends on the patient's clinical improvement.[15] Serial echocardiograms are used both on an inpatient and outpatient basis to determine the patient's response to therapies.

Prognosis varies with specific etiology and the severity at presentation. Otherwise healthy patients who develop acute myocarditis may return to baseline function if the initial disease process is identified and treated appropriately. Some patients develop chronic myocarditis or have severe enough disease to require cardiac transplantation.[16]

FINAL DIAGNOSIS

Acute myocarditis due to suspected viral or idiopathic etiology.

KEY TEACHING POINTS

- A careful history and physical examination are crucial to suspecting mycarditis and initiating the appropriate diagnostics.
- Myocarditis can be acute, subacute, or chronic.
- Initial management is focused on controlling symptoms of HF; hypotensive patients may require inotropes and invasive support with IABP and/or ECMO.
- Cardiac ultrasound is an invaluable tool to assess for the presence and severity of HF.
- CMR is the diagnostic imaging study of choice.

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

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Arrow to the Chest

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A 33-year-old male was brought to the emergency department after a penetrating arrow injury to the chest. Initial evaluation revealed the arrow was penetrating the sternum, lung, and aortic arch. Because the patient was in a remote area, timely transfer to a specialized center for definitive operative repair was delayed approximately 24 hours. Treatment was focused on minimizing risk of hemorrhage with tight blood pressure control, while tube thoracostomy was deferred to avoid a change in intrathoracic pressure. The left-sided hemothorax was monitored with serial point-of-care ultrasounds. Ultimately he was successfully transferred and underwent successful surgical intervention. [Clin Pract Cases Emerg Med. 2019;3(4):327–328.]

CASE PRESENTATION

A 33-year-old male was brought to the emergency department following an archery accident. The accident occurred when the patient was competing in an archery tournament and an arrow shot from a compound bow was released from approximately 140 meters away, piercing his chest. Upon initial examination, he was conscious with the arrow piercing his chest at the level of the upper third of the sternum; an estimated 12 centimeters of the arrow was implanted in the chest. He was hemodynamically stable and maintaining his oxygenation. Initial point-of-care ultrasound (POCUS) revealed a left-sided hemothorax without pneumothorax or hemopericardium. An emergent chest computed tomography (CT) revealed the arrow penetrating through the sternum, mediastinum, and passing through the aortic arch (Images 1 and 2) with a moderate left-sided hemothorax. Due to lack of cardiothoracic or trauma surgeons in the country, preparations were made for transfer of the patient for definitive repair.

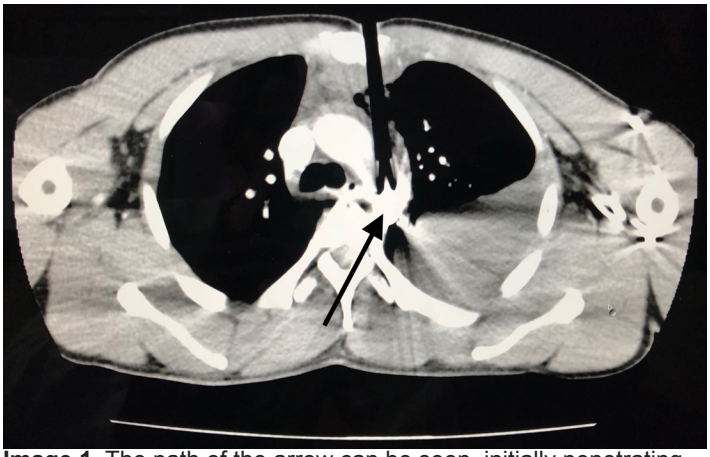

Image 1. The path of the arrow can be seen, initially penetrating the sternum and anterior lung and ultimately lodging in the aortic arch (arrow).

DISCUSSION

Traumatic injuries to the thoracic aorta carry a high mortality rate.[1] Although transesophageal echocardiography was previously considered first line for assessing transthoracic aortic injuries, now its role is limited to unstable patients as CT angiography is the investigational modality of choice.[2] Hemorrhage control and emergency operative therapy are the mainstay of treatment.[3] When emergent surgical intervention is not possible optimizing conditions for hemorrhage control is key.

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

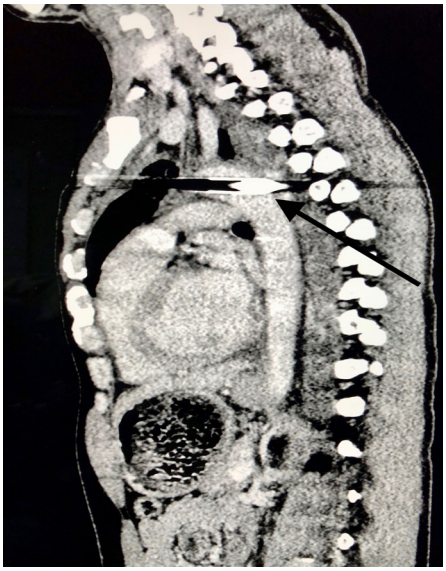

Image 2. The black arrow reveals the depth of the arrow extending through the aortic arch.

CPC-EM Capsule

What do we already know about this clinical entity?
Traumatic injuries of the thoracic aorta carry a high mortality rate. Computed tomography angiography is the imaging modality of choice. Hemorrhage control is key.

What is the major impact of the image(s)?
This unusual image of a penetrating aortic injury with the object in place is illustrative of a situation requiring careful consideration for medical management.

How might this improve emergency medicine practice?
This case demonstrates a pathway to optimize conditions for hemorrhage control in resource- poor areas where a delay to definitive surgical treatment is inevitable.

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Defensive Medicine: A Case and Review of Its Status and Possible Solutions

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Malpractice liability systems exist, in part, to provide compensation for medical malpractice, corrective justice for those injured by it, and to incentivize quality care by punishing substandard care. Defensive medicine is loosely defined as practice based primarily on the fear of litigation rather than on expected patient outcomes. It is largely motivated by a physician's belief that the malpractice system is unfair, slow, and ineffective; these perceptions make malpractice concerns one of the largest physician stressors. A physician's perception of malpractice rarely correlates with the stringency of their state's tort system, overestimates their own risk, and overestimates the cost of defensive practices. While estimates are difficult to make, defensive medicine likely only accounts for 2.8% of total healthcare expenses. The phrase "tort reform" has been frequently used to suggest fixes to the malpractice system and to defensive practices. Safe harbors, clinical practice guidelines, comparative fault reform, reducing plaintiff attorney fees, and apology laws have each been evaluated as potential remedies to defensive practice, although most are unproven and all must be deployed in a state-by-state approach. [Clin Pract Cases Emerg Med. 2019;3(4):329–332.]

INTRODUCTION

In 2004 a physician saw a patient with back pain and a leg abscess. The patient was a likeable guy, morbidly obese but dieting, and stable. He had a normal neurologic exam and a leg wound that appeared to be cellulitis in and around a venous stasis ulcer. The rate of methicillin resistance was still low in 2004 and he was treated and discharged with cephalexin and ibuprofen. When the physician was served a year later, he learned that he had missed an epidural abscess that paralyzed the patient and led to his demise nine months later. Six years later, the same physician saw a patient with atraumatic, nonspecific, thoracic spine pain, a normal neurologic exam, and a cellulitic area on the leg. He had no risk factors for perivertebral infection.

The case reminded the physician of his prior lawsuit, and while he normally would not have ordered magnetic resonance imaging (MRI) of the back, in this case he ordered the test as a result of the previous missed diagnosis. This physician was practicing defensive medicine. The test was being ordered almost entirely out of fear of litigation. There was little concern it would show an epidural. But the MRI turned out to be positive. The patient received antibiotics and surgery and did quite well. Malpractice attorneys like to say they save more lives than physicians. While physicians might strongly disagree with the statement, the lawyer who represented the first patient certainly helped the second.

While defensive medicine can lead to more cautious care, the physician is by definition less motivated by medical outcome than by legal risk. Most physicians have a strong opinion that defensive medicine results in high financial costs and unnecessary testing, and that there is an easy fix through tort reform. The literature paints a more nuanced story, with controversy surrounding the prevalence of defensive practice, the dollars spent on it, and whether or not tort reform could reduce the frequency of defensive practice. In the cases above, the defensive practice led to an unexpected but emergent finding, which can confuse the analysis even further.

Some physicians, depending on their specialty, report that they alter their practice out of fear of a lawsuit.[1] Despite this prevalence, quantifying the cost of defensive medicine is difficult and proposing solutions for it is even harder. This paper explores the definition

of defensive medicine, why it exists, its prevalence, and the costs associated with its practice. The author then reviews potential reforms that might reduce the practice of defensive medicine and discusses the limits of inferences that can be drawn from the limited data available.

DISCUSSION

Definitions

There are multiple definitions of defensive medicine. Kapp et. al. describes it as, "Clinical practice that is driven by the physician's perception of legal self-interest… rather than by concern about expectation of patient benefit."[2] This definition paints the practice as binary and masks the complexity of medical decision- making. In many cases, concern for the patient overlaps with a physician's personal concerns, and this overlap is not reflected in Kapp's definition. The disbanded U.S. Congress Office of Technology Assessment defined defensive medicine this way: "when doctors order tests, procedures, or visits, or avoid certain high-risk patients or procedures, primarily (but not solely) because of concern about malpractice liability."[3] Both of the definitions place blame for the practice on the physician's self-protection outweighing the patient's needs. The latter definition allows for a gray zone where multiple factors can influence a decision simultaneously.

Defensive medicine manifests as two types of risk-avoiding behavior. Assurance behavior involves providing additional testing, hospitalization, or consultation to minimize the perceived risk to the provider.[1] An example would be the patient described in the introduction. Avoidance behavior involves providers declining to offer complicated tests or treat potentially litigious patients in order to reduce the perceived malpractice risk.[1] An example might be an emergency physician not wanting to ask about elder abuse when facing an assertive family member.

Social Benefits and Physician Perception of the Liability System

All medical liability systems exist in part to provide compensation for medical malpractice, "corrective justice" for those injured (such as psychological closure), and the incentive to provide safe, quality patient care.[1,3] However, as a deterrent to the unsafe practice of medicine, the tort system has been shown to be ineffective.[4] There are those who believe defensive medicine helps encourage physicians to be more diligent,[2] which was the case with the epidural abscess case; but this effect remains anecdotal and unquantified.

While malpractice liability has social benefit, physicians see another side of it. Physicians see the malpractice system as slow, ineffective, and biased against them.[1,2,5,6] Malpractice suits are considered one of the largest physician stressors.[7] In general, physicians' fears of the malpractice system are only loosely correlated with the actual stringency of their state's malpractice tort system,[6,8] and often are in excess of risks.[9] For example, in the five states with the highest malpractice risk, 68% of physicians reported engaging in defensive medicine. Yet in the five states with the lowest malpractice risk, the number only decreases to 64%.[8] In addition, while there is a belief that a physician's own malpractice experience shapes his or her degree of malpractice avoidance, studies do not confirm this tendency.[1,5] In the epidural abscess case, previous malpractice experience altered future care.

Quality of Evidence and Confounding Variables

While opinions on the presence and magnitude of defensive medicine are profound, there is little evidence to support those opinions. The majority of studies of defensive medicine in Medline and Westlaw (57%) were based on physician surveys, with only 9% based on primary statistical analysis and 7% on literature reviews (mostly of survey studies).[2] Many studies are based on a single specialty or specific disease (such as heart attacks, spinal disc disease, etc.). The presence of author bias is palpable on all sides of the issue.

Medical decision-making is a complex process that incorporates defensive medicine with other influencers. Those influencers include quality care, financial incentive, patient satisfaction, self-image, professional reputation, and the desire to avoid conflict. Isolating any one variable is exceptionally difficult, and most surveys cannot single out malpractice concerns except through hypothetical simulation. Many use graded scales of perceived malpractice risk to try to simulate situations in which defensive medicine can be identified. Others will attempt to quantify the respondents' malpractice-avoidance and correlate that with costs.[6,10]

Quantifying the Cost of Defensive Medicine

While almost every physician survey shows defensive medicine to be ubiquitous,[1-3,5,6,8,10,11] the total cost and percentage of orders affected is unclear. On in-patient medical services, it is estimated that 2.9% of costs are purely defensive, and another 10.1% are somewhat defensive.[10] One inpatient-based study showed that spending more reduced malpractice risk. Providers with higher hospitalization cost (mean $39,379) had a 0.3% risk of claims per year, while those with lower costs (mean $19,725) had a 1.5% risk of claims per year.[1] In this case, assurance behavior was effective at reducing malpractice risk.

The total cost of defensive practice ranges from $46 billion to $300 billion, although most estimates are between $50-65 billion.[5] This is less than 3% of total healthcare costs. Those in the extreme will claim up to 25% of healthcare costs are generated by defensive medicine,[12] although that number is a high-end estimate of total healthcare waste, of which defensive practice is only one

element. Mello et al. provided one of the most detailed assessments and fully recognized the multiple assumptions made. They concluded that 2.8% of healthcare costs were defensive in nature (in 2008), which equated to $55 billion.[3]

Effect of Tort Reform

It has been proposed (usually by physicians) that significant tort reform could decrease defensive medical practice and thereby decrease medical costs. If true, this would allow physicians to be more comfortable making decisions without undue psychological pressure to mitigate malpractice risks. Unfortunately, in addition to overestimating the actual potential savings, physicians likely underestimate the obstacles to this approach.

Caps on non-economic damages

There is conflicting evidence as to whether tort reform changes practices. For example, an oft-quoted study from 1996 showed that caps on non-economic damages ("pain and suffering") for Medicare patients reduced hospital costs in patients with myocardial infarcts and ischemic heart disease.[13] Later studies using the same methodology and same patient type did not confirm this finding.[14] Mello and Kachalia reviewed several caps on non-economic damages with a conclusion that reducing non-economic damage ceilings had an indeterminate effect on healthcare spending, although there were reductions in spending in some subgroups of spending (e.g., a slower rate in the growth of malpractice premiums, possible reduction in defensive practice, and compensation awards).[15] Bioethicist Ezekiel Emanuel criticizes malpractice caps as not reducing healthcare spending and increasing the risk that patients injured by negligence might not be fully compensated,[8] thereby undermining some of the beneficial social effect of malpractice.

Capping attorney fees

In 2017, an analysis of state malpractice reforms and their effects on malpractice showed the only reform that decreased physician spending on insurance was capping attorney contingency fees.[14] As attorney fees are generally 35-40% of awards (after costs),[3] and the attorney often pays the costs out of pocket, decreasing the contingency fee reduces attorney profits, while keeping their costs the same. This de-incentivizes attorneys and ameliorates physician anxiety by reducing the total number of cases, particularly those with lower potential judgments. In theory, plaintiffs would retain the ability to achieve "corrective justice," although it would be harder to find representation, and the financial structure might change. Should the plaintiff win, he or she would keep more of the judgment than under the current fee system. If one assumes that the cause of defensive medicine is an overabundance of frivolous lawsuits, it is a reasonable approach.

Comparative fault versus contributory negligence

Comparative fault reforms, when enacted, significantly increased malpractice cost.[14] Comparative fault is a tort law concept in which providers are held liable for the proportional percentage of damages based on their contribution to the outcome. [14] The provider may be held liable, even if the patient's actions contributed to an untoward outcome. Comparative fault reforms usually replaced contributory negligence rules, in which if a patient was at 1% (or greater) fault for an injury, the patient could not obtain and damages.[14] It is not surprising that the effect was increasing cost of coverage.

The changes seen in Yu's study were in cost of insurance.[14] It is important to note that the cost of insurance is based on factors other than the amount of defensive medical spending. The rates are in large part set based on risk assessment by malpractice carriers. In this sense, they are a proxy for measuring the degree of physician risk of a malpractice case.

Safe harbors for evidence-based guidelines

One proposal that may have merit is the creation of "safe harbors" for evidence-based guidelines.[5,8] In these harbors, using clinical practice guidelines (CPG) developed under rigorous quality standards would protect the physician from legal judgment. Some national organizations, such as the American Board of Internal Medicine with its *Choosing Wisely* campaign, have led the push for this approach. They seek to improve quality while reducing defensive practice. There is survey evidence that doctors do not trust CPGs to legally protect them,[2] but there currently are no safe harbor laws in place to test this theory. The guidelines can be used by defendants and expert witnesses to show a standard of care, although the protective effect of CPGs has not been measured.

Apology laws

Apology laws have been enacted in several states to allow physicians to apologize for errors or poor outcomes without the apology being admissible in court. In theory, apologies reduce patient anger and maintain trust, thereby reducing claims. Meaningful data on their efficacy is lacking, although many have called for further exploration.[8]

Malpractice-specific courts

Specialized courts for healthcare have also been proposed, although they have not been adopted in any state. Healthcare courts could provide a layer of consistency to diminish physician concerns about unfair treatment by a jury of lay people. While the jury makeup might not change, the governance of the courts would theoretically be more reproducible.[8] When all considerations are included, the Congressional Budget Office analysis concluded that the total percentage of healthcare expenses that could be saved

through tort reform would be a paltry 0.3%.[11] For this reason, nontraditional approaches, such as communication and resolution, judge-directed negotiation, and administrative compensation systems, are being explored by the Agency for Healthcare Research and Quality.[16]

Medical and Legal Case Outcome

The epidural abscess patient passed away nine months after his emergency department visit. He was paralyzed below the diaphragm for most of that time. It is unclear whether his course would have been any different had the physician made the diagnosis. While there are always areas for improvement, the physician treated him the same way as almost every other doctor would have, and only the most defensive (or brilliant) diagnostician would have ordered an MRI on him. The case was settled for $400,000. The physician did not try to force it to trial.

CONCLUSION

While malpractice serves a social benefit, it also creates stress for physicians and an increase in the practice of defensive medicine. The result is approximately 2.8% of medical expenses being spent to avoid litigation, rather than benefit patients. While this is a small percentage, it is a large dollar amount. Tort reform has limited potential to impact the practice.

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

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Importance of Multiple-window Assessment for the Diagnosis of Ascending Aortic Dissection Using Point-of-care Ultrasound: Report of Three Cases

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Acute ascending aortic dissection has a high mortality rate and requires rapid diagnosis and treatment. Point-of-care ultrasound (POCUS) can aid in the diagnosis. The aortic root is usually evaluated in the parasternal long-axis view; however, a dissection flap is not always visible in this projection. We present three cases of acute, type A aortic dissection in which the dissection flap was only evident in the apical five-chamber and subxyphoid views. These cases suggest that POCUS may play a pivotal role in the initial diagnosis of acute ascending aortic dissection and highlight the importance of viewing multiple windows to fully evaluate this possibility. [Clin Pract Cases Emerg Med. 2019;3(4):333–337.]

INTRODUCTION

Acute ascending aortic dissection (AD) is a potentially catastrophic disease associated with high in-hospital mortality (58%). Even with surgical repair, fatality rates are high (26%); therefore rapid recognition, diagnosis, and treatment is warranted.[1] AD is difficult to diagnose in the emergency department (ED) because it is both a rare clinical condition and one that does not always present in a classic fashion.[2] Routine initial tests readily available in the ED (e.g., electrocardiogram [ECG], chest radiograph (CXR), and laboratory markers) have variable reliability in making a diagnosis of acute AD. Diagnosis typically involves computed tomography (CT) angiography, transesophageal echocardiography (TEE), or magnetic resonance imaging (MRI).[3]

Point-of-care ultrasound (POCUS) has become an invaluable diagnostic tool in the initial evaluation and management of patients with hemodynamic instability, respiratory insufficiency, or chest pain, and it is safe, rapid, and readily available in many EDs and intensive care units (ICU). A point-of-care focused cardiac ultrasound (FOCUS) study may provide useful information when acute aortic pathology is suspected.[4] The American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI) state that the evaluation of the aortic root is best done in the parasternal long-axis view.[5] However, a dissection flap is not always visible in this projection. Still, the emergency physician must consider this possibility given the significant risk of missing the diagnosis. We present a series of three cases of acute, type-A AD in which the TTE performed at admission revealed a dissection flap only in the apical five-chamber and subxyphoid views.

CASE REPORTS

Case 1

A 63-year-old male with a history of hypertension presented to the ED with four hours of chest pain of moderate intensity. Vital signs were blood pressure (BP) 110/47 milligrams of mercury (mmHg), heart rate (HR) 85 beats per minute (BPM), respiratory rate (RR) 16 breaths per minute (bpm), and oxygen (O_2) saturation of 99% at room air. The patient was alert, had no signs of pulmonary or systemic congestion, and had normal cardiac sounds without murmurs. The ECG showed inverted T waves in V4, V5, and V6. Immediate medical management was ordered for an acute coronary syndrome. A FOCUS was performed, which showed a normal functioning left ventricle, but a severely dilated aortic root, measuring 6.1 centimeters (cm) in the parasternal long-axis view. No pericardial fluid was identified. An apical

five-chamber view was used to visualize the ascending aorta, which clearly showed a dissection flap (Image 1, Video 1).

We suspected an acute, type-A AD, and ordered a TEE to confirm the diagnosis. TEE showed a left ventricular ejection fraction (LVEF) of 60-65%, severe aortic insufficiency and a Stanford type-A AD with the dissection flap at 2.3 cm of the valve plane and extending to the descending aorta. The patient was taken urgently to the operating room for a Bentall procedure. He was then transferred to the ICU and ultimately discharged.

Case 2

A 64-year-old woman presented with a chief complaint of sudden chest pain, difficulty breathing and collapse. The patient was initially taken to the nearest hospital where she was found in cardiac arrest and received advanced cardiopulmonary resuscitation for 30 minutes, with subsequent return of spontaneous circulation (ROSC). She was immediately transferred to our hospital for post cardiac arrest management and intensive medical care. Upon arrival, the patient was intubated, and her vital signs included a BP of 40/30 mmHg, HR of 53 BPM, RR of 16 bpm and O_2 saturation of 99% with a fraction of inspired oxygen of 0.5. She had a Glasgow Coma Scale of 6/15, with 3-millimeter (mm) symmetrical and hyporeactive pupils. Heart sounds were regular, rhythmic, with no murmurs, and pulmonary auscultation revealed bilateral rales. After initial medical stabilization in the ED, the patient was transferred to the ICU where she was received by the on-call emergency physician. The FOCUS exam performed at admission to the ICU revealed a hyperdynamic left ventricle, and a normal right ventricle with no signs of ventricular overload; there was no evidence of pericardial fluid, and the inferior vena cava was 1.7 cm with a 50% respiratory collapse.

CPC-EM Capsule

What do we already know about this clinical entity?
Acute ascending aortic dissection is a potentially catastrophic disease. Experts recommend the parasternal long-axis view for ultrasound diagnosis.

What makes this presentation of disease reportable?
In these cases the dissection flap was only evident in the apical five-chamber and subxyphoid views.

What is the major learning point?
This series highlights the importance of viewing multiple windows to fully evaluate the possibility of acute ascending aortic dissection.

How might this improve emergency medicine practice?
Viewing multiple windows may improve the diagnostic accuracy of point-of-care ultrasound to evaluate the possibility of acute aortic dissection.

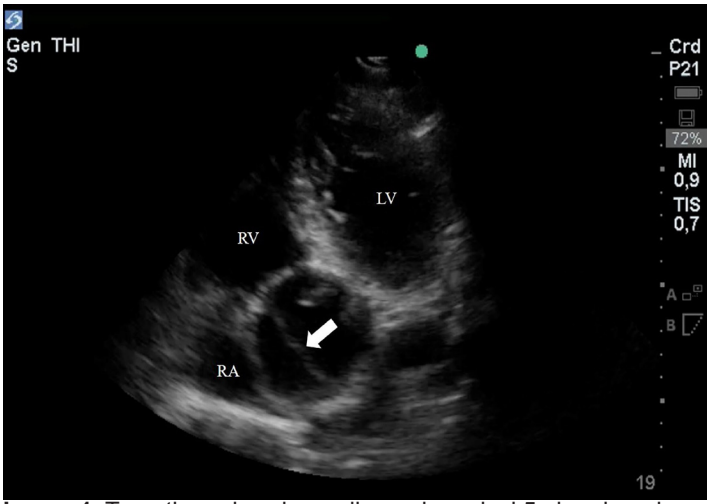

Image 1. Transthoracic echocardiography apical 5-chamber view of a patient with acute chest pain with a dissection flap (white arrow) visualized in the ascending aorta.
LV, Left ventricle; *RV,* right ventricle; *RA,* right atrium.

A dilated aortic root of 4.5 cm was found in the parasternal long-axis view. An apical five-chamber view was used to better visualize the ascending aorta, and a dissection flap was visualized (Image 2, Video 2). We then evaluated the proximal abdominal aorta, which also showed the dissection flap. The patient underwent CT imaging, which confirmed the type-A AD. Unfortunately, she suffered from severe hypoxic encephalopathy due to the prolonged cardiac arrest, and her neurological status did not improve after intensive care medical management. In agreement with the family, the patient was not taken to a surgical repair of the AD, and she died two days after admission.

Case 3

A 77-year-old woman with no past medical history was found lying unconscious. Two hours prior she had complained of severe pain of no clear anatomic location or characteristics. At arrival to the ED she was found to be in profound shock, with severe bradycardia. Her vital signs included a HR of 30 BPM, BP of 50/30 mmHg and an O_2 saturation of 60%. Initial management

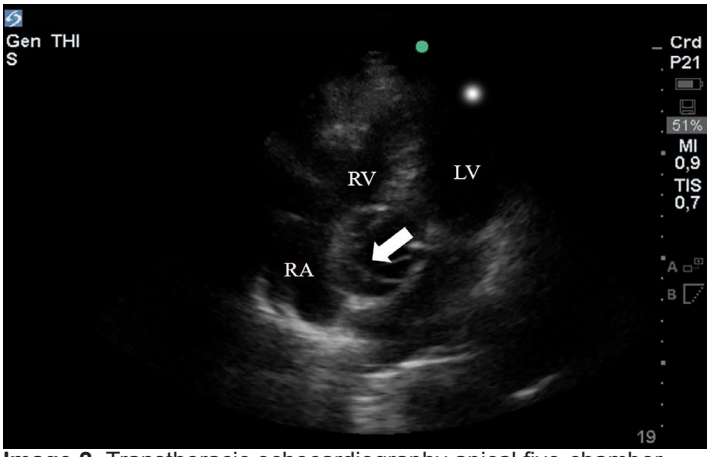

Image 2. Transthoracic echocardiography apical five-chamber view of a patient in post-cardiac arrest with a dissection flap (white arrow) visualized in the ascending aorta.
LV, left ventricle; *RV,* right ventricle; *RA,* right atrium.

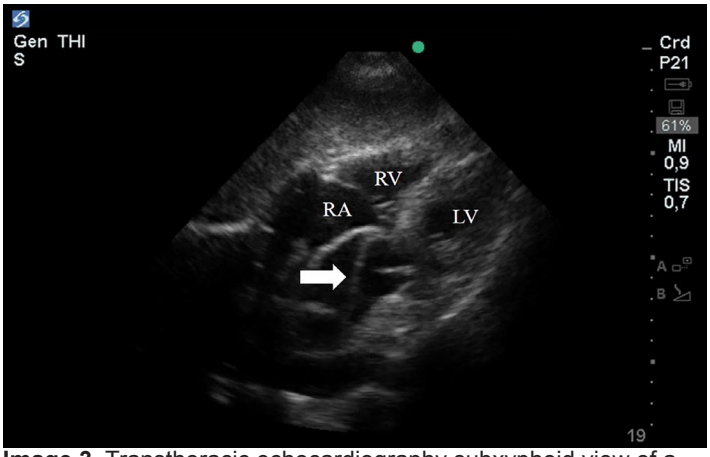

Image 3. Transthoracic echocardiography subxyphoid view of a patient in post-cardiac arrest with a dissection flap (white arrow) visualized in the ascending aorta.
LV, left ventricle; *RV,* right ventricle; *RA,* right atrium.

was started with atropine and dopamine infusion, but the patient became pulseless. Advanced cardiac life support commenced, with ROSC two minutes after active chest compressions.

In the immediate post-arrest period, a POCUS was performed. The subxyphoid window revealed a dissection flap in the ascending aorta (Image 3, Video 3), with extension to the abdominal aorta. TEE confirmed the diagnosis of type-A AD with extension to the abdominal aorta. Cardiovascular surgeons decided on emergent surgical repair. However, the patient persisted with severe hypotension despite high vasopressor support and massive transfusion strategies. Further ultrasonographic evaluation revealed free fluid in the splenorenal pouch, which was absent at arrival, and so aortic rupture was suspected. Her condition continued to deteriorate and, unfortunately, the patient died three hours after admission.

DISCUSSION

Acute AD is an emergent and potentially fatal disorder that can cause complications such as cardiac tamponade, aortic valve insufficiency, or hypoperfusion syndromes. Population-based studies show an incidence of acute dissection from 2 to 3.5 cases per 100,000 person-years.[1] The most common risk condition for AD is hypertension (present in 75% of the cases). Other risk factors include the following: smoking; direct blunt trauma; the use of illicit drugs (such as cocaine or amphetamines); genetic disorders associated with abnormalities of the aortic wall (i.e., Marfan, Loeys-Dietz, and Ehlers-Danlos syndromes); bicuspid aortic valve; and inflammatory or infectious conditions involving the aorta.[1] The International Registry of Aortic Dissection reports an overall in-hospital mortality of 27.4%, with the highest mortality among patients managed medically without surgical intervention (58%). Even those patients taken to immediate surgical repair have a mortality of 26%.[1]

The Stanford classification defines type A and type B ADs as those involving and not involving the ascending aorta, respectively. When the dissection involves the ascending aorta, emergency surgical repair is usually indicated, whereas medical therapy is the initial strategy for acute dissections involving only the descending aorta.[2] AD has a wide range of clinical presentations and may mimic other more common conditions. Patients usually present with chest, back or abdominal pain that is abrupt in onset, severe in intensity, described as ripping or tearing, and can radiate from the chest or back to the abdomen or to the lower extremities. Physical exam is usually unrevealing, but some signs can be encountered such as a pulse deficit, a systolic BP differential between extremities, a focal neurological deficit, a new aortic insufficiency murmur, hypotension, or shock.[1]

ECG changes in AD are usually nonspecific, with 30% of patients showing no abnormalities and 42% showing nonspecific ST-segment and T-wave changes. CXR may show findings suggestive of AD (widening of the mediastinum, widening of the aortic contour, displaced calcification, aortic kinking, and opacification of the aorticopulmonary window) in 88.6% of patients. However, it can be normal in 11.3% of patients; thus, a normal CXR does not exclude AD.[1]

Patients with suspected thoracic AD require early and accurate diagnosis. Aortography has been replaced by less-invasive imaging techniques including TEE, helical CT, and MRI, which have clinically equally reliable diagnostic values for confirming or ruling out thoracic AD with a pooled sensitivity of 98%-100% and specificity of 95%-98%.[6]

TTE is a valuable clinical tool for the assessment of critically ill patients, including those with acute chest pain syndromes where differential diagnoses are broad. The ASE and the ECVI state that the evaluation of the aortic root is best done in the parasternal long-axis view.[5] A dilated aortic root has a sensitivity and specificity of 77-91% and 72-95%, respectively, for the diagnosis of ascending AD. More importantly, visualization of an ascending AD flap by TTE has specificity as high as 98%, therefore becoming a valuable tool to confirm this condition.[7-10] However, a dissection flap is not always visible in the parasternal long-axis view.

As reported in these three cases, the visualization of multiple windows such as the apical five-chambers and subxyphoid views can sometimes reveal intimal abnormalities otherwise not evident. The former can be obtained as an extension to the apical 4-chamber with anterior tilting of the transducer, and the latter with the probe placed in the subxyphoid space, aimed to the left and angled cephalad toward the thorax. If a TTE is suggestive of AD, the clinical policy from the American College of Emergency Physicians recommends immediate surgical consultation or transfer to a higher level of care (Level C recommendation).[11]

Additionally, POCUS can be useful in assessing high-risk features or complications such as pericardial effusion or severe aortic insufficiency and in diagnosing other serious conditions. This diagnostic tool can be of valuable importance for the differential diagnosis of acute chest pain. As illustrated in Case 1, ECG changes could lead to a misdiagnosis of an acute coronary syndrome. Furthermore, starting anti-aggregation, anticoagulation, or thrombolysis could have potentially fatal effects when the underlying disease is an AD. The prompt diagnosis and timely management of this severe aortic pathology could result in better clinical outcomes and reduced costs of care, although this is yet to be proven. Even so, the use of POCUS for the diagnosis of acute ascending AD is increasing.[12,13]

CONCLUSION

POCUS is a valuable tool in the diagnosis and management of critically ill patients. It is safe, rapid, and readily available in many EDs and ICUs. These three cases suggest that POCUS may play a pivotal role in the initial diagnosis of acute ascending aortic dissection and highlight the importance of viewing multiple windows to fully evaluate this possibility. It is our belief that POCUS should be a part of the initial clinical evaluation of patients with hemodynamic instability, chest pain, or respiratory insufficiency to expedite the diagnostic evaluation and to initiate and guide emergent treatment.

Video 1. Transthoracic echocardiography apical 5-chamber view of a patient with acute chest pain with a dissection flap visualized in the ascending aorta.

Video 2. Transthoracic echocardiographyapical five-chamber view of a patient in post-cardiac arrest with a dissection flap visualized in the ascending aorta.

Video 3. Transthoracic echocardiography subxyphoid view of a patient in post-cardiac arrest with a dissection flap visualized in the ascending aorta.

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

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Retinal Artery and Vein Occlusions Successfully Treated with Hyperbaric Oxygen

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We present six cases of central retinal artery occlusion (CRAO) and central retinal vein occlusion (CRVO) that we recently treated with hyperbaric oxygen (HBO_2). Patients in three of the six cases, including the CRVO case, experienced near to complete restoration of their vision. Another case had marked improvement. Our findings are similar to other case studies with approximately 65-70% improvement in patients treated for CRAO. Physicians should be aware that rapid referral of CRAO and CRVO patients to HBO_2 is efficacious. Such patients should be placed on 100% oxygen by non-rebreather mask as soon as the diagnosis is suspected, pending transportation to HBO_2. [Clin Pract Cases Emerg Med. 2019;3(4):338–340.]

INTRODUCTION

Central retinal artery occlusion (CRAO) is an emergent condition, typically presenting as sudden painless visual loss.[1] Risk factors for CRAO include giant cell arteritis, atherosclerosis, atrial fibrillation, and thromboembolic disease. Permanent eye injury usually occurs after two hours of the occlusive event. CRAO is caused by embolism in the central retinal artery. Branch artery occlusion can also occur in the smaller (ciliary) branches of this artery. The occlusion leads to ischemia in the retina leading to pallor and the classic finding of "cherry red" macula due to increased visualization of the macula through the retina.[1]

Treatment methods include ocular massage to dislodge the embolus by creating a pressure differential, medications to decrease intraocular pressure (IOP), increasing partial pressure of carbon dioxide to cause retinal artery dilation with carbogen, a mixture of 95% oxygen and 5% carbon dioxide, intra-arterial fibrinolysis or systemic thrombolytics, and in extreme cases anterior chamber paracentesis to create an acute drop in IOP to dislodge the embolism. None of these interventions have demonstrated significant success.[2] Recently hyperbaric oxygen (HBO_2) has been approved by the Undersea and Hyperbaric Medical Society (UHMS) for treatment of CRAO due to evidence of significant efficacy.[3]

Central retinal vein occlusion (CRVO) has similar presenting symptoms and pathology to CRAO. CRVO is acute monocular vision loss from occlusions of the central retinal vein resulting in edema and ischemia to the retina. This occurs due to the central retinal artery and vein being the principal blood supply and drainage for the retina. Risk factors include hypertension (HTN), diabetes mellitus (DM), glaucoma and hypercoagulable conditions.[4] Treatment considerations are similar to CRAO; however, anti-vascular endothelial growth factor medications are sometimes indicated to prevent macular edema and neovascularization, which can lead to glaucoma. HBO_2 has been a proposed treatment modality for CRVO due to the similarities in pathology when compared to CRAO.

In this article we will discuss six cases, four of which were CRAO, one a branch retinal artery occlusion (BRAO), and one of CRVO. All were treated with HBO_2 with improvement. The protocols used in these cases are from the 13th edition of the UHMS HBO_2 therapy guidelines. Treatments were generally 90 minutes at prescribed pressure, which was 2.5-2.8 atmospheres absolute with air breaks and additional time for compression and decompression.

CASE SERIES

Case One

Patient A, a 73-year-old female with a past medical history of coronary artery disease (CAD), congestive heart failure (CHF), atrial fibrillation, aortic and mitral valve replacement, HTN, hyperlipidemia (HPL), and DM, presented

from an outside facility with acute painless monocular vision loss when bending over to pick something up. The patient was initially evaluated by an ophthalmologist and diagnosed with CRAO. Visual acuity (VA) in the right eye oculus dextrus (OD) was not testable due to blindness; left eye oculus sinister (OS) had baseline visual acuity of 20/25. The patient underwent HBO_2 treatment within 13 hours of last known normal time and tolerated five treatments with improvement in the peripheral visual field; however, the central visual field defect remained.

Case Two

Patient B, a 59-year-old male with a past medical history of HTN, DM, and aortic stenosis with mechanical valve replacement, was admitted for CRAO diagnosed at an outside facility. He underwent HBO_2 treatment within 23 hours of initial injury. VA of OD was zero, OS 20/25. The patient underwent five HBO_2 treatments per protocol and regained some peripheral vision during the hospitalization and upon discharge was able to count fingers (Table).

Case Three

Patient C, a 39-year-old female with a past medical history of anxiety and hypothyroidism, presented with left sided retrobulbar headache. She was diagnosed with paraclinoid internal carotid artery aneurysm and underwent pipeline embolization with full symptomatic improvement and normal visual acuity upon discharge. She presented three days later and was found to have a left BRAO with initial visual acuity of 20/100 in the affected eye. She underwent HBO_2 therapy within 10 hours of initial insult. After five HBO_2 treatments her visual acuity returned to baseline (Table).

CPC-EM Capsule

What do we already know about this clinical entity?
Central retinal artery occlusion (CRAO) is an emergent condition. None of the generally suggested treatment interventions have demonstrated significant success until the recent use of hyperbaric oxygen (HBO_2).

What makes this presentation of disease reportable?
We report six cases, four CRAO, one branch retinal artery occlusion, and one central retinal vein occlusion with varied results and a discussion of the rationale for efficacy.

What is the major learning point?
HBO_2 may salvage vision loss in retinal artery and vein occlusions.

How might this improve emergency medicine practice?
Rapid identification and referral to hyperbaric therapy is important to improve the chances of visual recovery in retinal artery and vein occlusions.

Case Four

Patient D, a 73-year-old male, presented with a history of head injury at age 11 causing blindness in his left eye, peripheral vascular disease, HTN, chronic obstructive pulmonary disease, and DM. He had acute onset of visual loss in his right eye after waking from a nap. He was diagnosed with CRAO at an outside facility. His initial visual acuity could not be obtained due to blindness in both eyes. After three HBO_2 treatments his visual acuity improved to 20/50 OD. The patient was unable to tolerate further HBO_2 treatments due to confinement anxiety (Table).

Case Five

Patient E, a 62-year-old female with a past medical history of HTN, carotid artery stenosis, CAD, and tobacco abuse, presented as a transfer from an outside facility due to right sided painless visual loss. HBO_2 treatment was initiated 23 hours after initial symptom onset. The patient was only able to tolerate two and a half HBO_2 treatments due to confinement anxiety. She was pretreated with lorazepam on her second HBO_2 treatment unsuccessfully, and unfortunately declined further treatment. Visual acuity had improved from light perception to ability to visualize hand motion (Table).

Case Six

Our final patient F, a 46-year-old male, presented with acute painless monocular visual loss in the left eye. Symptoms started 48 hours prior to presentation. This patient had a past medical history significant for HTN, DM, and HLD, and Sjogren's syndrome with previous CRVO of the right eye with blindness. He was found to have a new CRVO in his left eye. He underwent 10 HBO_2 treatments with near-complete improvement in visual acuity to 20/30 at discharge (Table).

DISCUSSION

The above-mentioned cases had improvement in vision with HBO_2 therapy for vaso-occlusive injury to the eye. Patients in three out of the six cases experienced near to complete restoration of their vision. Another case had marked improvement. Confinement anxiety was an issue in two

Table. Summary of cases of sudden monocular blindness treated with hyperbaric oxygen.

Patient	Hours from vision loss to HBO_2	Number of HBO_2 treatments	Outcome	Comment
A	13	5	Some peripheral field improvement only	
B	23	5	Zero vision to finger counting	
C	10	5	Complete resolution	Branch retinal artery occlusion
D	9	3	Improved to 20/50	Confinement anxiety
E	23	2.5	From light perception to ability to visualize hand motion	Confinement anxiety
F	48	10	Near-complete resolution 20/30	Central retinal vein occlusion

HBO_2, hyperbaric oxygen.

cases. Our findings are similar to other case studies with approximately 65-70% improvement in patients treated for CRAO[3] with an excellent result in CRVO.

While the eye is primarily supplied by the retinal artery, there is also some contribution by the choroidal vessels (ciliary arteries). Under normal circumstances, the choroidal supply is inadequate to support the retina; however, under hyperbaric conditions the choroidal circulation can supply the retina with adequate oxygen. This can allow the retina to survive until the retinal arterial (or venous) occlusion resolves via intrinsic thrombolytic mechanisms. HBO_2 also ameliorates subsequent reperfusion effects and edema.[3]

CONCLUSION

Hyperbaric oxygen has established a clear efficacy for treating CRAO. There have been multiple case reports with promising outcomes for CRVO as well.[4-6] Both emergency physicians and ophthalmologists should be aware that rapid referral of CRAO and CRVO patients to HBO_2 therapy is efficacious. Such patients should be placed on 100% oxygen by non-rebreather mask as soon as the diagnosis is suspected[7] pending transportation to HBO_2.

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

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Pennies for Your Thoughts: A Case Series of Pancytopenia Due to Zinc-induced Copper Deficiency in the Same Patient

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A 47-year-old schizophrenic male presented on three separate occasions with pancytopenia and sideroblastic anemia due to copper deficiency from massive zinc penny ingestion. The poisoning was treated differently on each visit: intravenous (IV) copper plus surgical decontamination and chelation with calcium disodium versenate (CaNa2EDTA); IV copper plus whole bowel irrigation; and IV copper with surgical decontamination only. Serum zinc half-lives were 80.0 hours, 233.2 hours, and 83.9 hours, respectively. Importantly, chelation with CaNa2EDTA did not significantly alter the elimination half-life. This is the first reported case of the same patient being treated on three different occasions with three different regimens for this condition. [Clin Pract Cases Emerg Med. 2019;3(4):341–344.]

INTRODUCTION

Copper deficiency due to zinc toxicity from ingested pennies is a rare entity. Since 1982, all United States one-cent coins have been minted from zinc with copper-plating.[1] Zinc-containing pennies are ubiquitous in this country, and their small size makes them easily ingestible. Treatment of zinc toxicity typically includes a combination of gastric decontamination and copper supplementation, with or without calcium disodium versenate ($CaNa_2EDTA$) chelation.[2-10] The existing literature on this subject is currently limited to individual case reports and speculation about ideal treatment. Unfortunately, little comparative data exists to help determine which treatment may be superior for eliminating zinc or even improving clinical outcomes.

Since the elimination kinetics of zinc are not well understood, enhanced elimination practices have not yet been established. It is not known whether chelation confers significant benefit, as there is limited data in humans.[11] We present the case of an individual with recurrent severe zinc poisoning presenting to our institution on three separate occasions, and treated with three different treatment regimens based on variations in his clinical presentation. This natural experiment provides data on zinc elimination associated with different treatment modalities. Pawa et al. published the first presentation and hospital course for this patient in 2008.[9]

CASE REPORT

A 47-year-old male patient with a history of schizophrenia and human immunodeficiency virus presented on three separate occasions with pancytopenia due to copper deficiency resulting from severe zinc toxicity. He was a habitual consumer of pennies, as he felt it improved his singing voice. His first emergency department (ED) visit was precipitated by multiple episodes of syncope. During the initial workup, he was noted to have sideroblastic anemia with hemoglobin of 4.5 grams per deciliter (g/dL), leukopenia, and elevated creatinine level suggesting acute kidney injury. Serum copper was 7 micrograms per deciliter (mcg/dL), and zinc was 2891 mcg/dL.

Abdominal radiographs showed several hundred coins in his upper gastrointestinal (GI) tract. During the initial hospitalization, he rapidly became pancytopenic. On hospital day 10, treatment was initiated with $CaNa_2EDTA$ and copper supplementation, followed by gastrotomy and removal of 212 coins. Many of the coins showed various degrees of dissolution and fragmentation. Approximately 50 coins could not be removed, and they were advanced into the colon during surgery. Zinc levels gradually declined, and the copper level increased. The anemia and renal failure resolved, and the patient was eventually discharged after 27 days of hospitalization.

Eight years later, the same patient presented to the ED with abdominal pain, vomiting, and pancytopenia. Plain radiographs once again showed numerous coins in the GI tract. Copper and zinc levels were noted to be < 3 mcg/dL and 1050 mcg/dL, respectively. On hospital day 2, he was treated with whole bowel irrigation (WBI) via nasogastric tube with one liter per hour of a polyethylene glycol-balanced salt solution (GoLYTELY) for one month (except for a five-day period when hospital stores were depleted). The pancytopenia was treated with intravenous (IV) copper sulfate and granulocyte colony-stimulating factor until his blood counts normalized. The patient defecated over 200 pennies during the course of his hospital stay. His zinc levels came down gradually, and his pancytopenia resolved. He was discharged home after 33 days of hospitalization.

Approximately 10 months later, routine outpatient blood work for this patient again showed pancytopenia, with low serum copper (< 3 mcg/dL) and elevated serum zinc levels (965 mcg/dL). Abdominal radiographs demonstrated multiple coins in the stomach, as well as a sharp-pointed screw. To avoid perforation he was taken to the operating room for foreign body removal. A total of 180 pennies and the screw were successfully retrieved with gastrotomy. IV copper was administered, the pancytopenia resolved, and the serum zinc concentration normalized. The patient was discharged home after 21 days hospitalization. Laboratory values from the three hospitalizations for this patient are provided in summary form in Table 1.

Data from all three hospitalizations were used to determine zinc pharmacokinetics. For half-life calculations, the initial zinc value was defined as the value most proximal to the initiation of treatment for zinc intoxication. The final zinc value was defined as that serum zinc level closest to one-fourth of the initial serum zinc concentration. For purposes of elimination kinetics, this was determined to represent two half-lives. Data from the half-life calculations for the three modalities are provided in Table 2.

CPC-EM Capsule

What do we already know about this clinical entity?
Zinc poisoning is known to cause sideroblastic anemia. Chelation treatment has been suggested, but neither the benefit of chelation nor the best treatment are known.

What makes this presentation of disease reportable?
This is the first published report examining the effects of different treatment regimens for zinc poisoning in the same patient.

What is the major learning point?
Of the treatment options used, early surgical removal of the zinc had the greatest effect on the zinc levels. Copper supplementation also may be beneficial.

How might this improve emergency medicine practice?
Patients with zinc poisoning may present very ill. This case provides some guidance on early treatment of this unusual disease entity.

DISCUSSION

Copper deficiency caused by zinc intoxication with sideroblastic anemia and pancytopenia has been previous described.[8,12-15] Because of complex interactions at absorptive sites in the small intestine, zinc toxicity can also lead to secondary copper deficiency. Copper and zinc are competitively absorbed in the proximal small intestine,[16] and either can remain as a free metal or become bound to metallothionein (MT) and stored within enterocytes. Zinc bound to MT is excreted through the fecal route, within sloughed intestinal cells.[16] The unbound zinc molecule, however, can be absorbed into the circulation and is unchanged with renal excretion.[16] MT binds to copper with greater affinity than to zinc, and the MT-copper (Cu) complex is preferentially retained in the intestinal cells.[16]

Table 1. Serum laboratory values on each of the three visits for zinc toxicity.

Admission number	Treatment	Time (h)	Initial Zn (mcg/dL)	Final Zn (mcg/dL)	Elimination half-life (h)
1	Chelation + Surgery	163.0	1720	419	80.0
2	WBI	467.0	1050	262	233.2**
3	WBI + Surgery	147.3	965	286	83.9

** There are two conflicting samples for this calculation, and we suspect that there was an incorrect time stamp on one of them. If we use the alternative sample, which was drawn after initiation of whole bowel irrigation, this value drops to 162 hours.
h, hours; *Zn,* zinc; *mcg/dL,* micrograms per deciliter; *WBI,* whole bowel irrigation.

Table 2. Elimination kinetics for the three modalities of treatment.

Laboratory value	Visit 1	Visit 2	Visit 3	Normal range
Hemoglobin (g/dL)	4.5	4.6	4.0	13.3 - 17.1
Creatinine (mg/dL)	1.5	n/a	n/a	0.6 - 1.1
WBC (K/mm^3)	2.5	1.1	1.4	3.5 - 10.6
Platelet count (K/mm^3)	125	89	149	150 - 460
Copper (mcg/dL)	7	<3	<3	70 - 155
Zinc (mcg/dL)	2891	1050	965	60 - 130

g/dL, grams per deciliter; *mg/dL,* micrograms per deciliter; *WBC,* white blood cells; *K/mm^3,* thousand cells per cubic millimeter; *mcg/dL,* micrograms per deciliter; *n/a,* not available.

The synthesis of MT is regulated by the amount of zinc ingested. When large amounts of zinc are ingested, more MT proteins are produced, forming more MT-Cu complexes, which are subsequently excreted.[16] Massive zinc ingestion thereby decreases copper absorption, and leads to an increase in copper excretion.[14,16] Since copper is a necessary cofactor for hematopoiesis,[17] it is believed that copper deficiency prevents hematopoietic progenitor cells from replicating and differentiating, resulting in pancytopenia.[18]

Although zinc intoxication with pancytopenia due to massive ingestion of zinc-containing coins has been previously reported, the optimal treatment has not yet been established. The mortality risk associated with elemental zinc poisoning is also unknown, although a few previous case reports in humans have been associated with patient death.[3,19] Based on a single case report[2] chelation with $CaNa_2EDTA$ has also been suggested as a viable treatment. In that case, the patient was treated with surgery and eventually with chelation, but ultimately developed sepsis and died. It does not appear that the patient had ever received copper supplementation.[3]

Parenteral copper supplementation is another treatment option, as IV copper bypasses GI absorption. This may prevent copper binding with MT within the enterocytes and subsequent loss through intestinal cell sloughing. Based on the mechanism of zinc-induced copper deficiency, parenteral copper may improve the rate of immune reconstitution by supplementing copper stores for hematopoiesis. Our patient received IV copper supplementation with each hospitalization and normalized his white blood cell counts within one week of initiation of treatment, despite a significant zinc burden.

In our patient, it appears that the addition of $CaNa_2EDTA$ offered little benefit to enhance the elimination of zinc. The elimination rate associated with surgery alone is quite similar (80.0 vs 83.9 hours) to that for surgery plus $CaNa_2EDTA$ chelation. Theoretically, $CaNa_2EDTA$ chelation is believed to bind free zinc and prevent or reduce further MT expression, thereby reducing copper excretion. However, this kind of benefit could not be supported by these data.

CONCLUSION

With the benefit of multiple presentations of the same patient with recurrent zinc toxicity, we were able to compare zinc elimination with whole bowel irrigation, surgery and surgery plus $CaNa_2EDTA$ chelation. Surgical removal of the zinc source (in these cases, copper-plated zinc U.S. pennies) seems to be the most important factor related to zinc elimination. Whole bowel irrigation alone resulted in a much longer elimination half-life. Chelation with $CaNa_2EDTA$ seemed to have little impact on the overall elimination of absorbed zinc. As parenteral exogenous copper was provided during each hospital admission, the effects of copper supplementation cannot be quantified based on these data. In the one previously reported fatality from massive coin ingestion, copper was not supplemented.[3]

Based upon the mechanism of zinc-induced copper deficiency, parenteral copper supplementation may be of benefit in restoring hematopoeisis, by bypassing the GI tract. However, there is currently minimal data to support or refute its use in treating this disease entity. At this time, the optimal treatment for zinc intoxication resulting in copper deficiency from massive ingestion remains unknown. Based on our experience, however, surgical decontamination with parenteral copper supplementation should be considered. Furthermore, based upon this limited case series with the same patient, decontamination with whole bowel irrigation may be inferior to surgery, and chelation with $CaNa_2EDTA$ may offer no improvement in elimination half-life.

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

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A Case of Central Venous Sinus Thrombosis in a Young Woman

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Altered mental status is a common symptom in emergency department evaluations and may be present in as many as four to ten percent of patients.[1] The etiology can be difficult to determine without significant evidence from laboratory, radiographic and physical examination. The diagnostic approach is largely driven by the provider's clinical judgment based on the available history. Consequently, less-common diagnoses can be easily missed or delayed if a reasonable suspicion does not exist when considering possible causes. Cerebral venous sinus thrombosis (CVST) is one such uncommon, seldom-considered disease that carries a significant morbidity and mortality. Its clinical presentations vary and it disproportionally affects young to middle-aged individuals. Knowledge of the disease, particularly the risk factors, is key to making the diagnosis. We will discuss the case of a patient who presented with CVST and intraparenchymal hemorrhage in a resource-limited environment. [Clin Pract Cases Emerg Med. 2019;3(4):345–348.]

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is a relatively rare yet clinically significant disease that can affect young patients in the prime of their lives. Its presentations can range from the subtle to the dramatic and profound. However, if identified in a timely manner, appropriate treatment results in full recovery in many cases.[2] The current gold standard for diagnosis of CVST is magnetic resonance imaging venography (MRV), although computed tomography venography (CTV) was shown to have nearly comparable capability to identify CVST.[3] This is important for settings in which MRV is not readily available in order to expedite disposition and initiation of anticoagulant therapy. Here we discuss the case of an otherwise healthy female who presented with intraparenchymal hemorrhage and altered mental status (AMS) as a result of CVST. With appropriate anticoagulation therapy, she made a full recovery.

CASE REPORT

A 31-year-old female, with a past medical history significant for a right upper extremity deep venous thrombosis a decade prior, presented to the emergency department (ED) with a chief complaint of AMS. The history was obtained from the patient's husband as her condition allowed only minimal participation in the history and physical exam. Symptoms had developed over the preceding 24 hours, beginning with a mild headache and progressing to stupor, aphasia, and finally urinary incontinence. The husband stated the symptoms began gradually, adding that the patient's only medication was an oral, estrogen-containing contraceptive. She had been in her usual state of health prior to onset of symptoms in the prior 24 hours.

The patient was afebrile with vital signs in the normal range for her age. Her physical exam was notable for a well-nourished, well-developed young woman without apparent signs of distress. There were no focal abnormalities to her neurologic exam noted in the ED. There were no signs of trauma on her physical exam. Her Glasgow Coma Score (GCS) was 10; she was aroused only to painful stimuli and was not following commands. Her blood glucose was within normal limits and there was no response to empiric dose of intravenous (IV) naloxone. Her laboratory workup in the ED was unremarkable.

The patient was deemed stable enough to go to the radiology suite. A frontal lobe intraparenchymal hemorrhage with evidence of early trans-tentorial herniation was noted on non-contrast CT (Image). The differential diagnosis included an arterial-venous malformation, tumor, or a hemorrhagic conversion of a CVST.

In the ED she was treated with mannitol, IV dexamethasone, and levetiracetam for seizure prophylaxis.

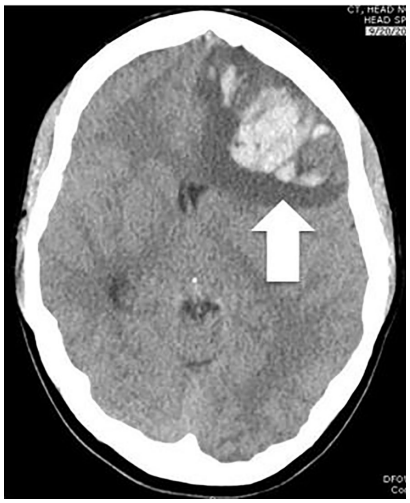

Image. Non-contrast computed tomography of the patient demonstrating a frontal lobe intraparenchymal hemorrhage (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Central venous sinus thrombosis (CVST) is an uncommon, but clinically significant cause of headache that affects the majority of patients in early adulthood.

What makes this presentation of disease reportable?
Our patient manifested CVST as altered mental status on her index visit to the emergency department. This is an uncommon and potentially confounding presentation.

What is the major learning point?
Given a high index of suspicion, appropriate imaging modalities (magnetic resonance venography, computed tomography venography) will help assure timely diagnosis.

How might this improve emergency medicine practice?
Awareness of the varied presentations of CVST and the best imaging modalities for its diagnosis contributes to a general understanding of treatment options.

The on-call neurosurgeon was consulted to see the patient in the ED, and the decision was made to proceed to the operating room. The patient was admitted to the neurosurgical service and underwent a decompressive craniectomy. When she was stable postoperatively the diagnosis of a superior sagittal sinus thrombosis was made with MRV. This imaging was delayed 24 hours from time of presentation secondary to availability of specialized radiology technicians.

She was started on a heparin infusion postoperatively after the diagnosis was confirmed. Following surgery the patient was transferred to a major transfer receiving hospital for postoperative and rehabilitation care. The patient had an excellent recovery. She was discharged from the hospital functionally independent and able to return to work in her previous career field.

DISCUSSION

CVST is a relatively rare but clinically significant disease. The overall incidence in the adult population is approximately 1.32 per 100,000-person years.[4] Mortality attributed to CVST ranks between 5.5-18% in recent series. Those patients who receive a timely diagnosis have a good chance for recovery; between 57-86% of patients achieve complete functional recovery.[5]

CVST is slightly more common in women, particularly in the 20- to 35-year-old age group. This difference is likely secondary to hypercoagulability associated with pregnancy and oral contraceptive use.[6] The mean age of presentation in several large studies was 38 years of age.[6] Hypercoagulable states are likely the major, and may be the only, identifiable risk factor for CVST. A Dutch study found an age-adjusted odds ratio (OR) of 13 for oral contraceptive use and risk of CVST.[7] Ten hereditary prothrombotic conditions such as Factor V Leiden, deficiency of proteins C and S, and antithrombin III may account for 10-15% of cases of CVST.[7] The OR for women using oral contraceptives who also carry a diagnosis of a prothrombotic defect was calculated at 30.[7]

Unlike in our case where AMS was the chief complaint, often a simple headache is the presenting symptom in 70–90% of cases.[3] More dramatic presentations such as stroke-like symptoms including focal deficits such as hemiparesis and hemisensory disturbance, seizures, impairment of level of consciousness, and papilledema occur in one-third of cases.[3] Intracerebral hemorrhage occurs in 35-39% of patients suffering CVST.[8]

The diagnosis of CVST can be elusive. As many as one in 18 cases of CVST are misdiagnosed at the index visit to the ED as measured by ED return visits.[9] Classically MRV has been thought to be the gold standard for diagnosis; however, institutional and clinical constraints may make obtaining this study impossible. Magnetic resonance imaging (MRI) with gradient echo T2* susceptibility-weighted sequences with MRV has a higher sensitivity for detection of CVST and also has the advantage of demonstrating age-dependent signal features.[10] However, it is not

without its own limitations, particularly as a result of mimicry of sinus thrombosis by normal anatomic variants such as sinus atresia or hypoplasia, asymmetric sinus drainage, and filling defects as a result of arachnoid granulations or intra-sinus septa.[3] In such cases, the inclusion of digital subtraction angiography (DSA) may be necessary to establish the diagnosis as well as to rule out dural arteriovenous fistulas and distal aneurysms prior to the initiation of anticoagulant therapy in cases where subarachnoid hemorrhage is also present.[10]

Non-contrast CT is near universal in its availability, which often makes CT the first imaging study for headaches if imaging is indicated. Unfortunately, changes indicative of CVST are only visible on approximately 30% of non-contrast CTs.[3] One visible abnormality is the dense/filled delta sign, a hyperdensity in the posterior portion of the superior sagittal sinus. Other changes that may be apparent on contrast-enhanced CT include the "empty delta sign," reflecting the opacification of collateral veins in the wall of the superior sagittal sinus, and the "cord sign," a curvilinear hyperdensity over the cerebral cortex due to thrombosed cortical veins.[7]

When access to MRV is limited it is widely accepted that CTV is a reasonable alternative in the identification of CVST with sensitivity and specificity of 95% and 91%, respectively, when compared to DSA.[3] Additionally, advantages of CTV include timely image acquisition, no contraindication to pacemaker or ferromagnetic hardware, increased image resolution, and fewer equivocal findings per some reports.[10] It is also less invasive than diagnostic tests such as DSA. Conversely, it would be a less suitable alternative should the patient have iodine-contrast material allergies or poor renal function, limiting the ability to introduce contrast, while the intrinsic factor of radiation exposure also exists.[3]

Of note, lumbar puncture and cerebral spinal fluid (CSF) analysis is likely unhelpful in establishing a primary diagnosis of CVST. Abnormalities are nonspecific (increased opening pressure, increased red blood cell counts, increased protein content, and pleocytosis), occurring in 84% of cases without pathognomonic features.[2] Rather, CSF analysis is likely helpful at narrowing the differential diagnosis for CVST by identifying clinical mimics such as meningitis and subarachnoid hemorrhage.

Once diagnosed, initial management in CVST is focused on correcting and managing serious complications such as increased intracranial pressure to prevent herniation, seizures, and stroke. As was the case with our patient, decompressive craniectomy may be required if left untreated or if mass effect resulting in a herniation syndrome occurs.[11]

Definitive treatment for CVST is generally focused on anticoagulation with thrombolysis. Surgical management is reserved for the most severe cases. While both unfractionated (UFH) and low molecular weight heparin (LMWH) have been demonstrated to be effective, UFH is more appropriate if surgical intervention may be needed and rapid reversal is desired.[11-13] Meta-analysis of two randomized controlled trials with a total of 79 patients showed no statistically significant relative risk of death or dependence when patients were treated with IV UFH or subcutaneous nadroparin, a LMWH (relative risk 0.46, 95% confidence interval, 0.16 to 1.31).[3] In one randomized controlled trial, anticoagulation showed treatment benefit. Moreover, no extension of hemorrhages present on initial evaluation or new hemorrhages were observed.[14]

Systemic thrombolysis, catheter-directed thrombolysis, and mechanical thrombectomy may be used for cases refractory to anticoagulation. Li G et al. treated 52 patients with severe CVST using mechanical thrombectomy combined with injecting urokinase via a catheter and reported 87% of these patients achieved complete recanalization.[15]

When CVST results in poor outcomes, they are often associated with risk factors such as central nervous system infection, malignancy, thrombosis of the deep venous system, intracranial hemorrhage, GCS score < 9, mental status disturbance, age > 37 years, and male sex.[3] Recurrence, however, is rare at 2.8 %.[16]

CONCLUSION

This case demonstrates the severity with which CVST may present and how outcomes can be favorable with timely diagnosis and treatment. If risk factors are present in the setting of atraumatic brain bleeds, providers should maintain a reasonable degree of suspicion for CVST. When appropriate, CTV may be used as an acceptable alternative to MRV if the latter is not readily available. Such practice will enable providers in rural and resource-limited facilities to provide care more efficiently for patients with this rare but potentially life-altering condition.

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

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Point-of-care Transperineal Ultrasound to Diagnose Abscess in the Emergency Department

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Perineal and rectal pain are common presentations in the emergency department (ED). In the majority of cases, clinical examination is sufficient to detect local anorectal pathologies. However, perianal and rectal abscesses and fistulas are often the primary concerns prompting diagnostic imaging in the ED. Currently, computed tomography is the preferred imaging modality. Recently, transperineal ultrasound has emerged as an optimal imaging modality for the diagnosis of perineal and perianal abscesses. We present a case in which point-of-care ultrasound accurately detected an intersphincteric abscess, and review the appropriate ultrasound technique to evaluate patients with suspected perianal and rectal abscesses. [Clin Pract Cases Emerg Med. 2019;3(4):349–353.]

INTRODUCTION

Uncomplicated anorectal abscesses are typically diagnosed during clinical examination without the need for further imaging. Along with the symptoms of localized pain, mass effect, and erythematous swelling, many of these patients have a visible abscess on the external perianal skin.[1] Infection typically arises when bacteria or fecal matter obstruct crypto-globular glands, causing either superficial or deep abscesses to form.[1,2] Specifically, these abscesses arise either between the internal and external anal sphincters (intersphincteric abscesses), or externally on the skin.[3] In deeper abscesses, such as intersphincteric abscesses, computed tomography (CT) is the most common imaging modality to detect the presence and extension of the abscess.[4] However, CT shortcomings in the diagnosis of perirectal abscesses have been reported in multiple studies.

In a retrospective study by Caliste et al. CT missed 23% of abscesses that were confirmed with surgery.[4] In this study, the sensitivity of CT in detecting perirectal abscesses in immunocompetent patients was 77%.[4] Magnetic resonance imaging (MRI) and endorectal ultrasonography are alternative imaging modalities with higher accuracy. However, their use in the emergency department (ED) is limited, partly because of high cost, inconvenience to patient and physician, significant time needed to complete the exam, and lack of availability in the ED.[5-8]

In recent years, there has been increasing interest in transperineal ultrasound (TPUS) for multiple applications including the diagnosis of perineal and perirectal abscesses and fistulas.[9] In the ED, a point-of-care TPUS can be employed favorably due to low cost, wide availability, lack of ionizing radiation, and minimal inconvenience and discomfort to patients. Due to these benefits, TPUS has the potential to be included in the diagnostic workup of perianal abscess and rectal discomfort as the initial imaging modality of choice in the ED. Despite these advantages, this ultrasound technique is currently underused in the ED due in part to inadequate provider familiarity with the technique.[10] Appropriate training is essential to enable more emergency physicians and patients to reap the benefits of TPUS.[10] This article highlights the utility and feasibility of TPUS in a patient presenting to the ED with a chief complaint of rectal pain.

CASE REPORT

A 52-year-old male, with no past medical history, presented to the ED with a four-day history of rectal pain and generalized body pain. He reported that his rectal pain was worse with

ambulation and bowel movements. He endorsed changes in bowel habits and constipation that began two days before his ED presentation. He denied fever, chills, nausea, vomiting, abdominal pain, lower extremity pain, swelling, dysuria, hematuria, and hematochezia. Of note, the patient had recently returned from Ethiopia. He was not taking any medication on a regular basis. He denied alcohol or tobacco use. His vital signs were as follows: blood pressure 159/77 millimeters of mercury, heart rate 88 beats per minute, temperature 98.4° Fahrenheit, respiratory rate 16 breaths per minute, and peripheral oxygen saturation 99% on room air.

On exam, he was with no acute distress. His abdominal exam revealed a soft, non-distended, and non-tender abdomen. A digital rectal examination revealed normal anal sphincter tone with a small fissure located in the anterior line of the anal canal, and tenderness to palpation. No hemorrhoids or rectal masses were detected in the digital rectal exam. Guaiac test was negative. The remainder of his physical exam was unremarkable. An anoscopic examination was deferred because of patient discomfort. Laboratory testing was as follows: white blood cell count 13.24 × 10^9/liter (L) (4.5 to 11.0 × 10^9/L), hemoglobin 14.5 grams per deciliter (g/dL) (13.5 to 17.5 g/dL), platelet count 276 × 10^3/ microliter (μL) (150-400 × 10^3 /uL). His comprehensive metabolic panel was unremarkable.

At this juncture, we performed a point-of-care TPUS that showed a discrete intersphincteric abscess (Image 1 and Video) confirmed by subsequent CT (Image 2). Surgical consult

CPC-EM Capsule

What do we already know about this clinical entity?
Although clinical examination is usually sufficient to detect anorectal pathology, computed tomography imaging is generally performed when the provider suspects a perianal or rectal abscess.

What makes this presentation of disease reportable?
Providers can utilize point-of-care ultrasound (POCUS) to detect perineal and perianal abscesses in the emergency department (ED) which can expedite appropriate consultation and treatment.

What is the major learning point?
In cases where the provider suspects an anorectal abscess POCUS may be used as a quick and accurate initial imaging study for patients in the ED.

How might this improve emergency medicine practice?
With appropriate training for providers, POCUS could become the preferred diagnostic imaging modality to look for perineal pathologies in the ED.

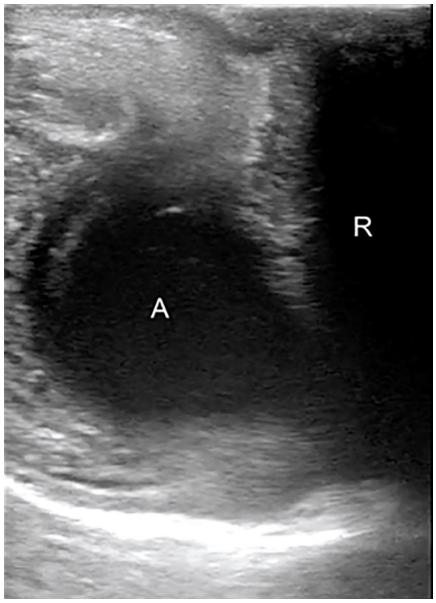

Image 1. Sagittal sonographic view of the upper anal canal with space-occupying hypoechoic lesion (A) adjacent to the posterior canal and possible connection to the anal canal (R).
R, rectal ampulla; *A,* abscess.

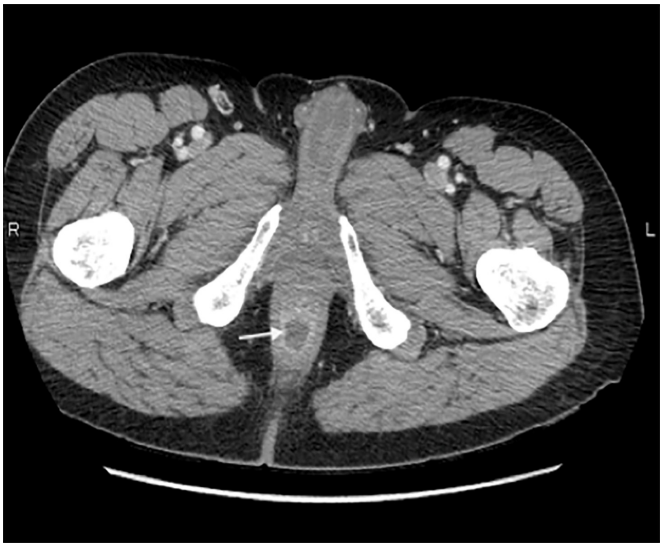

Image 2. Axial computed tomography shows the intersphincteric abscess measuring 2.0 x 1.8 centimeters posterior to the anal canal (arrow).

in the ED recommended antibiotic therapy, gastrointestinal decompression, anti-inflammatory treatment, and supportive rehydration therapy. The patient was discharged home with a plan to return to the outpatient clinic for his follow-up.

The ultrasound examination was performed using SonoSite X-Porte machine (SonoSite, Bothell, WA) with 5-1 megahertz (MHz) phased array and 13-6 MHz linear transducers. Scanning was performed with the patient in the lateral decubitus position with the upper hip fully flexed. To obtain a mid-sagittal view, the phased array and linear transducers were placed on the perineum with the probe orientation marker directed toward the symphysis pubis (cranioventral). Adhesive Tegaderm transparent film was applied to cover the transducers.[11,12]

A coronal transperineal ultrasound of the anal canal showed a hypoechoic anal canal. A discrete, space-occupying hypoechoic perianal lesion was seen adjacent to the posterior anal canal measuring 1.8 × 1.5 centimeters (cm), with a possible connection to the posterior wall (Image 1 and Video). Color Doppler scanning showed no vascularity to the lesion; no extension to the perineal skin was detected. The possibility of perianal abscess was considered. Further scanning in transverse view was performed by obtaining images with the linear transducer directly over the external anal sphincter. This provides an image of the lumen of the anorectal canal, and surrounding soft tissue.

DISCUSSION

TPUS has the potential to be used as the initial diagnostic imaging modality for perineal pathologies in the ED (Table). Several factors contribute to its increasing popularity, the most important being the availability of ultrasound at the bedside for a quick, convenient, and reliable scan. In this case, performing a TPUS accurately detected an intersphincteric abscess with possible connection to rectum, which was confirmed by CT as a follow-up study. Currently, there is little evidence to determine the accuracy of TPUS in the ED. However, several studies suggest high sensitivity and specificity in different clinical settings.[13-18]

In a recent study by Wedemeyer et al. 25 patients were evaluated to examine the use of TPUS in diagnosing perianal inflammatory disease. They compared images obtained from TPUS and MRI, and found that perianal abscesses were correctly diagnosed by TPUS in 28% (7/25) of the patients. In 24% (6/25) of the patients, MRI confirmed the presence of an abscess. However, with one patient MRI failed to correctly diagnose the abscess. TPUS did not miss any abscesses that were identified via MRI.[15] In summary, sonography demonstrated high accuracy in the identification of perianal abscesses.

To further investigate the use of TPUS compared to CT and MRI, Rubens et al. recruited patients to examine the general anatomy and pathology of the rectal region. The authors noted that both CT and MRI lacked spatial resolution abilities, were more expensive, and did not have the real-time imaging capabilities of sonography. When examining a perirectal abscess in a 45-year-old patient with rectal pain, the author used an axial TPUS, which revealed a 2 × 2.5 cm hypoechoic collection. Another longitudinal superficial sonogram demonstrated a 4-cm collection of fluid, which later required surgical intervention. This study endorsed TPUS as the preferred imaging tool when the pathology can be visualized through this sonographic approach.[16] Similarly, Chandwani et al. recommended point-of-care ultrasound as a diagnostic tool to confirm the presence of anorectal abscess, to visualize the spread of infection, and to properly drain the abscess in the ED.[17]

In a prospective observational study contrasting TPUS and MRI, Plaikner et al. recruited 30 patients who underwent both MRI and TPUS to compare the effectiveness and accuracy of the different modalities. In six cases out of 30 the diagnoses by MRI did not match those made by TPUS. Four cases were correctly diagnosed by TPUS, and two by MRI.[18] While MRI still has a place in the diagnosis of perianal diseases, TPUS demonstrates at least similar efficacy. The authors concluded that TPUS should be considered whenever anorectal symptoms occur.[18]

Despite the apparent advantages of TPUS, as outlined in multiple studies (Table), TPUS is currently underused in the acute clinical setting.[14-18] One potential reason is the lack of training most providers receive in imaging anorectal pathologies. It is important to consider that a substantial "learning curve" exists with TPUS because of the complex anatomy being imaged, and because of the substantial variability in patient presentation and location of rectal abscesses. In deeper abscesses the use of a high-frequency transducer is limited by its shallower penetration, which hinders visualizing deep structures. Nonetheless, Hwang et al. suggested that sonographers can become competent after completing the exam on just 12 patients.[19] Therefore, this major limitation of TPUS may be addressed with minimal additional provider training.

CONCLUSION

The majority of patients with anorectal pathology can be evaluated without a need for further imaging in the ED. However, in cases with potential risk of anorectal abscess, TPUS may be used as a quick and accurate initial imaging study for patients in the ED. TPUS has been proven to be a reliable and useful imaging modality in different clinical settings and can be used at the bedside by emergency physicians with proper training. It is important, however, to keep in mind that a "learning curve" exists with TPUS because of the complex perineal anatomy and limitations of high-frequency transducers to accurately detect deeper

Table. Perianal abscess assessment with transperineal ultrasound versus magnetic resonance imaging/computed tomography.

Study	Design	No	Findings	Conclusions
Caliste et al.[4]	Retrospective observational	113	Among patients with a confirmed perianal abscess, CT was negative in 23% of patients. The overall sensitivity of CT in the identification of perirectal abscesses was 77%.	CT lacks sensitivity in the diagnosis of perirectal abscess. This imaging tool missed nearly 25% of surgically confirmed perirectal abscesses. Therefore, another adjunct imaging modality is necessary to increase diagnostic accuracy.
Mallouhi et al.[5]	Prospective observational	87	Gray scale sonography had good accuracy in the detection and characterization of perianal inflammatory disease. For the detection of perianal abscesses, gray scale sonography sensitivity and specificity was 100% and 94%, respectively. With the addition of color Doppler sonography, accuracy in the diagnosis of perianal inflammatory disease increased.	Grey scale and color Doppler sonography have high detectability of both perianal abscesses and fistulas. When used together, these two imaging tools have increased diagnostic confidence.
Domkundwar and Shinagare[6]	Prospective observational	30	In 30 patients with confirmed anal fistulas, TPUS correctly identified 11 patients with abscesses (37%). Abscesses were hypoechoic and anechoic collections visualized on sonography.	TPUS has the potential to become the first imaging tool to diagnose patients with perianal fistulas and abscesses. Specifically, TPUS allows accurate detection of perianal abscesses. TPUS is easily available, allows real-time visualization, can be used in patients with anal stenosis, and requires no special equipment. TPUS is especially helpful when immediate diagnosis is needed, and when a more detailed imaging modality (CT and MRI) would delay diagnosis.
Stewart et al.[13]	Prospective observational	54	TPUS accurately identified perianal fistulas and abscesses in 46 patients. Specifically, TPUS diagnosed 15 abscesses correctly; 26 patients with perianal fistulas and abscesses underwent surgery following TPUS. Surgery confirmed 85% of TPUS findings.	At the Toronto General Hospital, TPUS has been implemented as the primary routine procedure to evaluate patients with any disease in the perianal region.
Chandwani et al.[17]	Case study	1	In a patient with chief complaint of rectal pain with tenesmus, point-of-care ultrasound with a 5.0 MHz curvilinear probe correctly identified a 3.6cm perianal abscess.	Using point-of-care ultrasound in the emergency department has recently increased in popularity. This imaging tool is well suited for the evaluation of patients with symptomology reflecting a potential perirectal abscess.
Plaikner et al.[18]	Prospective observational	67	36 abscesses were detected by MRI, 38 by TPUS, and 30 by surgical examination. When comparing TPUS and MRI, there was good agreement with the diagnosis of perianal abscess.	Transabdominal ultrasonography (TAS) had increased accuracy in the diagnosis of superficial rectal infections, while MRI was more suited for the identification of deeper perirectal infections.
Hwang et al.[19]	Prospective observational	43	In 43 pediatric Crohn's patients, 18.8% of TAS examinations revealed rectal abscesses; 75% of these abscesses were associated with active fistulas.	TAS and color Doppler sonography is advantageous in the evaluation of perianal fistulas and abscesses in pediatric patients with Crohn's disease.

TPUS, transperineal ultrasound; *MRI,* magnetic resonance imaging; *CT,* computed tomography; *Mhz,* megahertz; *cm,* centimeters; *TAS,* transabdominal ultrasonography.

structures. Further studies to investigate the feasibility and the accuracy of this method in ED patients are recommended.

Video. A discrete, space-occupying hypoechoic perianal lesion is seen adjacent to the posterior anal canal measuring 1.8 x 1.5 centimeters, with a possible connection to the posterior wall.

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

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Cardiac Arrhythmia Following an Epileptic Seizure

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Sudden unexplained death in epilepsy (SUDEP) refers to a death in a patient with epilepsy that is not due to trauma, drowning, status epilepticus, or another apparent cause. Although the pathophysiology of SUDEP is incompletely understood, growing evidence supports the role of seizure-associated arrhythmias as a potential etiology. We present a unique case of a patient presenting with ventricular tachycardia shortly following a seizure, along with corresponding laboratory data. Awareness of high risk arrhythmias in seizure patients could lead to advances in understanding pathophysiology and treatment of this complication of seizure disorder and ultimately prevention of SUDEP. [Clin Pract Cases Emerg Med. 2019;3(4):354–356.]

INTRODUCTION

Sudden unexpected death in epilepsy (SUDEP) refers to death in a patient with epilepsy that is not due to trauma, drowning, status epilepticus, or another known cause for which there is often evidence of an associated seizure. It has been estimated that SUDEP accounts for approximately 10% of deaths in patients with seizure disorder each year in the United States, and accounts for 20-30% of epilepsy-related deaths, although this may differ across study populations.[1-2] Typically the patient is found dead, and the diagnosis is confirmed only when clinical criteria are met and autopsy reveals no alternative cause of death.[1-2] The etiology of SUDEP is uncertain, although observations in individual cases suggest possible cardiogenic, pulmonary, and/or primary neurologic pathophysiologic mechanisms.[3-4] We present a case of a patient presenting with ventricular tachycardia immediately following a seizure, along with corresponding laboratory data.

CASE REPORT

A 17-year-old male with a history of cognitive impairment and epilepsy presented via emergency medical services (EMS) to the emergency department (ED) following a witnessed seizure. Upon arrival, the patient was noted to be postictal; thus, history was obtained from the patient's mother who reported that he had experienced symptoms consistent with a generalized tonic-clonic seizure earlier in the day. It was unknown if the patient returned to baseline; however, the mother offered that following this seizure, he did state that he needed a refill of his home levetiracetam. She called EMS for transport to the ED. Upon EMS arrival, the patient suffered another seizure, described by EMS as generalized tonic-clonic activity lasting approximately three minutes. The patient was found sitting on the couch and subsequently fell forward onto the ground. EMS administered five milligrams (mg) of intramuscular midazolam and then established intravenous (IV) access. He had no further seizure-like activity during transport or upon arrival to the ED.

An electrocardiogram obtained during transport documented a monomorphic ventricular tachycardia (Image 1). Shortly thereafter, this rhythm degenerated into a polymorphic ventricular tachycardia (Image 2).

The patient arrived to the ED in normal sinus rhythm. During transport, he was not witnessed to have seizure activity, nor did he lose pulses. No further medications or interventions were administered or performed, and no additional arrhythmias were noted during hospitalization.

Upon arrival to the ED, the patient was somnolent, moving all extremities spontaneously, and responsive only to pain. A point-of-care venous blood gas was obtained and significant for a pH of 6.78 (normal 7.35-7.38), carbon dioxide (CO_2) 100 millimeters of mercury (mmHg) (normal 44-48 mmHg), a normal

blood glucose, and lactate of 13.8 mg per deciliter (dL) (normal <2.0 mg/dL). The patient received two liters of IV fluids and a two-gram IV loading dose of levetiracetam. The patient was also placed on supplemental oxygen by nasal cannula. Subsequent blood gases thereafter showed improvement in both metabolic and respiratory acidosis (pH 7.0, CO_2 62 mmHg, lactate 10 mg/dL). The patient's high-sensitivity troponin T resulted at 22 nanogram per liter (ng/L) (normal <14 ng/L). The magnesium, potassium, and calcium levels were normal. Electrocardiogram obtained in the ED was significant for sinus tachycardia and a corrected QT interval of 398 milliseconds.

The patient was admitted to the pediatric intensive care unit and underwent correction of all metabolic abnormalities. Later, the serum levetiracetam resulted at <2.0 micrograms per milliliter (ug/mL) (therapeutic levels 12.0-46.0 ug/mL) and the valproic acid level at 112 ug/mL (therapeutic levels 50-125 ug/mL). The patient was seen by the pediatric cardiology team, had a normal pediatric echocardiogram, and was discharged home with a 30-day cardiac monitoring device, ultimately demonstrating no additional arrhythmias.

CPC-EM Capsule

What do we already know about this clinical entity?
Sudden, Unexplained Death in Epilepsy (SUDEP) accounts for death in 20-30% of patients with epilepsy. However, the pathophysiology is not well understood.

What makes this presentation of disease reportable?
Pre-hospital rhythm strips captured a cardiac arrhythmia in a patient following a seizure, suggesting that SUDEP may be caused by a cardiac arrhythmia.

What is the major learning point?
Emergency department providers must be aware of cardiac arrhythmias or near-SUDEP in patients immediately following a seizure.

How might this improve emergency medicine practice?
With better understanding of SUDEP and cardiac arrhythmias in post-ictal patients, providers can take measures to prevent apnea and correct subsequent metabolic derangements.

DISCUSSION

Meta-analysis of peri-ictal cardiac arrhythmias reveals that ictal asystole, ictal bradycardia, and postictal atrial flutter/fibrillation are the most common presenting arrhythmias related to seizure, and are often self-limiting.[4] Proposed mechanisms include direct stimulation of the central autonomic network (i.e., cingulated gyrus, amygdala, or insular cortex) and seizure-induced catecholamine release leading to vasovagal responses.[3] Both peri-ictal ventricular tachycardia and fibrillation have been described in epilepsy patients with no underlying cardiac disease.[5] It has also been suggested that pathologic cardiac repolarization (including QT prolongation, QT shortening, and increased dispersion) is responsible for tachyarrthymias in these patients, and ultimately leads to SUDEP.[5] Recent data also suggests a significant association between potentially high risk cardiac arrhythmias and the duration of ictal/postictal oxygen desaturation.[6] Ultimately, cerebral anoxia from asystole ceases seizure activity.

The above case describes a patient with postictal ventricular tachycardia in the setting of markedly deranged blood gas values, pointing to respiratory dysfunction and subsequent metabolic and respiratory acidosis as an additional plausible etiology of SUDEP. However, cardiac arrhythmias sometimes coincide with epileptic seizures, and some individuals may have components of both cardiogenic syncope and epilepsy. In a recent case report of a patient presenting to an ED with a seizure, automatic implanted cardioverter defibrillator interrogation revealed ventricular

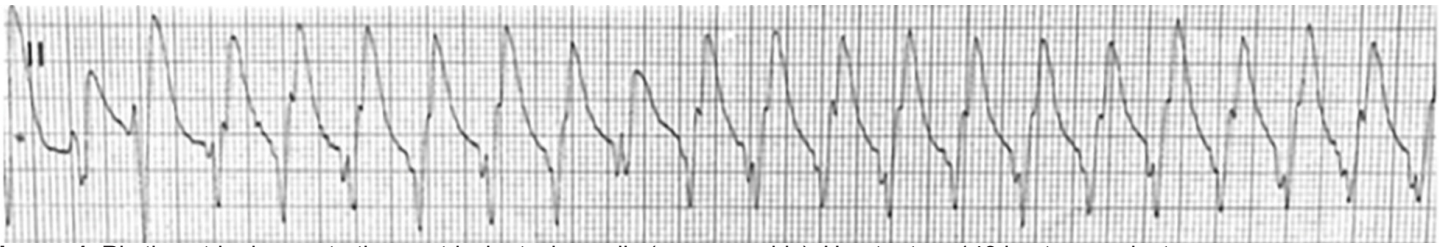

Image 1. Rhythm strip demonstrating ventricular tachycardia (monomorphic). Heart rate = 143 beats per minute.

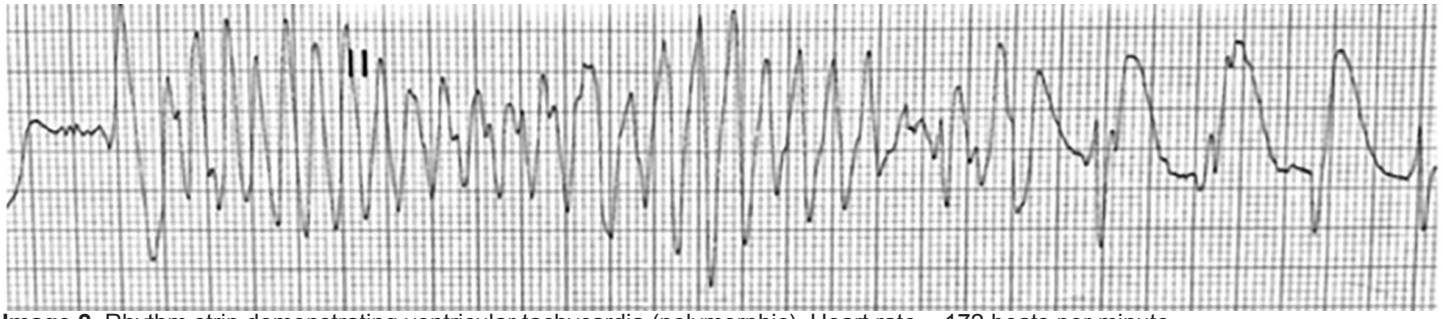

Image 2. Rhythm strip demonstrating ventricular tachycardia (polymorphic). Heart rate = 172 beats per minute.

tachycardia and fibrillation.[8] In our case, this patient had no recurrence of cardiac arrhythmias following interventions as described above, and fortunately did not succumb to SUDEP.

In the seizing patient, measures must be taken to avoid apnea and correct hypoxemia and metabolic derangements. ED providers must be aware of cardiac arrhythmias or near-SUDEP following a seizure. Early nursing interventions, including administration of supplemental oxygen, oropharyngeal suctioning, and patient repositioning have been shown to reduce the duration of respiratory-induced hypoxemia.[7] Providers should also be aware of the use of benzodiazepines as a potential cause of worsening respiratory depression and subsequent respiratory acidosis, leading to the development of arrhythmias. Finally, in postictal patients, physicians should consider electrocardiograms and continuous telemetry monitoring while rapidly reversing metabolic derangements.

CONCLUSION

More knowledge about the cardiovascular status of epileptic patients during, between, and immediately after seizures is needed to better understand and prevent high-risk arrhythmias and SUDEP by measures such as cardioprotective drugs, respiratory therapy, or implantation of a defibrillator.[3] Specifically, more knowledge and awareness of this phenomenon in the emergency medicine community is necessary to best care for these patients in the acute setting.

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

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Acute Toxin-mediated Rhabdomyolysis During Treatment With Trimethoprim-sulfamethoxazole

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Rhabdomyolysis is a condition in which skeletal muscle breakdown causes the release of intracellular components into the bloodstream – defined as elevations in serum creatine kinase levels. The etiology of rhabdomyolysis is varied and may be the result of toxin-mediated mechanisms or metabolic derangements, or they may develop secondary to other conditions such as seizures, trauma and prolonged immobilization. In this case, we present a patient with suspected acute toxin-mediated rhabdomyolysis in the setting of trimethoprim-sulfamethoxazole (TMP-SMX) therapy for urinary tract infection. To our knowledge, this marks the fifth case report of an otherwise healthy patient diagnosed with rhabdomyolysis thought to be secondary to TMP-SMX. [Clin Pract Cases Emerg Med. 2019;3(4):357–360.]

INTRODUCTION

While not an uncommon diagnosis in the emergency department, rhabdomyolysis is often difficult to diagnose clinically and may present in a variety of contexts and clinical scenarios. Patients often present with non-specific generalized myalgias, aches, fatigue, urine color changes, and/or fevers. Others present without symptoms. Rhabdomyolysis presents on a spectrum from asymptomatic elevations in serum creatine kinase (CK) to life-threatening acute electrolyte disturbances requiring hemodialysis.[1-2]

Identification of rhabdomyolysis usually begins in the proper clinical context based on history and physical, a suspected mechanism, and elevations in CK detected on laboratory analysis. Although an elevation of CK levels at least five-fold greater than the upper limit of normal is a commonly accepted parameter for definitive diagnosis, no true consensus exists.[3-4] The etiology of rhabdomyolysis is varied; it may be the result of pharmacologic or biologic toxin-mediated mechanisms, or metabolic derangements, or it may develop secondary to other conditions such as seizures, trauma, and prolonged immobilization.[1-3] When considering the precipitating mechanism, it is helpful to consider four main categories: ischemic, pharmacologic, physical, and biologic.[1] Commonly cited medications with a known association with rhabdomyolysis include statins and fibrates. Recreational drugs, psychiatric mediations, and a variety of antimicrobial agents have also been associated with acute rhabdomyolysis.[1,2,5]

In this case, we present a patient with suspected acute toxin-mediated rhabdomyolysis in the setting of new trimethoprim-sulfamethoxazole (TMP-SMX) therapy for urinary tract infection (UTI). While rhabdomyolysis in the setting of TMP-SMX has been well described in the immunocompromised, no widespread association exists with immunocompetent patients. To our knowledge, this marks the fifth case report of an otherwise healthy, immunocompetent patient diagnosed with rhabdomyolysis thought secondary to TMP-SMX with no other clear insult or mechanism to explain elevations in CK levels.

CASE REPORT

A 41-year-old African American male with a past medical history of overactive bladder, benign prostatic hyperplasia, obstructive sleep apnea requiring nocturnal home continuous positive airway pressure, presented to the emergency department

(ED) with a chief complaint of increasing proximal bilateral lower extremity muscle pain for five days. Pain was associated with increasing difficulty with ambulation. The patient also noted persisting hematuria, urgency and hesitancy for the prior nine days. He had fever to 101.2° Fahrenheit (F) for the prior five days. Symptoms were associated with intermittent episodes of nausea and vomiting for the prior two days. Four days prior to presentation, the patient reported having seen his outpatient urologist who diagnosed a UTI. He was prescribed TMP-SMX double strength 160 milligrams (mg)/800mg. and endorsed medication compliance.

On ED arrival, the patient was found to be febrile to 102.3°F and hemodynamically stable. On exam, he appeared grossly uncomfortable. The bilateral lower extremity proximal muscle strength was 4/5 without point tenderness. There was no evidence of lower extremity edema, erythema or rash. Distal pulses were intact. The patient was also noted to have mild left lower quadrant abdominal and suprapubic tenderness to palpation; there was no costovertebral angle tenderness to palpation. Physical exam was otherwise unremarkable.

Initial laboratory analysis showed an aspartate transaminase (AST) 274 units per liter (u/L) (reference range, 4-40 u/L), alanine transaminase (ALT) 102 u/L (reference range, 4-41 u/L), CK muscle/brain 3.11 nanograms per milliliter (ng/mL) (reference range, 1-6.6 ng/mL) with a CK 60,665 ng/mL (reference range, 30-200 u/L). Labs obtained from the patient's outpatient urologist from four days prior to presentation showed an initial leukocytosis to 14.4 10^3 per cubic millimeter (mm^3), which resolved prior to ED arrival, a urine analysis consistent with UTI: positive large blood with greater than 50 red blood cells per high-powered field, 50 white blood cells per high-powered field, and positive leukocyte esterase. A urine culture was positive for *Escherichia coli* sensitive to TMP-SMX per the outpatient urologist. The outpatient provider did not request CK levels at initial visit, but at that time AST/ALT levels were 22 u/L and 30 u/L, respectively.

Chest radiograph and computed tomography of the abdomen and pelvis in the ED were negative for acute pulmonary or intra-abdominal pathologies. In the ED, the patient received one gram of ceftriaxone for UTI, two liters of normal saline, acetaminophen for fever, and morphine for pain control. TMP-SMX was discontinued. He was admitted to the inpatient internal medicine service with a concern for non-traumatic acute rhabdomyolysis and pyelonephritis. Further workup by the inpatient medicine team excluded other possible causes for acute rhabdomyolysis. Respiratory viral panel and human immunodeficiency virus studies were negative. Urine toxicology was positive for opiates. Epstein-Barr viral studies were consistent with a prior infection with immunity. Thyroid and glucose-6-phosphate dehydrogenase studies were normal. CK and AST levels peaked on days two and three of admission, respectively (Figure).

The patient continued to receive intravenous and oral fluids as well as pain control. Over the eight days of admission the patient continued to improve. He reported decreasing muscle pain and was increasingly able to ambulate without difficulty. During the course of his hospital stay, the patient sustained no electrolyte abnormalities, and creatinine and blood urea nitrogen were within normal limits. There was no evidence of acute kidney injury. The patient was discharged on day eight with resolution of symptoms and a down-trending of CK and AST/ALT.

CPC-EM Capsule

What do we already know about this clinical entity?
Rhabdomyolysis may develop secondary to metabolic derangements, seizures, trauma, or prolonged immobilization, or via a toxin-mediated mechanism.

What makes this presentation of disease reportable?
We present a case of trimethoprim-sulfamethoxazole (TMP-SMX) induced rhabdomyolysis in an otherwise healthy patient.

What is the major learning point?
In otherwise unclear etiologies of rhabdomyolysis in applicable patients, recent TMP-SMX use should be considered as a possible precipitating insult.

How might this improve emergency medicine practice?
Early recognition of this possible emerging association between TMP-SMX and rhabdomyolysis in immunocompetent patients may alter emergency department management and treatment.

DISCUSSION

Trimethoprim-sulfamethoxazole is a bacterial folic acid synthesis inhibitor used to treat a variety of conditions including UTI, skin and soft tissue infections in healthy patients, and pneumocystis pneumonia or toxoplasmosis in the immunocompromised patient. Side effects generally are mild and include rash, fever, muscle aches and diarrhea. TMP-SMX is also known to have an association with more severe side effects including Steven-Johnson syndrome and toxic epidermal necrolysis, although these are quite rare.[6] TMP-SMX induced rhabdomyolysis is more often seen in the setting of immunocompromised patients or in the setting of allogeneic stem

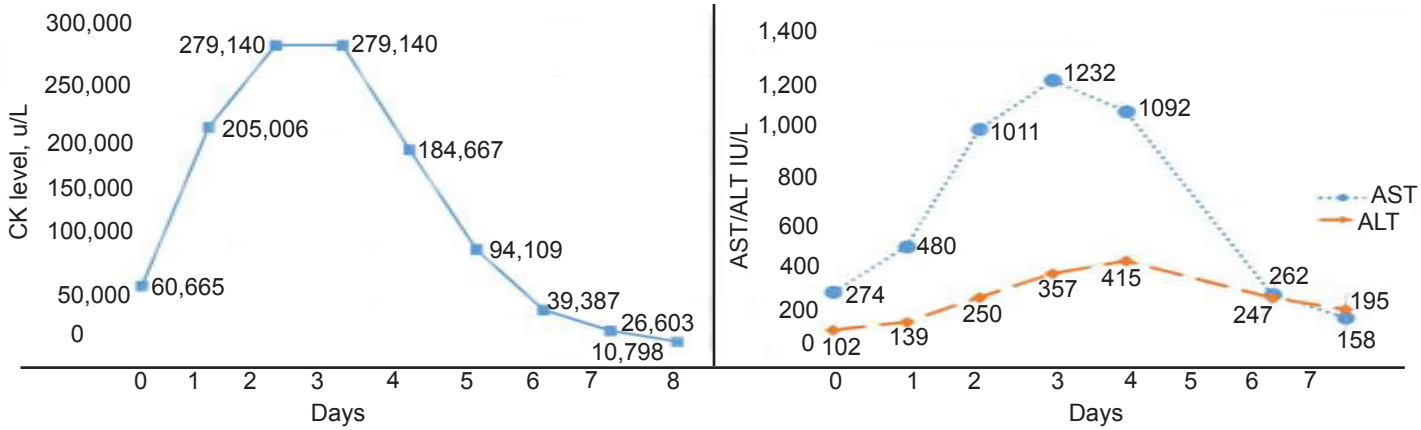

Figure. Creatinine kinase (CK), aspartate transaminase (AST) and alanine transaminase (ALT) trends during admission.

cell transplant recipients.[7-8] To our knowledge, this report now represents the fifth case of TMP-SMX induced rhabdomyolysis in the setting of an otherwise healthy, immunocompetent patient.

In 2014 Ainapurapu et al. reported a case of a previously healthy 40-year-old Hispanic female. She initially presented with two days of bilateral lower extremity muscle aches and weakness. She reported she was prescribed TMP-SMX for a UTI two days prior and had taken four doses. She presented with an initial CK of 20063 u/L, with an otherwise negative workup for other etiologies explaining her rhabdomyolysis.[8]

A second case in 2015 by Petrov et al. was that of a 64-year-old male, active smoker, who also presented complaining of bilateral lower extremity pain and a decreasing ability to ambulate secondary to discomfort. The patient reported self-treating a presumed UTI with TMP-SMX left over from a prior episode of UTI. He reported taking TMP-SMX 40mg/800mg three times a day for two weeks. On first presentation to the ED, the patient was found to have a CK of 1524 u/L, and he was referred to a neurologist with suspicion for polymyositis. TMP-SMX was not discontinued and the patient's pain continued to worsen. The patient also was prescribed ibuprofen, celecoxib, piroxicam, ketorolac, and meloxicam. On subsequent presentations to the ED, the patient was found to have a CK of 614,691 u/L, elevated transaminases, and multiple electrolyte derangements. Muscle biopsy was suggestive of muscle fibers without striations and a loss of homogeneity. Other causes of rhabdomyolysis were ruled out. TMP-SMX was discontinued, and over the course of admission the patient reported improvement in all symptomatology and was subsequently discharged.[9]

In 2017, Moye et al. presented a case of a 43-year-old African American female with a past medical history of depression and alcohol-use disorder who presented to the ED after eight days of bilateral lower extremity leg pain in the setting of taking five doses of TMP-SMX 160mg/800mg for a UTI. She reported a history of daily alcohol consumption and 10mg vortioxetine. She first began to experience leg pain two days after initiation of TMP-SMX when she re-presented to her primary care provider. There she was switched to ciprofloxacin. The patient continued to experience worsening leg pain, which prompted her visit to the ED. There she was found to have a CK of 26,231 u/L with elevated transaminases AST/ALT of 352/111 u/L, respectively.[10] She was admitted, started on intravenous (IV) fluids and folic acid supplementation. During her admission, CK peaked at 45,020 u/L without increase in serum creatinine. She was discharged on day eight after improvement in her lower extremity discomfort and with a CK of 2809 u/L.

Also in 2017, Goyal et al. presented a case of an 18-year-old female with a past medical history of Arnold-Chiari malformation presenting from her primary care provider (PCP) complaining of severe weakness and diffuse muscle pains in all extremities for two days. She endorsed recent completion of a course of TMP-SMX for UTI from her PCP. At time of admission, she was noted to have severe proximal muscle weakness in all four extremities and diffuse muscle tenderness. On admission her CK level was 20,418 u/L, which peaked the next day to greater than 42,670 u/L, the maximal quantifiable limit per the institution's laboratory. She initially received aggressive fluid resuscitation without improvement. Ultimately, she was started on IV immunoglobulin G with eventual resolution of elevations in CK.

There are a few concerning similarities with each of these presentations. In each case TMP-SMX was initiated for a presumed UTI. Subsequently, each patient presented complaining of bilateral lower extremity proximal muscle weakness or pain. Each case demonstrated elevations in CK consistent with rhabdomyolysis. TMP-SMX was discontinued resulting in improvement of clinical status and resolution of CK levels. Each patient had a negative workup for other etiologies of rhabdomyolysis and was without concern for immunocompromised state.

There are certainly possible confounding factors in each patient presentation. Confounders include but are not limited to pre-existing medical conditions and past medical history,

outpatient prescription medications, home self-medication, substance abuse and non-modifiable factors including gender and age. However, the overarching consistencies – indication for therapy, the temporal relationship to initiation of TMP-SMX therapy, and chief complaint at presentation – are noteworthy and are fairly consistent between cases.

CONCLUSION

After evaluating for and eliminating other possible toxic, biologic or physical and traumatic etiologies of acute rhabdomyolysis in our patient, we concluded that TMP-SMX was the most likely insulting agent. Furthermore, while serum CK was not obtained prior to initiation of TMP-SMX, the temporal relationship of normal AST four days prior to presentation – and parallel trending of AST/ALT with CK at time of presentation and during admission– suggests that our patient's rising AST/ALT was likely secondary to skeletal muscle breakdown and may serve as a surrogate for CK. TMP-SMX was discontinued and over the course of admission the CK, proximal lower extremity myalgia, ability to ambulate, and overall clinical status gradually improved.

In each of the four prior cases, as with ours, the indication for TMP-SMX therapy was a UTI. Our case report adds to a growing body of anecdotal evidence that suggests TMP-SMX may be associated with acute rhabdomyolysis in immunocompetent patients. While the authors are not aware of any large, multicenter study evaluating for a true association of TMP-SMX with rhabdomyolysis, additional larger scale studies are warranted. Providers should be made aware of this possible emerging association between TMP-SMX and rhabdomyolysis in immunocompetent patients.

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

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Mango Dermatitis After Urushiol Sensitization

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Prior exposure to poison ivy and poison oak, which are plants in the Anacardiacea family and contain high levels of urushiol, appear to be a risk factor for delayed hypersensitivity reactions to mango fruits. Cross-sensitization between these plants and mangos is believed to be secondary to an overlap in the urushiol antigen and 5-resorcinol, found predominately in mango peels. This unique combination of sensitization and reaction constitutes a type IV hypersensitivity response, mediated and driven by T cells reacting to similar antigens. We present a case of an otherwise healthy man, with a remote history of poison ivy exposure, who presented with a delayed but significant reaction to mango fruit. Obtaining the patient's history of prior plant exposures and reactions was key to isolating the likely underlying causation of his presentation. [Clin Pract Cases Emerg Med. 2019;3(3):361–363.]

INTRODUCTION

Urushiol is an allergenic substance found in the Anacardiaceae family, most commonly known for poison ivy (*Toxicodendron radicans*) and poison oak (*T. diversilobum*).[1] Presentations of patients who come in direct contact to urushiol-containing plants include pruritus, erythema, vesicles, bullae, and localized edema. While systemic reactions like anaphylaxis can cause airway compromise, the burning and inhalation of urushiol-containing plants can cause acute airway complications such as tracheitis and pulmonary edema.[2] Although contact dermatitis and airway reactions are well documented in the literature, a lesser-known reaction from prior urushiol exposure is hypersensitization to mango fruits. We present a case of a 41-year-old man with suspected mango dermatitis, incited by mango handling after remote exposure to poison ivy.

CASE REPORT

An otherwise healthy 41-year-old man presented to the emergency department (ED) with a severely worsening, four-day-old diffuse, pruritic rash, which began in the inguinal regions bilaterally but had since spread to his trunk and extremities. The patient's primary complaint was insomnia secondary to his pruritis. He initially denied any new medications or other exposures but did endorse a distant episode of contact dermatitis to poison ivy two years prior. On arrival, his vitals included temperature of 98.3º Fahrenheit, blood pressure of 133/87 millimeters of mercury, heart rate of 69 beats per minute, respirations of 18 per minute, and a pulse oximetry of 100% on room air.

The patient's physical exam was remarkable for a macular, blanching, non-vesicular, erythematous rash on all extremities, chest, and back, sparing the palms, soles, and oral mucosa. Lungs were clear to auscultation in all fields. Further diet history detailed consumption of two mangos two days prior to the onset of the rash. The patient's wife, who accompanied him to the ED, had also consumed mangos two days prior but was asymptomatic. Although both the patient and his wife handled the mango peels, the wife did not endorse prior plant exposures resulting in rash.

Given the patient's substantial discomfort, intravenous (IV) access was established, and 50 milligrams (mg) of diphenhydramine and 50 mg ranitidine were parenterally administered. This resulted in significant relief of pruritus and mild improvement in visible rash. After a brief observation period, the patient was discharged home on 60 mg of oral prednisone for five days and 20 mg of oral loratadine, as needed. Of note, the patient deferred the first steroid dose in the ED due to the evening time of presentation and a remote history of insomnia after taking steroids. He was contacted five days after his ED visit with almost complete resolution of symptoms and significant improvement in insomnia with over-the-counter (OTC) oral diphenhydramine. Extending steroid treatment for an additional week was discussed, but the patient declined based on the improvement of his symptoms.

He was contacted again approximately three months after his ED visit and denied any rebound symptoms.

CPC-EM Capsule

What do we already know about this clinical entity?
Urushiol is an allergen of poison ivy and poison oak. Prior exposure to it can cause a type 4 hypersensitivity response upon subsequent handling of mango peels.

What makes this presentation of disease reportable?
We describe a delayed, nonspecific dermatitis whose etiology was discovered only upon obtaining a detailed history of prior allergic reactions to poison ivy.

What is the major learning point?
Patients who previously experienced hypersensitivity reactions to poison ivy or poison oak are at risk for hypersensitivity reactions when handling mango peels.

How might this improve emergency medicine practice?
When approaching an undifferentiated rash, an accurate history of prior reactions and allergies can help identify the etiology of the rash.

DISCUSSION

The classic hypersensitivity framework of the Gel and Coombs system defines four main classes of reactions: types I-IV. In brief, type I reactions are mediated by antigens cross-linking immunoglobulin (Ig) E, causing mast cells and basophils to release histamine and other vasoactive contents.[3] Type I responses range from seasonal allergies to asthma to the extreme of anaphylaxis. Type II reactions are predominately mediated by IgG and IgM, stimulating phagocytes and natural killer cells to either uptake IgG and IgM tagged antigens or active complement.[3] Examples of type II responses include hemolytic anemia and basement membrane disease. Type III reactions are also mediated by IgG but cause pathology by the formation of immune complexes.[3] These complexes deposit around small vessels and tissues and can manifest in diseases such as systemic lupus erythematosus and glomerulonephritis. Finally, type IV reactions are T-cell mediated. After initial sensitization to an antigen, T-cells release damaging cytokines upon subsequent exposure to structurally similar antigens.[4] Unlike types I-III, type IV responses do not involve Igs. These responses can manifest from contact dermatitis to Stevens-Johnson syndrome.[4]

Urushiol is a well-known hapten to skin proteins that induces a type IV hypersensitivity response.[2] After initial sensitization to urushiol, typically from contact with poison ivy or poison oak, subsequent exposures to urushiol produce a cell-mediated memory response after two to three days.[2] Limited studies demonstrated a cross-hypersensitivity response between urushiol and the mango compound, 5-resorcinol, found predominantly in the skin, leaves, and stems of mango fruits.[5] 5-resorcinol, along with other phenols, are collectively known as "mango latex," which acts as a preservative with anti-microbial properties.[5] Hershko et al. identified that mango pickers with severe rashes had prior exposures to poison ivy or poison oak when compared to pickers with mild or no rashes working in the same conditions.[6] Interestingly, these allergens appear to be negligible in the actual fruit of the mango, and patients with a history of mango dermatitis may still enjoy the fruit if peeled by another person.[7] Although the pathophysiology of this cross-reaction is not well described, understanding this phenomenon is important due to the abundance of mango fruits worldwide.

Management of mango dermatitis and contact dermatitis from poison ivy or poison oak is nearly identical and primarily entails avoidance of inciting factors and symptomatic treatment. Post-exposure, patients should be advised to gently rinse the affected area with cold, soapy water, ideally within 30 minutes, to minimize dermal absorption. Cool compresses and calamine lotions are OTC options for symptomatic management.[1] Adjunct systemic corticosteroids are indicated in moderate to severe dermatitis, typically dosed at 1 mg per kilogram per day (kg/day) for 14-21 days.[1] Although the length of corticosteroid therapy is not extensively studied, the prolonged course is recommended over shorter courses to prevent rebound dermatitis.[8]

Of note, atopic dermatitis (a type I hypersensitivity response) and contact dermatitis (a type IV hypersensitivity response) can present similarly, and understanding the differences between the two can guide treatment, such as the use of antihistamines, and identifying inciting factors. This patient was given IV antihistamines on arrival, to provide symptomatic relief for a presumed, undifferentiated allergic reaction that disrupted the patient's sleep. Based on our current understanding of pruritus, especially in non-histamine mediated pruritus (i.e., contact dermatitis), the use of either IV or oral antihistamines has limited evidence for use. However, antihistamines can provide a sedating effect that may be useful in select patients who are unable to sleep secondary to extreme pruritus.[9]

CONCLUSION

Rashes are common presentations to the ED. While most are non-anaphylactic and therefore generally non-emergent, key dietary and exposure histories are helpful in determining the etiology and treatment of undifferentiated rashes in the ED setting. Clinicians should recognize that cross-reactions between allergens are frequent and remain suspicious about cross-hypersensitivity reactions in any patient with a known history of allergic reactions.

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

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Fatal Tension Hemothorax Combined With Exanguination: A Rare Complication of Neurofibromatosis

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Neurofibromatosis (NF) is a common autosomal dominant disorder that can be subdivided into type 1, type 2, and schwannomatosis. Patients with NF1 typically develop café-au-lait spots, scoliosis, and benign neurofibromas. In addition, NF1 predisposes to vascular complications including stenosis, arterial ectasia, and aneurysms. Here, we report the case of an otherwise healthy 32-year-old man who developed a fatal tension hemothorax due to vertebral artery aneurysm rupture. Based on the available literature, we discuss the presentation, workup, and available therapeutic approaches to this complication of neurofibromatosis. [Clin Pract Cases Emerg Med. 2019;3(4):364–368.]

INTRODUCTION

Approximately 100,000 individuals in the United States have neurofibromatosis (NF), an autosomal dominant disorder categorized as type 1 (NF1), type 2 (NF2), or schwannomatosis (SWN). The most common form, NF1, affects about 1 in 3,000 people in the U.S. and manifests by the age of 10; it is due to spontaneous mutation in almost 50% of cases.[1] Symptoms of NF1 present as skin abnormalities including café-au-lait spots, Lisch nodules and freckling in the axilla and inguinal regions. Between 0.4-6.4% of patients with NF1 develop vascular abnormalities, including arterial ectasia, stenosis, and aneurysms.[1]

The vascular malformations as well as the friable tissues of the innumerable tumors predispose to spontaneous hemorrhage.[2] Neurofibromata can erode into the vascular system, or bleeding can occur from the vascular supply of the tumors. [3] Due to the abundance of vessels in the thoracic cavity, patients with NF1 are at risk of developing a spontaneous hemothorax. Bleeding into the thoracic cavity may present with chest pain or shortness of breath developed from reduced tidal volume.[4] We report a case of a fatal tension hemothorax due to extrathoracic aneurysmal rupture in a patient with NF1.

CASE REPORT

A 32-year-old man with pertinent past medical history of NF1 and remote surgical spinal fusion (C3-T11, performed 11 years prior) presented to the emergency department (ED) with an acute exacerbation of his chronic neck pain without focal neurological complaints. The exacerbation occurred the previous day, and the patient unsuccessfully attempted to control his symptoms with prescribed opioid analgesics. He had experienced multiple syncopal events with movement, which he attributed to the intensity of his pain.

Initial vital signs showed sinus tachycardia at 125 beats per minute and tachypnea at 35 breaths per minute. The patient was afebrile (36.3 ° Celsius), normotensive (121/72 millimeters of mercury (mmHg), and oxygenated well (pulse oximetry oxygen saturation 97%) on room air. Physical exam showed a well-developed but ill-appearing young man. He was pale with reduced breath sounds over the right hemithorax. On cardiovascular exam, jugular venous distention, murmur, or peripheral edema were absent. There was no external evidence of trauma. A rectal exam was negative for blood.

Initial laboratory evaluation showed significant leukocytosis at 35.8 k/mm^3 (thousand cells per cubic millimeter) (4.0-10.4 k/mm^3), hemoglobin 9.9 grams per deciliter (g/dL) (13.8-17.3 g/dL), lactate 9.2 millimoles per liter (mmol/L) (<2.1 mmol/L), creatinine 1.8 milligrams (mg) per deciliter (mg/dL) (0.66-1.2 5 mg/dL), and venous pH of 7.26 (7.35-7.45) with partial pressure of carbon dioxide of 34 mmHg (35-45 mmHg), and bicarbonate of 16 mmol/L

(23-27 mmol/L). No prior recent laboratory values were available for comparison. Point-of-care ultrasonography was negative for pneumothorax, pericardial effusion, right heart strain, peritoneal fluid, and abdominal aortic aneurysm. Right extrapleural fluid and collapsible inferior vena cava were noted. A portable supine anterior-posterior chest radiograph (Image 1) again showed right-sided extrapleural fluid.

The patient's condition deteriorated approximately 15 minutes after arrival to the ED. He became unresponsive and shortly thereafter suffered a ventricular fibrillation cardiac arrest. Cardiopulmonary resuscitation (CPR) was initiated, and the patient had return of spontaneous circulation after eight minutes of CPR with two attempts of asynchronous cardioversion and 1 mg intravenous (IV) epinephrine. During chest compression, new right-sided neck swelling developed. Point-of-care ultrasonography showed free fluid between the soft tissue layers. The patient was intubated without complications. Several minutes later, he developed pulseless electrical activity (PEA) and CPR was resumed.

A transesophageal echocardiogram performed during CPR pulse-checks showed coordinated myocardial contraction too weak to produce palpable pulses (pseudo-PEA). A needle aspiration of the right thoracic cavity revealed frank blood, coupled with the collapsible inferior vena cava, pseudo-PEA, and radiograph findings the diagnosis of hemothorax and subsequent cardiac arrest due to internal exsanguination was made. The patient received four units of packed red blood cells and had return of palpable pulses. The decision was made to defer placement of a chest tube as an autologous blood recovery system was not immediately available, the patient oxygenated well, and thoracostomy would have relieved any hydrostatic pressure on the source of hemorrhage, thus hastening the patient's exsanguination. The patient remained hemodynamically stable for 15 minutes, and the decision was made to obtain imaging to plan for either endovascular or surgical hemorrhage control.

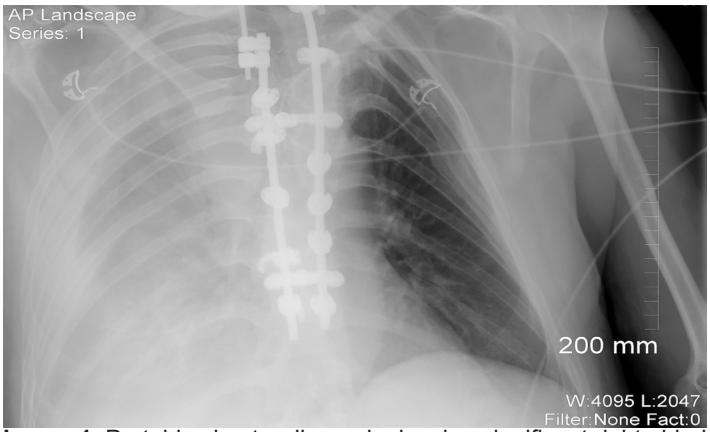

Image 1. Portable chest radiograph showing significant right-sided extrapleural fluid.

Computed tomography of the neck and chest detected a ruptured, right-sided vertebral artery pseudoaneurysm at the origin from the subclavian artery with active extravasation (Image 2). The local hematoma had dissected through the soft tissues of the neck into the right hemithorax. Interventional radiology and vascular surgery were consulted to assist with hemorrhage control, and the patient was transferred to the intensive care unit. Massive transfusion protocol was initiated but the patient became progressively hypotensive despite vasopressor therapy. During preparation for chest tube placement, the patient again went into PEA arrest and CPR was initiated. During resuscitation, a right-sided chest tube was placed emergently and clamped after two liters of blood had been evacuated. After tube thoracostomy, the patient had a change in rhythm from bradycardic PEA to sinus tachycardia with detectable pulses shortly thereafter.

After tube thoracostomy, the patient emergently proceeded to the operating suite. En route, he again suffered a

CPC-EM Capsule

What do we already know about this clinical entity?
Patients with neurofibromatosis are prone to the development of a spontaneous hemothorax due to intrathoracic neurofibromata as well as intrathoracic aneurysms.

What makes this presentation of disease reportable?
We report the first case of an extrathoracic vertebral artery aneurysm rupture that resulted in the development of a tension hemothorax.

What is the major learning point?
Hemorrhage control may be challenging. Thoracostomy predisposes to exsanguination, while massive transfusion protocols can create tension physiology.

How might this improve emergency medicine practice?
Incidentally-found, unruptured aneurysms in patients with neurofibromatosis should undergo urgent evaluation, as rupture is often fatal.

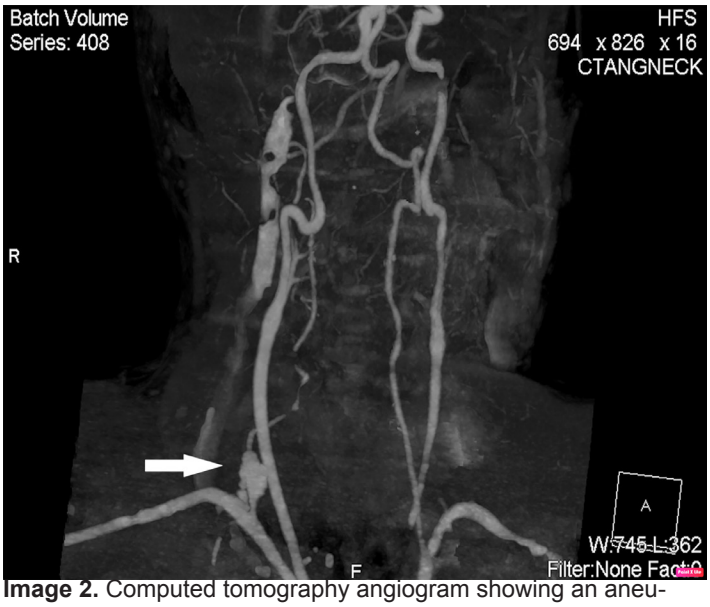

Image 2. Computed tomography angiogram showing an aneurysm (arrow) at the right vertebral artery origin with poor opacification of the remainder of the vessel.

PEA cardiac arrest due to exsanguination after evacuation of the right hemothorax. Despite ongoing resuscitation attempts, the patient expired in the operating room.

DISCUSSION

We report the first case of a tension-type hemothorax due to NF. Given the acuity of the patient, no imaging was obtained to radiographically verify the interim development of a tension hemothorax. The immediate improvement with thoracic decompression by tube thoracostomy supports the diagnosis, as does the preceding massive transfusion protocol which likely added the volume required to convert a hemothorax to a tension hemothorax. It is unlikely that a hypovolemic arrest would have occurred immediately after massive transfusion protocol and would have led to return of spontaneous circulation with CPR and chest tube placement alone.

Hemothoraces have been reported as a rare complication of this disease, typically due to hemorrhage from neurofibromata invasion of thoracic vessels or direct bleeding from the tumors.[4,5] Spontaneous hemothoraces due to aneurysmal rupture have been reported in the literature.[6-9] An overview of the cases with extracranial vertebral artery aneurysms in patients with NF1, their presenting complaints, interventions, and outcome are provided in Table 1.

Primary vascular pathology includes arterial ectasia, stenosis, aneurysm, or fibromuscular dysplasia.[10] Percutaneous coil embolization and stent-graft placement has been successfully employed,[11] although surgical management is often required.[12] The aorta and renal arteries are the most common vessels affected by vascular pathology. A review of the literature showed 20 reports of extracranial vertebral artery aneurysms in patients with NF1.[13,14] Radiculopathy or neck pain appear to be manifestations of unruptured aneurysms, whereas rupture can produce a neck mass with airway compromise or hemothorax.[14]

Due to the high risk of a fatal outcome with rupture, early diagnosis and elective repair prior to rupture is paramount. Vascular abnormalities of any type occur in up to 6.4% of patients[14]; thus, focal pains and paresthesias should be thoroughly evaluated in patients with NF1, with a low threshold for vascular imaging. Once rupture occurs, patients require immediate volume resuscitation and endovascular or surgical intervention.

CONCLUSION

Tension hemothorax due to vertebral artery aneurysm is a rare complication of NF1. Patients with NF1 who present with neck pain, back pain, radiculopathy, cervical hematoma, or hemothorax should be suspected of having vertebral or intercostal artery aneurysm. Unruptured aneurysms should be urgently addressed with endovascular therapy and followed to prevent rebleeding. When there is hemodynamic instability, early surgical intervention should be considered to prevent tension physiology from developing. There may need to be routine screening for patients with NF1 for vascular malformations of the cranial arteries. In our patient, differentiating between chronic and acute neck pain potentially delayed diagnosis of vertebral aneurysmal rupture causing the massive hemothorax.

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

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Table. Demographics, location, presenting symptoms, interventions, and outcomes of patient with neurofibromatosis type 1 and extracranial vertebral artery aneurysms.

Author (year)	Age (years)/ sex	Side	Spine level	Signs and symptoms	Ruptured or unruptured	Treatment	Outcome
Schubiger and Yasargil (1978)	50/Male	Left	C2-C6	Radiculopathy	Unruptured	Surgery	Good
Pentecost et al. (1981)	1/Female	Left	T1	Limited neck movement, arm weakness	Unruptured	Observation	Disabled
Detwiler et al. (1987)	52/Female	Left	C2	Neck mass, neck pain, bruits	Unruptured	Endovascular balloon	Good
Negoro et al. (1990)	43/Female	Left	C1	Neck pain, cervical hematoma	Ruptured	Endovascular balloon	Good
Muhonen et al. (1991)	52/Female	Left	C2	Neck mass, neck pain, arm weakness	Unruptured	Endovascular balloon	Good
Schievink and Piepgras (1991)	43/Female	Left	C7	No symptoms	Unruptured	Observation	Good
Ohkata et al. (1994)	48/Female	Left	C4-C7	Radiculopathy	Unruptured	Surgery	Good
Horsley et al. (1997)	56/Female	Left	C5-C7	Neck pain, arm paresthesias, neck mass	Ruptured	Endovascular coil	Good
Hoffman et al. (1998)	59/Male	Right	C6	No symptoms	Unruptured	Observation	Good
Ushikoshi et al. (1999)	40/Female	Left	C1	Cervical hematoma	Ruptured	Endovascular balloon	Good
Miyazaki et al. (2004)	52/Female	Left	C5-C7	Radiculopathy, hypotension, altered consciousness, hemothorax	Ruptured	Endovascular balloon then surgery	Dead
Arai et al. (2007)	38/Male	Left	Unknown	Chest pain, dizziness, vomiting, hemothorax	Ruptured	None	Dead
Hieda et al. (2007)	36/Female	Left	Ostium of vertebral artery	Back pain, chest pain, dyspnea, hypotension, hemothorax, coma	Ruptured	Endovascular coil	Dead
Hiramatsu et al. (2007)	67/Male	Left	Proximal segment of vertebral artery	Dizziness	Unruptured	Endovascular coil	Good
Pereira et al. (2007)	14/Female	Right	C5-C6	Radiculopathy	Unruptured	Endovascular balloon	Good
Peyre et al. (2007)	18/Female	Right	C5-C6	Radiculopathy	Unruptured	Endovascular coil	Good
Horie et al. (2008)	30/Female	Right	C6-C7	Radiculopathy	Unruptured	Endovascular balloon and coil	Good
Hige et al. (2010)	60/Female	Left	Unknown	Cervical hematoma, respiratory failure	Ruptured	Endovascular coil	Disabled
Morvan et al. (2011)	36/Female	Left	C3-C4	Headache, neck pain, vomiting, subarachnoid hemorrhage	Ruptured	Endovascular coil	Unknown

C, cervical; *T,* thoracic.

Table. Continued.

Author (year)	Age (years)/ sex	Side	Spine level	Signs and symptoms	Ruptured or unruptured	Treatment	Outcome
Hiramatsu et al. (2012)	31/Male	Right	C6	Neck pain, cervical hematoma, radiculopathy	Ruptured	Endovascular coil	Good
Gouaillier-Vulcain et al. (2014)	32/Male	Left	C8	Radiculopathy	Unruptured	Surgery and endovascular stent	Good
Uneda et al. (2016)	35/Female	Right	C3-C4	Radiculopathy	Ruptured	Endovascular coil	Good
Present case	32/Male	Right	C7	Neck pain, back pain, syncope	Ruptured	Surgery	Dead

C, cervical.

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Intracranial Hemorrhage and Pneumocephaly After Cervical Epidural Injection

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Cervical epidural injections are commonly used to treat patients with radicular neck pain. The following is a description of a case of subarachnoid hemorrhage, subdural hemorrhage, and pneumocephaly following cervical epidural injection. [Clin Pract Cases Emerg Med. 2019;3(4):369–371.]

INTRODUCTION

Cervical epidural injections (CEI) are frequently used to treat patients with radicular neck pain. There were 173,925 upper spinal epidural injections performed on Medicare beneficiaries in 2004.[1] Common complications from the procedure are increased neck pain (6.7%) and headache (4.6%).[2] The reported overall complication rate is 16.8%.[2] However, the literature includes many cases of serious complications such as brain and spinal cord infarction.[3] The pathophysiology of these complications is thought to be embolism of particulate steroid from accidental intra-arterial injection. The following describes a case of subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH), and pneumocephaly following CEI.

CASE REPORT

An 88-year-old female presented to the emergency department (ED) with headache following steroid CEI. She had the injection performed prior to arrival as an outpatient. She had received propofol 50 milligrams (mg) intravenous during her procedure. Following the CEI, she became bradycardic. She also reported headache, nausea, and vomiting. On presentation to the ED her headache was severe. She also reported left upper extremity weakness. Past medical history was significant for atrial fibrillation, lung cancer, chronic kidney disease, and hypertension. Her home medication was significant for aspirin 81 mg tablet daily with no other anticoagulant or antiplatelet agents.

On physical examination vital signs were temperature 36.2° Celsius, pulse 67 beats per minute, respiratory rate 18 per minute, pulse oximetry 98% on room air, and blood pressure 188/102 millimeters of mercury. On general exam she appeared distressed. Head was atraumatic. Her eyes had normal conjunctivae, normal extraocular movements, and her pupils were equal, round, and reactive to light. Neck was supple. Cardiovascular exam demonstrated a normal rate, regular rhythm, normal heart sounds and intact distal pulses. Pulmonary exam showed normal effort, with neither wheezes nor rales auscultated; however, she did have scattered rhonchi. Neurological exam showed her to be alert and oriented to person, place, and time. She followed commands appropriately, her gaze was normal, she had no visual field cuts, and no facial palsy.

On motor exam, her left upper extremity showed some effort against gravity but hit the bed before 10 seconds. Motor exam of right upper extremity was normal. Motor exam of both lower extremities exhibited drift but did not hit the bed before five seconds. She otherwise had no sensory deficits, no ataxia, no aphasia, and no dysarthria. Furthermore, there was no extinction to double simultaneous stimuli testing. Her National Institutes of Health Stroke Scale was four. A computed tomography (CT) of her brain showed pneumocephaly, SDH, SAH, and intraventricular hemorrhage (Images 1 and 2).

The patient was admitted to the intensive care unit (ICU). Her blood pressure was managed with labetalol. A CT angiogram (CTA) of the head did not show any aneurysm. She was treated with nimodipine in the ICU. Four days after the procedure repeat brain CT showed resolution of pneumocephaly and intracranial hemorrhage.

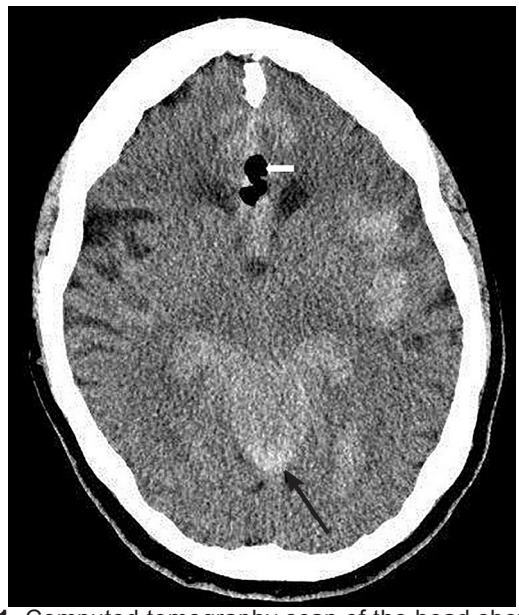

Image 1. Computed tomography scan of the head showing pneumocephalus (white arrow), and subarachnoid hemorrhage (black arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Cervical epidural injections are known to cause serious complications such as brain and spinal cord infarctions.

What makes this presentation of disease reportable?
We describe the complications of subdural hemorrhage, subarachnoid hemorrhage, and pneumocephaly in the same patient after a cervical epidural injection.

What is the major learning point?
Emergency physicians should consider intracranial complications in patients who present with a headache after cervical epidural injection.

How might this improve emergency medicine practice?
Knowing the potential complications after cervical epidural injections will allow for rapid initiation of diagnostic work-up.

Prior to discharge her repeat neurologic exam showed resolution of all deficits.

DISCUSSION

Dural puncture after CEI is a known complication.[2] There have been case reports of intracranial complications after epidural injectons.[4,5,6] However, literature review failed to find reports of SDH, SAH, and pneumocephaly in the same patient after CEI. This is a unique case report describing all three complications in the same patient. Pneumocephalus is thought to occur as a result of inadvertent injection of air into the subdural space.[4] Furthermore, SDH has been described following lumbar puncture.[5] It is postulated to result from direct dural puncture.[5]

Straining actions such as coughing may increase cerebrospinal fluid (CSF) pressure at puncture site leading to CSF leak from the site. The loss of CSF can lead to shift in brain tissue, which can result in shearing of blood vessels. The shearing of blood vessels can lead to their rupture, which can cause SDH. The patient had vomited post procedure. This may have caused CSF leak from an inadvertent dural puncture site triggering the chain of events leading to SDH. In addition, SAH has also been described following lumbar puncture.[6,7] SAH is thought to result from unintentional direct puncture of a spinal vessel causing it to leak.[6,7] The most common etiology of spontaneous SAH is an aneurysm rupture.[8] The patient did have a CTA of the head, which did not show any aneurysms.

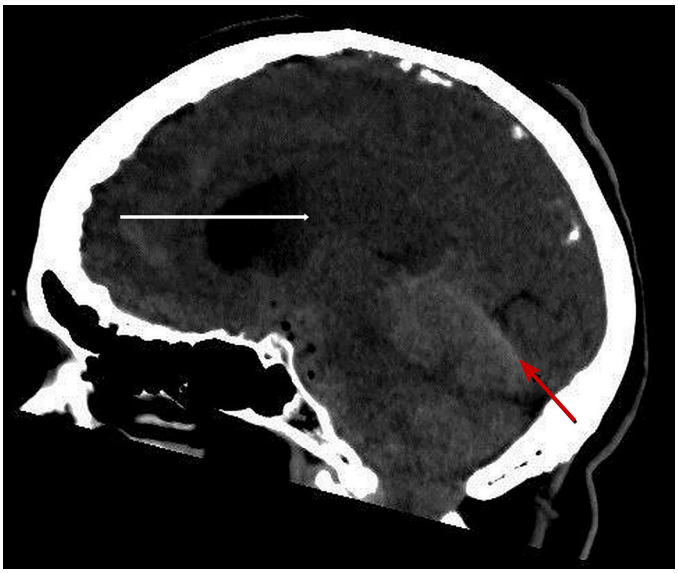

Image 2. Computed tomography of head showing subdural hemorrhage (red arrow) and intraventricular hemorrhage (white arrow).

CONCLUSION

It is important to consider intracranial complications, when a patient presents to ED with headache following a CEI. The differential diagnosis should include SDH, SAH, and pneumocephalus. Evaluation should include head CT. Treatment is supportive in the ED and likely admission to ICU.

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

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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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Occult Vascular Transection Identified by Point-of-care Ultrasound Demonstrating Evidence of Retrograde Flow

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Acute vascular injury can be a cause of significant disability and morbidity. High clinical suspicion and a thorough physical examination are key components to facilitate a timely diagnosis. We present a case of acute vascular injury after isolated penetrating trauma. Physical examination demonstrated a strong distal radial pulse; however, point-of-care ultrasound facilitated an evaluation of the directionality of arterial flow, demonstrating that flow was retrograde via the palmar arch. We subsequently identified a proximal and complete arterial laceration. [Clin Pract Cases Emerg Med. 2019;3(4):372–375.]

INTRODUCTION

Early diagnosis of acute vascular injury is critical to expedite treatment interventions and ultimately improve patient outcomes and prevent disability and amputation. A complete physical exam and a high index of suspicion are pivotal for early identification of an acute vascular injury. When "hard signs" are present clinical consensus mandates immediate surgical intervention.[1] "Hard signs" of vascular injury include absent distal pulses, limb ischemia, pulsatile flow, hemorrhage with shock, or an expanding hematoma. Alternatively, the presence of "soft signs" of vascular injury (such as significant hemorrhage reported at the time of the injury, diminished but palpable pulses, a stable hematoma, a peripheral nerve deficit, or anatomic proximity of the wound to a major artery) should prompt further diagnostic testing.[1]

Point-of-care ultrasound (POCUS) is an accessible tool in the emergency department (ED) that can assist in the diagnosis of an acute arterial injury. Previous studies demonstrate how ultrasound aids in the diagnosis of an acute arterial injury through the identification of the presence or absence of flow, the presence of pseudoaneurysms, abnormalities in waveform and flow velocity, and the quality of the Doppler signal.[2,3,4,5] When using ultrasound for a suspected arterial injury, it is also important to consider directionality, as strong collaterals can generate retrograde flow and mimic a normal exam. We present a case of acute vascular injury after an isolated, small penetrating trauma to the forearm. The patient presented with "soft signs" and normal findings. Using POCUS and accounting for directionality, we were able to demonstrate an occult arterial injury, which was masked on physical examination by strong collateral flow.

CASE REPORT

A 30-year-old previously healthy male presented to the ED with a laceration to the left forearm. The injury occurred just prior to arrival when the patient was carving a Halloween pumpkin with a large steak knife, which slipped and punctured the volar aspect of his left forearm, halfway between his elbow and his wrist. His wife noted projectile bleeding immediately after the injury. Emergency medical services arrived and applied a tourniquet proximal to the injury of the affected limb. Upon arrival to the ED, the tourniquet was removed. On physical examination "oozing blood flow consistent with venous injury" and no pulsatile flow was present. He reported 5/10 pain over the area of the wound. He denied numbness, paresthesias, decreased range of motion, or change in skin color.

The patient had no significant past medical history and was not on any medications. The vital signs were as follows: temperature of 98.9° Fahrenheit; pulse of 80 beats per minute; blood pressure of 140/79 millimeters of mercury. Physical examination was significant for a 1.5 centimeter (cm) full thickness laceration to the volar aspect of the left forearm, with slow pooling of blood in the wound. Radial pulses were 2+ distal to the laceration. There was no pulsatile bleeding, and bleeding was well controlled with pressure to the wound. Sensation was grossly intact, capillary refill was less than two seconds, and

motor strength – including grip, flexion and extension of wrist and all digits, and abduction and adduction of digits – were intact. The skin was normal without mottling or pallor.

The patient's blood work in the ED was notable for a mild anemia hemoglobin of 12.9 grams per deciliter (g/dL) (reference range 13.7 - 17.1 g/dL) and hematocrit of 41% (40.5-50.0%) and mild thrombocytopenia of 148 thousand cells per cubic millimeter (k/mm^3) (reference range 150 – 460 k/mm^3). We performed a POCUS with the 10-megahertz linear array transducer to evaluate for potential arterial injury. Using color Doppler, the POCUS demonstrated pulsatile flow in the radial artery. However, when the directionality of the flow was evaluated, POCUS confirmed the suspicion for a vascular injury by demonstrating retrograde flow in the radial artery at the level of the wrist crease (Image 1 and Video).

Vascular surgery was consulted and requested computed tomography angiography (CTA) of the extremity for operative planning. The CTA confirmed the ultrasound findings, demonstrating a focal cutoff of the radial artery at the level of the laceration with a patent ulnar artery providing retrograde perfusion to the distal radial artery (Image 2). The patient was taken to the operating room immediately from the ED for intra-operative exploration, which revealed a complete radial artery transection. After repair of his radial artery, the patient was hospitalized, placed on a heparin drip for 24 hours, and given clopidogrel and aspirin. On postoperative day two a duplex ultrasound demonstrated patency of the radial artery and the patient was discharged.

CPC-EM Capsule

What do we already know about this clinical entity?
Acute vascular injury is a cause of disability. A detailed exam is key to diagnosis, however diagnosis remains difficult when soft signs of vascular injury are present.

What makes this presentation of disease reportable?
A physical exam demonstrating a strong radial pulse was misleading. Point-of-care ultrasound (POCUS) identified retrograde flow, which drew concern for arterial injury.

What is the major learning point?
When history and the physical exam conflict consider assessing the direction of flow with POCUS.

How might this improve emergency medicine practice?
It demonstrates that POCUS can assist and expedite the diagnosis of acute arterial injury and improve patient care.

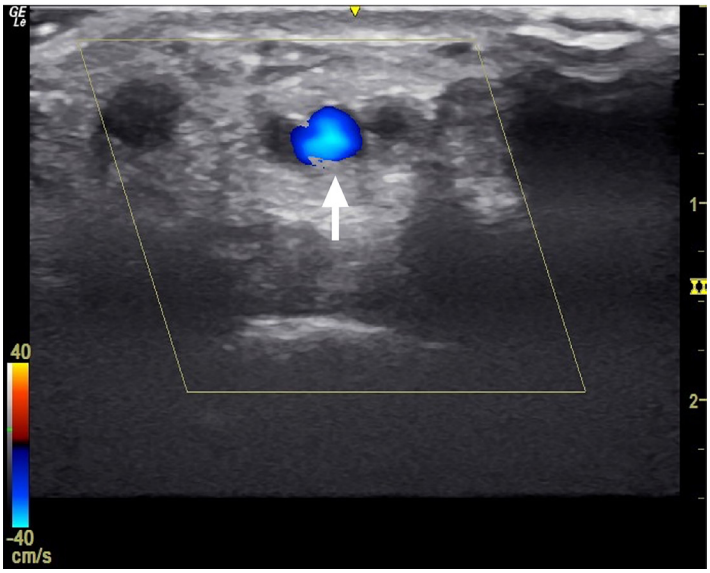

Image 1. The image exhibits the ultrasound of the radial artery distal to the injury in transverse view. The white arrow demonstrates the radial artery, with color Doppler over the vessel. The blue color within the radial artery signifies retrograde flow, with blood flow in the direction away from the ultrasound probe.

DISCUSSION

While this patient presented with a clinical history of pulsatile bleeding concerning for an arterial injury, upon arrival to our ED there were no "hard signs" of vascular injury. Historically, a clinical suspicion for vascular injury was determined by the presence of "hard" or "soft" signs of vascular injury. Previous literature has mandated surgical exploration of a wound for "hard signs."[1] However, the recommendations for "soft signs" are more nebulous.[1] In the absence of "hard signs," many physicians opt for a CTA to diagnose arterial injuries; however, CT carries potential risks in the form of radiation and allergic reactions. An alternative to this is ultrasound.

Rad et al.[3] compared CTA to ultrasound with Doppler waveforms examined at multiple locations along the affected limb of patients with concern for an arterial injury. If there was a waveform abnormality noted at any distal locations, the expert radiologist performing the study was then prompted to more closely evaluate the entire artery. This technique resulted in a sensitivity of 94.8% and a specificity of 91.6%. One false negative was attributed to a thick dressing that wasn't removed. Importantly, a second arterial injury was missed due to strong collateral blood

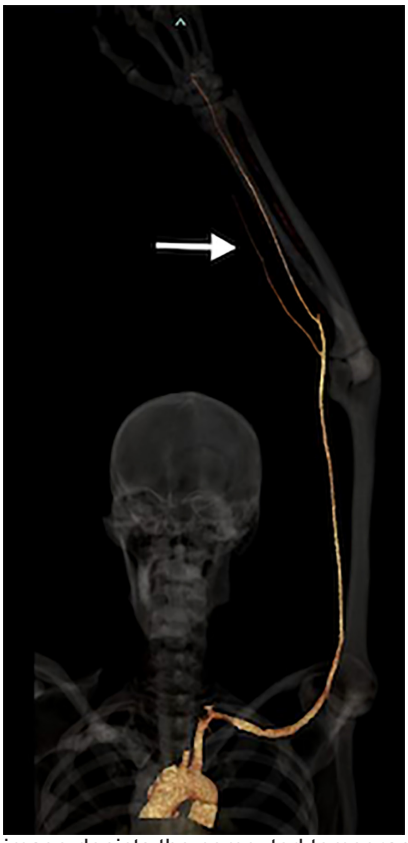

Image 2. The image depicts the computed tomography angiography reconstruction with bone shadow. The white arrow demonstrates the vascular cutoff of the radial artery in the area of the soft-tissue laceration.

flow that masked the injury, consistent with the clinical presentation of our patient.

Wani et al.[4] compared CTA to ultrasonography in patients with "soft signs" concerning for vascular injury. Patients were evaluated with ultrasound performed by a radiologist, and if positive, the patients were surgically explored. However, if the ultrasound was negative, patients were evaluated via CTA. A total of 150 patients underwent ultrasound, and 110 demonstrated positive findings and were taken for vascular surgery. Of the 40 patients who had no ultrasound findings, seven were found to have CTA findings concerning for vascular injury. The authors reported a sensitivity of 94% and a specificity of 82.5%.

Only one study to date has evaluated the use of POCUS to identify acute traumatic vascular injuries. Monoforano et al.[6] examined the accuracy of a focused Doppler ultrasound protocol in the rapid assessment of arterial injuries after penetrating trauma. Two board-certified physicians with specializations in ultrasound performed a two-point Doppler assessment of the posterior tibial and dorsal pedis arteries with POCUS, specifically assessing for the presence of flow and characteristics of the Doppler waveform. A pathologic waveform was defined as absent flow or a biphasic or a monophasic waveform in one of the examined arteries. Of the 149 limbs included in the study, 134 were correctly identified as having no acute injury while 15 limbs were correctly identified as having an acute arterial injury, first by standardized full color Doppler and then by CTA. The study found that through utilization of these methods and the implementation of a two-point Doppler protocol, a sensitivity of 100% and specificity of 100% with a positive predictive value of 100% could be achieved.

Not all studies of ultrasound have demonstrated such accurate test characteristics. In one such study the specificity was quite high at 99%, but the sensitivity was low at 50%.[7] The ultrasound studies were performed by either vascular technicians or a vascular surgery fellow specializing in vascular ultrasound. The authors noted, however, that both of the missed arterial injuries were small pseudoaneurysms. Furthermore, they attributed this low sensitivity to a low prevalence, as in this study only four injuries were found on angiography, and two of them were missed on ultrasound for a sensitivity of 50%.

A recent meta-analysis by DeSouza et al.,[8] however, appears to support ultrasound as a viable and accurate option. The authors examined a large database of studies that included ED patients with penetrating extremity injuries. Among those included in the meta-analysis were studies that used ultrasound, performed by both vascular technicians and radiologists, to investigate occult vascular injuries. The composite data showed a positive ultrasound for injury to have a positive likelihood ratio (LR) of 35.4 (95% confidence interval (CI), 8.3-151) and a negative ultrasound to have a LR of 0.24 (95% CI, 0.08-0.72). Collectively, this data suggests that ultrasound is an appropriate first modality with which to investigate occult arterial injuries.

CONCLUSION

Taken together, the current literature suggests that ultrasound is specific for vascular injuries, but may not be sensitive enough to rule out a vascular injury. If there is suspicion for vascular injury, despite a negative ultrasound, a CTA may be warranted. Careful ultrasonography, however, either by an emergency physician or a vascular technician, could potentially avoid unnecessary CTA in patients for whom the ultrasound shows strong evidence of vascular injury. Our patient had strong collateral flow from his ulnar artery, which masked the injury on physical examination alone. However, we were able to use the direction of flow on ultrasound to determine that this was in fact retrograde flow, indicating an injury to the proximal radial artery. Ultimately, determining the directionality of flow may improve the sensitivity of ultrasound, and, as in our clinical case, immediately inform the physicians that there is strong evidence of a vascular injury. This could allow emergency physicians and vascular surgeons to bypass CTA when appropriate to expedite clinical care through cost-effective use of diagnostic technologies.

Video. The video clip illustrates color Doppler of the radial artery using point-of-care ultrasound in the transverse view. The scale in the lower left corner indicates that blood flowing toward the probe is represented with red, while blood flow away from the probe is represented with blue. The white arrow indicates the radial artery. The probe was angled in a proximal orientation towards the patient's head. The blue color, within the radial artery, indicates blood flowing away from the transducer in a retrograde direction.

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

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Iatrogenic Aortic Dissection Presenting With Leg Pain Diagnosed With Point-of-care Ultrasound

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Iatrogenic aortic dissection (IAD) status-post-cardiac catheterization is a rare complication often isolated to the proximal aorta. This is a case of IAD isolated to the distal aorta in a 41-year-old female who presented to the emergency department with right leg pain after undergoing three cardiac catheterizations. The diagnosis of IAD was made upon discovery of an intimal flap in the distal aorta and femoral artery while performing a point-of-care ultrasound to evaluate for deep vein thrombosis. [Clin Pract Cases Emerg Med. 2019;3(4):376–379.]

INTRODUCTION

Point-of-care ultrasound (POCUS) is a useful diagnostic tool in the emergency department (ED). There is abundant literature supporting the use of ultrasound for the evaluation of undifferentiated patients in the ED.[1,2] This report describes the case of a patient who underwent POCUS to evaluate for a possible deep vein thrombosis (DVT) and was ultimately diagnosed with a distal aortic dissection extending into the right femoral artery. The etiology of this dissection is thought to be iatrogenic secondary to recent cardiac catheterization. Although aortic dissection is a heavily considered diagnosis in the emergency medicine setting, it is a rare complication of cardiac catheterization and subsequently may present with atypical symptoms.

CASE REPORT

A 41-year-old female with extensive medical history including hypertension, lupus nephritis, anti-phospholipid antibody syndrome, coronary artery disease, and previously treated Libman-Sacks endocarditis presented to the ED with persistent lightheadedness for one week and two days of recurrent nausea and vomiting with decreased oral intake. She had a pertinent surgical history of coronary artery bypass graft and aortic valve replacement secondary to the endocarditis. She was anticoagulated on warfarin and required hemodialysis. The patient also reported two days of right calf pain that occurred only when ambulating. She did not complain of chest pain, back pain, or abdominal pain.

The patient's initial vital signs included a blood pressure of 171/91 millimeters of mercury, heart rate of 92 beats per minute, respiratory rate of 18 breaths per minute, and oral temperature of 37.1 degrees Celsius. Her oxygen saturation was 99% on room air. On initial evaluation in the ED, the patient appeared in no distress and was alert and oriented to person, place, and time. She answered questions appropriately, and her neurologic examination showed no focal weakness or sensory deficits. Lungs were clear and cardiac exam was noted as regular rate and rhythm without murmur. The patient's abdomen was soft, non-tender, and non-distended. Her lower extremities were warm and well perfused with normal range of motion and no swelling or calf tenderness. Her peripheral pulses were intact and symmetric bilaterally.

Based on her history and physical examination, the treating physicians were most concerned for an acute viral process or foodborne illness. Nonetheless, given her complaint of right calf pain in the context of a chronic pro-coagulant state, they decided to evaluate for a DVT in the right lower extremity. The patient underwent a POCUS two-point

compression examination of the right leg, which showed normal compression of the right femoral and popliteal venous systems. However, an abnormal intraluminal echogenic signal was seen in the right femoral artery, which had the appearance of an intimal flap.

Color Doppler was used to confirm differential flow on either side of the flap (Image 1). The ultrasonographers proceeded to interrogate the abdominal aorta, and a dissection flap was noted in the transverse view (Image 2). A computed tomography (CT) angiogram of the chest, abdomen, and pelvis with run-off to the lower extremities was then performed, which showed an intimal flap starting in the distal abdominal aorta and extending into the right common iliac, external iliac, and superficial femoral arteries (Image 3).

A subsequent review of the patient's medical chart showed that she had been admitted to our institution one month prior for acute coronary syndrome and had been taken to the cardiac catheterization suite three times during that hospitalization. The hospital record noted that she was canalized in her femoral region three times, twice via her left femoral artery and once via her right femoral artery. The patient was assessed by the vascular surgery team in the ED. Their impression was this dissection was iatrogenic given her history of recent catheterization, and they recommended strict blood pressure control and admission. Given her extensive and complicated cardiovascular history she was ultimately admitted to the cardiac intensive care unit. Her blood pressure medications were adjusted, and she was discharged home three days later.

CPC-EM Capsule

What do we already know about this clinical entity?
Post-cardiac catheterization aortic dissection (AD) is a rare but known complication.

What makes this presentation of disease reportable?
To our knowledge, this is the first report of a post-cardiac catheterization aortic dissection isolated to the abdominal aorta and distal arteries.

What is the major learning point?
Iatrogenic AD is a rare but important complication of cardiac catheterization. Clinical astuteness during ultrasound evaluation is paramount.

How might this improve emergency medicine practice?
Consideration of iatrogenic AD in post-catheterization patients presenting with vague symptoms can lead to earlier diagnosis and treatment.

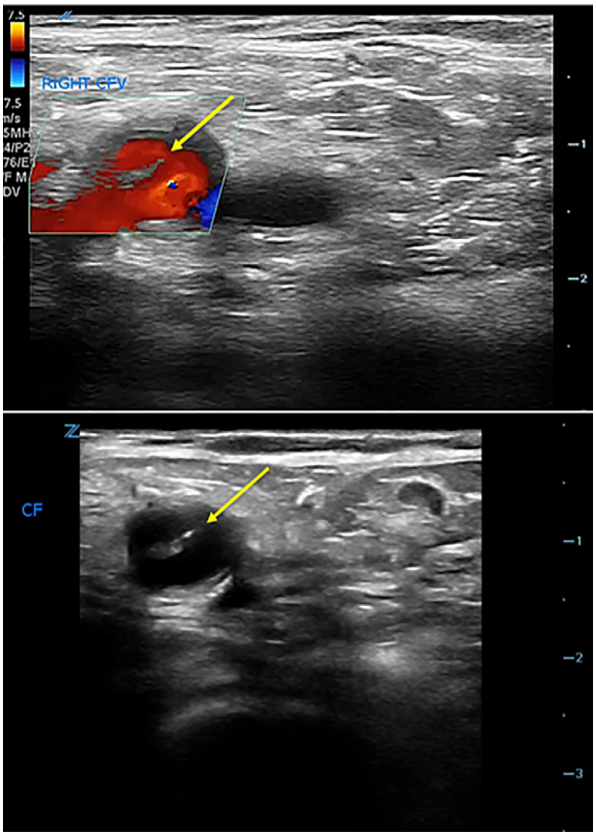

Image 1. Grayscale and color ultrasound demonstrating intimal flap (arrow) in the right common femoral artery.

DISCUSSION

The advantage of POCUS is that image acquisition and interpretation are performed in real time by clinicians who can quickly integrate unexpected findings into the overall clinical picture. In this case, we were able to reorient from performing a DVT study to fully investigating what appeared to be a femoral artery dissection. By tracing the intimal flap proximally to the abdominal aorta, we rapidly diagnosed the patient's aortic dissection and promptly initiated tight blood pressure control. A CT angiogram was then performed to define the extent of the dissection. Without POCUS, this diagnosis may have been delayed or missed. This case highlights the importance of remaining astute during the clinical evaluation of a complicated patient.

When aortic dissection is diagnosed using POCUS, the most common scenario is a proximal aortic dissection

associated with aneurysmal dilation of the aortic root.[3] Our case is unusual among reports of aortic dissection identified on ultrasound because the diagnosis was made by finding an intimal flap in the distal aorta and femoral artery. Aortic dissection after coronary angiography is a known complication, but it is rare. In a review by the Registry of Aortic Iatrogenic Dissection of 108,083 catheterization procedures that occurred from 2000-2014, the incidence of aortic dissection was 0.062%.[5] More recent, smaller case series report similar incidences.[6,7] Of note, these were all ascending dissections. To our knowledge, there are no published reports of iatrogenic aortic dissections (IAD) after coronary angiography isolated to the abdominal aorta and distal vasculature.

Etiology of IAD is suspected to be related to wire or catheter trauma, and there is suggestion that vessels with greater calcification are more prone to IAD.[10] In general, there are no guidelines for managing IAD. IAD has been successfully managed surgically with stenting, as well as conservatively.[7] In the available cases series, none of the IAD were of the abdominal aorta. In general, Stanford B or abdominal aortic dissections are managed either medically or with endovascular stenting.[8,9] Factors influencing more aggressive treatment include hemodynamic instability, false lumen expansion, and failure of medical management. Our patient was successfully treated with medical management alone.

Diagnosis of aortic dissection can be made by CT, magnetic resonance imaging, and ultrasound. Studies done to ascertain the sensitivity and specificity of ultrasound in the diagnosis of aortic dissection show wide variability from a sensitivity of 52-80% and a specificity from 0-100%.[13,15] The presence of an intimal flap, however, was found to be 100% specific and 67% sensitive for aortic dissection.[14,15] Although ultrasound should never be the definitive test for aortic dissection, as it lacks sufficient sensitivity, the presence of an intimal flap is highly specific.[4] Moreover, using POCUS to evaluate suspected aortic dissection has been shown to greatly reduce mean time to diagnosis, which can be life- and limb-saving with such a time-sensitive disease process.[3] Our patient had claudication symptoms, which could have progressed to frank leg ischemia if the dissection had worsened.

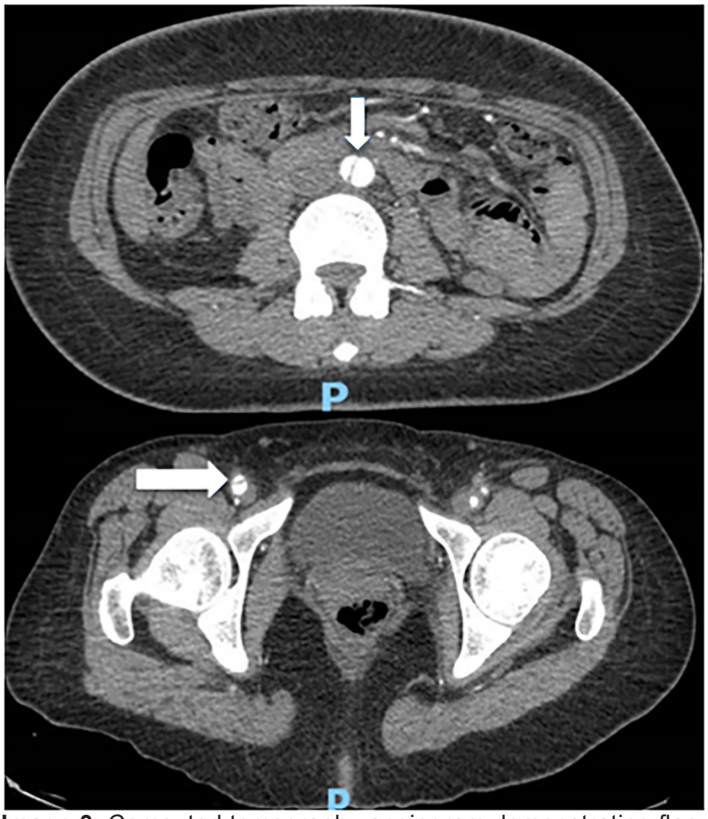

Image 3. Computed tomography angiogram demonstrating flap in mid-aorta (top arrow) and flap in right common femoral artery (bottom arrow).

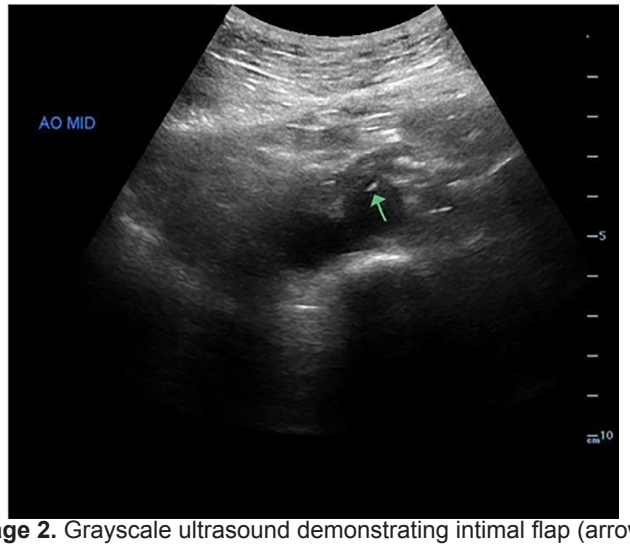

Image 2. Grayscale ultrasound demonstrating intimal flap (arrow) in mid-aorta.

CONCLUSION

Iatrogenic aortic dissection after coronary angiography is a rare diagnosis, and this case is the first report to our knowledge of a dissection isolated to the distal aorta and its branches. Remaining alert to the possible complication of post-catheterization aortic dissection is important for the emergency physician. This case also reinforces the importance of promoting and maintaining strong bedside sonographic skills within emergency medicine. Our patient was found to have fully compressible veins, but the recognition of a dissection flap in the femoral artery was a critical finding that could have been missed without a well-grounded and methodical approach.

Video. Grayscale ultrasound demonstrating intimal flap in the right common femoral artery.

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

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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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Varicella Zoster Virus Encephalitis

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Varicella zoster virus in the adult patient most commonly presents as shingles. Shingles is a painful vesicular eruption localized to a specific dermatome of the body. One of the potential complications of this infection is involvement of the central nervous system causing encephalitis. An increased risk of this complication is associated with the immunocompromised patient. In this case report, we review the history and physical exam findings that should raise clinical suspicion for varicella zoster encephalitis, as well as the epidemiology, risk factors, treatment, and prognosis of this type of infection. [Clin Pract Cases Emerg Med. 2019;3(4):380–382.]

INTRODUCTION

We present a case of a patient with varicella zoster virus (VZV) encephalitis caused by a combination of the patient having active virus reactivation in the form of shingles on the right leg, in addition to being immunocompromised due to a kidney transplant. According to the World Health Organization, encephalitis occurs in one out of every 33,000–50,000 cases of VZV. It also carries a less favorable prognosis compared to the other extracutaneous complications of VZV. This case report shows how prompt recognition and treatment of this type of infection can decrease mortality and progression of the infection in the high-risk, immunocompromised patient.

CASE REPORT

A 67-year-old man with a medical history of kidney transplant, chronic renal dysfunction, prior cytomegalovirus infection causing retinal damage and vision loss and prescribed valacyclovir presented to the emergency department (ED) with a complaint of hallucinations and weakness. This was the patient's fifth healthcare encounter in three weeks. The first visit was to the ED for heel pain, and he was discharged home after an unremarkable right foot radiograph. The patient then returned to the ED for his second visit with a painful vesicular rash along the second sacral dermatome of his right leg and was prescribed valacyclovir 1 gram orally three times a day for seven days for shingles. Vaccination status was unknown at the time of diagnosis.

On the third ED visit two days later, the patient presented with vomiting after being seen by his primary care doctor that morning. The patient was able to tolerate two doses of valacyclovir; and while being seen by his primary care doctor, his valacyclovir dosing was adjusted to account for his renal disease. The patient also was experiencing hallucinations but was discharged home with the explanation that his symptoms could have been due to dehydration after a "negative workup." On his fourth visit to the ED seven days later, the patient stated that he would "close his eyes and see bands playing and rolling plains of green grass." He stated that these images were very vivid but would go away when he opened his eyes. The patient also had difficulty ambulating and generalized weakness. A family member reported that he also had difficulty with finding words.

Vital signs during this fourth ED visit included the following: temperature 99.4° Fahrenheit; pulse 92 beats per minute; respiratory rate 20 respirations per minute; room air pulse oximetry 98%, and a blood pressure of 196/91 millimeters of mercury. Physical examination revealed crusted lesions following the second sacral dermatome on the posterior right leg extending from the sacral region to the lower calf. A neurological exam revealed generalized weakness and difficulty with ambulation without any focal deficits.

Laboratory testing, including complete blood count, metabolic panel and urinalysis were unremarkable except for serum blood urea nitrogen, creatinine and glomerular filtration rate, which were 23.1 milligrams per deciliter (mg/

dL) (normal range 6.0-20.0 mg/dL), 3.03 mg/dL (normal range 0.67-1.17 mg/dL) and 22 milliliters per minute (mL/min) (normal is >60 mL/min), respectively. Chest radiograph was unremarkable and brain computed tomography (CT) demonstrated only chronic mild to moderate degenerative changes. Based on the recent diagnosis of shingles, history of immunocompromise and hallucinations with weakness, lumbar puncture was performed. Results included elevated protein with lymphocyte predominance consistent with viral infection. Cerebral spinal fluid (CSF) culture was ordered, and the patient was administered one gram of acyclovir intravenously and admitted to the hospital.

On hospital day one CSF culture demonstrated VZV via polymerase chain reaction (PCR). The patient also underwent brain magnetic resonance imaging (MRI) on hospital day two, which showed moderate chronic microvascular ischemia and abnormal appearance of the distal left vertebral artery. Infectious disease, neurology and hospital medicine teams all evaluated the patient and agreed with the diagnosis of VZV encephalitis in the setting of recent shingles, CSF findings, and patient presentation. The patient was administered a two-week course of acyclovir with improvement of his hallucinations and presenting symptoms prior to discharge on hospital day four.

CPC-EM Capsule

What do we already know about this clinical entity?
Varicella Zoster Virus (VZV) affects approximately 30% of people in the United States. Encephalitis carries a less favorable prognosis compared to other extra-cutaneous complications of VZV.

What makes this presentation of disease reportable?
Emergency physicians are not suspicious of VZV encephalitis as a possible cause in elderly patients with altered mental status. This presentation raises awareness of the importance of considering VZV encephalitis in these patients.

What is the major learning point?
Keep a high index of suspicion for VZV encephalitis in the elderly population and those that are immunocompromised, especially with a recent history of rash.

How might this improve emergency medicine practice?
Presenting this case and raising awareness amongst emergency physicians can help prevent a delay in diagnosis and improve outcomes.

DISCUSSION

VZV affects approximately 30% of people in the United States during their lifetime.[1] Primary infection causes chickenpox or varicella. The virus is never fully eradicated from the body, however, as it travels and lies dormant in the cranial, dorsal root, or autonomic ganglion.[2] Secondary VZV skin eruption demonstrates a characteristic unilateral, vesicular, and painful eruption that follows a distinct dermatomal distribution. The typical pain pattern of the virus is caused by increased excitability of central nociceptors in the spinal cord causing inflammation and disruption to the nerve cells, making them more sensitive to painful stimuli.[3]

VZV can also cause many different central nervous system (CNS) pathologies if the infection invades the spinal cord or cerebral arteries, including cerebellar ataxia, arteritis, myelitis, meningitis, and encephalitis. CNS infection can occur with primary or secondary reactivation of the virus. Two main risk factors increase the risk for VZV, including age greater than 50 years old and immunocompromise due to reduced T cell-mediated immunity.[4] Transplant patients are at increased risk compared to the general public with an incidence rate of 17:1000.[5] The patient in this case study had both of these main risk factors.

VZV encephalitis causes a headache, fever, vomiting, and altered level of consciousness or even seizures. The patient in this case presented with vomiting, mental status changes, and hallucinations. These symptoms can be seen more commonly as side effects due to inappropriately renal-dosed valacyclovir. VZV encephalitis mortality rate for immunocompetent patients is approximately 15% and almost 100% in an immunosuppressed patient, especially if both the liver and lung are infected.[1,6] VZV encephalitis CSF analysis typically demonstrates lymphocytic pleocytosis and elevation of protein both of which occurred in this case. Positive PCR testing in CSF confirms VZV.[7] CSF anti-VZV antibodies can be performed but cannot be used alone as means for diagnosis of VZV-related neurological conditions.[2,8]

Common findings on brain CT specific for VZV encephalitis are a hypodensity in the temporal lobes with possible frontal lobe involvement. The basal ganglia are commonly spared. For MRI, the common findings for VZV encephalitis are edematous changes with hyperdensity in the temporal lobes and inferior frontal lobes with the basal ganglia being spared.[9,10]

Treatment of VZV encephalitis is intravenous (IV) acyclovir for seven days in the immunocompetent patient

and 10-14 days in the immunosuppressed patient. The patient, in this case, received IV acyclovir for four days and was discharged on two-week course of oral acyclovir. Steroids can be used to reduce inflammation if there is concern for vasculopathy.[11] The valacyclovir initially prescribed was discontinued by the patient after only two doses due to vomiting. Had the patient taken the full course of medication, neurologic side effects due to renal impairment could have been a cause for his presentation. The incidence of positive PCR CSF in immunosuppressed patients with shingles alone is unknown.

CONCLUSION

The presence of vomiting, hallucinations, and mental status changes should alert the emergency physician to consider VZV encephalitis, especially in the immunocompromised patient. Prompt lumbar puncture and early administration of IV acyclovir are critical. Also, antiviral medications may cause adverse neurologic effects, especially in older patients with renal disease.

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

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Kawasaki Disease Presenting as Acute Acalculous Cholecystitis

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Acute acalculous cholecystitis (AAC) is a rare, potentially serious disease that has been associated with Kawasaki disease (KD) in children. Studies suggest that patients presenting with severe abdominal symptoms secondary to KD have increased resistance to intravenous immunoglobulin (IVIG), and a higher rate of coronary artery aneurysms. We describe an eight-year-old boy who presented to the emergency department with severe abdominal pain and was diagnosed with AAC and KD. He was treated with IVIG and high-dose aspirin, achieving good response with complete symptom resolution. He had no coronary artery aneurysms or further complications and was discharged after three days. [Clin Pract Cases Emerg Med. 2019;3(4):383–386.]

INTRODUCTION

Kawasaki disease (KD) is a pediatric vasculitis that typically presents with a set of non-specific symptoms, such as abdominal pain, vomiting, diarrhea, rash, cough, rhinorrhea, and irritability, which can obscure a correct diagnosis.[1] Rarely, KD may present with an acute surgical abdomen caused by conditions such as acute acalculous cholecystitis (AAC), gallbladder hydrops, appendicular vasculitis, or hemorrhagic duodenitis.[2] Patients who present with gastrointestinal symptoms as a manifestation of KD often have a delay in the diagnosis of KD and initiation of therapeutic interventions, and can be subjected to unnecessary surgical interventions.[3] Further, KD is associated with coronary artery involvement in approximately 5% of affected patients.[4]

AAC is an inflammatory disease of the gallbladder that is associated with various systemic illness including KD.[5] Although AAC is usually benign and self-limited, in rare instances it may require surgical intervention to avoid life-threatening complications such as sepsis and death.[5,6] AAC has been reported to be a marker of more severe disease and is associated with resistance to intravenous immunoglobulin (IVIG, part of the standard treatment regimen for KD) and increased risk for coronary artery lesions.[3,4] Thus, prompt recognition of AAC is imperative. When AAC is diagnosed in the emergency department (ED), emergency physicians should consider the possibility of an underlying systemic illness, especially in children, to avoid delay in diagnosis and treatment and to prevent coronary lesions.[3]

To our knowledge, no instances of KD have been reported in the emergency medicine literature. Here, we present a case report describing a pediatric patient with AAC who responded well to IVIG and had no associated coronary lesions.

CASE REPORT

An eight-year-old boy was brought to the ED with right-sided abdominal pain, diarrhea, vomiting, and fever for the prior week. He had been evaluated in the ED on the first day of his illness and diagnosed with a viral infection. On day two of the illness the child had been evaluated by his pediatrician, who suspected a urinary tract infection on the basis of urinalysis results showing pyuria. On arrival at the ED, the patient had a blood pressure of 100/60 millimeters of mercury, a pulse rate of 96 beats per minute, a respiratory rate of 20 breaths per minute with oxygen saturation of 98% on room air, and an oral temperature of 98.2 degrees Fahrenheit (36.7 degrees Celsius). Physical examination revealed injected conjunctiva, desquamation of the lips, and tenderness of the right upper and lower abdominal quadrants, with rebound and guarding.

Laboratory studies showed a white blood cell count of 13,200 x 10^3 per microliter (mcL) (4.5-14.5 x10^3/mcL), an erythrocyte sedimentation rate 28 millimeters per hour (1-13 mm/hr), a C-reactive protein level 6.2 milligrams per deciliter (mg/dL) (3-5 mg/dL), a total bilirubin level of 3.6 mg/dL (0.2-1.2mg/dL) with direct bilirubin of 2.8 mg/dL (<0.03 mg/dL), an alkaline phosphatase level of 428 units per liter (U/L)

(35-104 U/L), and gamma-glutamyl transferase levels of 102 U/L (9-48 U/L). Formal abdominal ultrasonography revealed a distended gallbladder with scant pericholecystic fluid and sludge (Image 1). Computed tomography showed a distended gallbladder (Image 2).

A diagnosis of KD with AAC was made, and the patient was started on oral high-dose aspirin. He was transferred to a tertiary care center where he also received IVIG. All symptoms improved with treatment. His echocardiogram did not show any evidence of coronary artery aneurysms. The patient was discharged home with no further complications after hospital day three.

DISCUSSION

KD is a common cause of pediatric vasculitis.[7] Nonetheless, distinguishing KD from other febrile illness remains one of the biggest challenges that emergency physicians face.[8] In the United States KD has surpassed rheumatic fever as the leading cause of acquired heart disease in children, affecting 19 per 100,000 children under the age of five.[9,10]

The clinical criteria for the diagnosis of KD include at least five days of fever plus at least four of five principal clinical features: polymorphous rash; oral changes; bilateral conjunctival injection; cervical lymphadenopathy; and extremity changes.[1] If four or five of the clinical criteria are met, physicians may proceed to treatment; however, if only two or three of the five principal clinical criteria are met but clinical suspicion remains high, supplemental laboratory findings may aid diagnosis. Typically, complete blood count, hepatic panel, C-reactive protein level, erythrocyte sedimentation rate, and urinalysis are sufficient to supplement the two or three principal clinical criteria to enable the diagnosis. Such cases are much less common and are often referred to as "incomplete" or "atypical" KD.[1,7]

Cardiac complications such as coronary artery aneurysm are the main concern with KD; nonetheless, other organ systems can be affected.[11,12] For example, gastrointestinal (GI) symptoms with hepatobiliary abnormalities are the initial presentation in some patients.[5] Atypical signs and symptoms that should prompt clinical suspicion for KD are hepatic dysfunction, gallbladder hydrops, jaundice, cholestasis, paralytic ileus, and AAC.[2,7] Yi et al. noted that gallbladder distention alone in patients with KD is associated with coronary artery complications.[6] A 2018 multicenter study conducted in Italy showed that GI symptoms as a manifestation of KD indicate greater risk for severe coronary lesions.[3] Other factors included delayed treatment, low albumin level, and age younger than six months.[3]

Although AAC is uncommon in pediatric patients, recognizing it early is vital given the high incidence of

CPC-EM Capsule

What do we already know about this clinical entity?
Patients presenting with severe abdominal pain secondary to Kawasaki disease (KD) have increased resistance to intravenous immunoglobulin and a higher rate of coronary aneurysms.

What makes this presentation of disease reportable?
The patient presented with acute acalculous cholecystitis (AAC)secondary to KD and had no coronary artery aneurysms; he responded well to intravenous immunoglobulin.

What is the major learning point?
AAC in children is often associated with systemic illness, such as KD. Medical treatment rather than immediate surgical intervention is preferred.

How might this improve emergency medicine practice?
When AAC is diagnosed in the emergency department an underlying systemic illness such as KD should be sought to avoid delay in diagnosis and treatment.

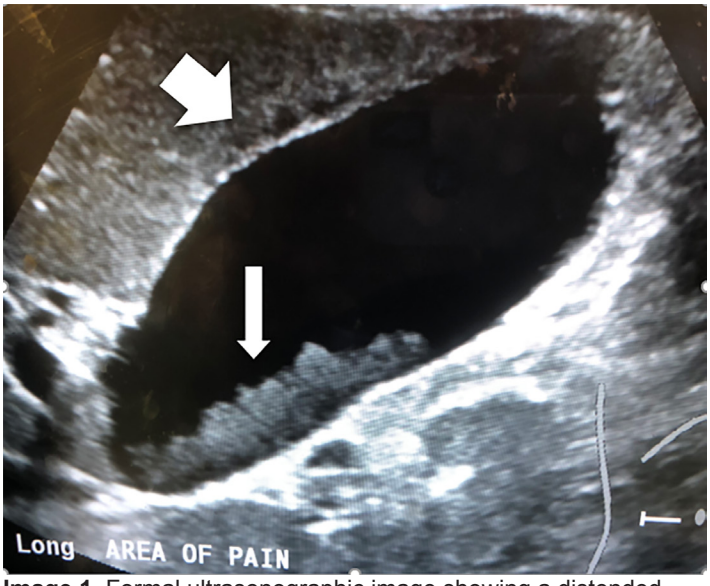

Image 1. Formal ultrasonographic image showing a distended gallbladder in long axis with scant pericholecystic fluid (thick arrow) and sludge (thin arrow).

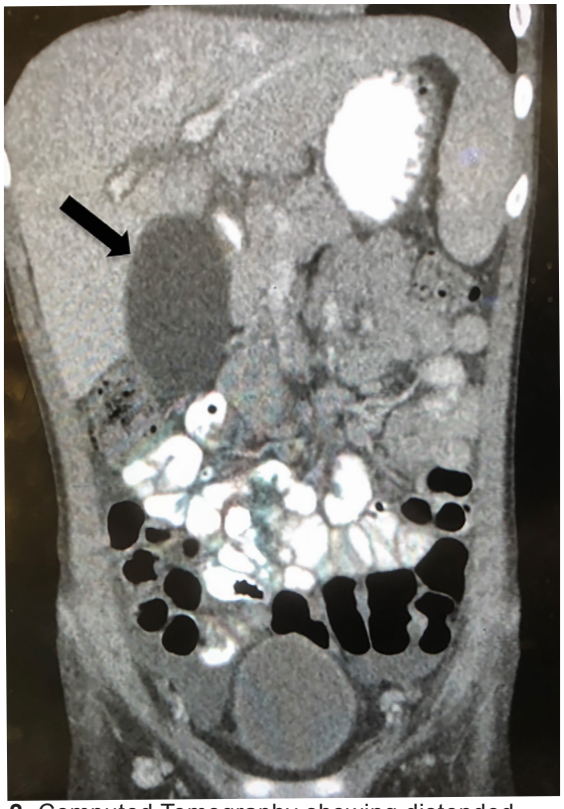

Image 2. Computed Tomography showing distended gallbladder (arrow).

coronary artery aneurysm associated with it.[6] For a diagnosis of AAC, two of four ultrasonic criteria must be met: gallbladder distention; increased wall thickness (>3.5 mms); presence of sludge; or presence of pericholecystic fluid.[4] This diagnosis can be made by point-of-care ultrasound in the ED.

Some have suggested that medical treatment, rather than immediate surgical intervention, is the preferred way to manage AAC in children with KD.[6,13] The standard medical treatment for KD is high-dose aspirin (80–100 mg/kg/day) in conjunction with four doses of IVIG.[1] Although aspirin is thought to have antiplatelet and anti-inflammatory effects, it does not reduce the risk for coronary artery aneurysm formation. In contrast, IVIG has been shown to reduce this risk, although it is most effective when administered within 7-10 days of illness onset.[1] Even so, a retrospective study by Chen et al.[4] found that KD patients with AAC treated with IVIG were more likely to be IVIG-resistant than were KD patients without AAC, thus bringing into question whether patients with AAC should receive IVIG therapy.

CONCLUSION

Because KD may present similarly to other benign or potentially deadly diseases, it remains a challenging disease for emergency physicians to recognize. It is imperative that they be aware of the unusual presentations of the disease, such as AAC, and its association with coronary aneurysms. KD must always be considered in the differential diagnosis of a child with prolonged fever. The treatment of AAC in a child with KD is initially medical rather than surgical.

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Pitfalls and Pearls in Delusional Parasitosis

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Delusional parasitosis is an uncommon psychiatric disorder that manifests as having parasitic delusions. Due to its rarity, delusional parasitosis is a challenging and costly diagnosis of exclusion and proves difficult to manage for many providers. Although this syndrome is frequently discussed in psychiatric and dermatology reports, it is not commonly described in emergency medicine (EM) literature. As a result, best practices for workup and treatment remain unclear from an EM perspective. Patients typically return multiple times for medical evaluation and exhaust numerous resources. In this case report we review the appropriate steps for initial evaluation of patients with suspected delusional parasitosis, differential diagnoses, and increase awareness for prudent treatment strategies. [Clin Pract Cases Emerg Med. 2019;3(4):387–389.]

INTRODUCTION

We present a patient with delusional parasitosis who had a typical, although turbulent, medical workup in the emergency department (ED). Alternative organic causes to explain his behavioral changes were successfully ruled out. However, following an exhaustive compilation of tests and frustrating return visits, the patient still did not receive the proper treatment and was eventually lost to follow-up.

CASE REPORT

A 48-year-old male with a past medical history of cystic acne, but otherwise insignificant medication and social history, presented with concern for a parasite on his face. He reported that on the previous night he thought a parasite had crawled out of a healed cystic lesion on his right cheek. He presented a jar with a bloody napkin and paper, stating that he had scraped the parasite from his face with a scalpel and brought it with him to the ED. His physical exam was notable for diffuse excoriations and scaly patches, a 3 x 4 centimeter (cm) abrasion on his right cheek and mild anxiety. Otherwise his vital signs and physical exam were unremarkable.

No obvious parasites were appreciated on examination of his skin or in the jar. Basic blood tests including a complete blood count (CBC) and basic metabolic panel were within normal limits. A urine toxicology test was evaluated and reported negative. During his stay, the patient called a provider to the bedside to evaluate his thumb for a parasite actively crawling out of his skin. The provider documented that no obvious insects or wounds were apparent at that time. Delusional parasitosis was suspected and psychiatry was consulted, but the patient eloped from the ED prior to being evaluated.

The patient returned to the ED two days later with a complaint of parasitic infection. This time he described seeing white maggots crawling from his skin and presented us with a jar with a flaky material. The patient presented to the outpatient infectious disease (ID) clinic and was directed to come to the ED to obtain a formal ID consult. Although there was low clinical suspicion for an infectious component, ID was consulted for the presumed benefit of reassurance from a consultation service. The flakes presented by the patient were analyzed for ova and parasites and reported to the patient as negative. He was then evaluated by psychiatry who reported that his presentation was consistent with a delusional parasitosis but did not recommend inpatient hospitalization given that he did not appear to be at risk for harm to himself or others. He eloped a second time prior to receiving discharge papers.

The patient returned for a third visit to the ED with similar complaints. His workup included basic labs and a non-contrast head computed tomography (CT), which were all unremarkable. He was instructed to follow up with psychiatry as an outpatient.

DISCUSSION

This case represents a patient with delusional parasitosis, also known as Ekbom syndrome, psychogenic parasitosis, dermatophobia, chronic tactile hallucinosis, parasitophobia, and cocaine bugs. This disease is rare, occurring at an estimated incidence of 1.9 cases/100,000 person-years. The average demographic affected by delusional parasitosis is female in her late 50s with almost a 3:1 female to male incidence.[1] Delusional parasitosis is described as a condition where a patient has a fixed false belief that he or she is infected by parasitic organisms and cannot be persuaded otherwise.[2] These patients typically function normally in their daily lives outside of this focused delusion.

Primary delusional parasitosis occurs when the delusion is the only manifestation. Secondary delusional parasitosis occurs when the delusion is merely a symptom that occurs in the setting of another psychiatric disorder, medical illness, or substance abuse.[3,4] Patients with delusional parasitosis typically present to the ED seeking medical and dermatologic care. Pruritus is typically described, occurring over the course of months. Patients often times present "parasitic" specimens, which are actually scabs, cloth fibers, and other materials. The physical exam is typically notable for multiple ulcerations, excoriations, and scars secondary to intense scratching with fingernails, knives, or pins.[5]

Although primary delusional parasitosis is a relatively benign disease, the role of an emergency physician (EP) is to rule out other secondary diseases that manifest as parasitic delusions. A high level of suspicion for a secondary cause should be maintained in younger patients. Common disease culprits for secondary delusional parasitosis are head trauma, dementia, cerebrovascular disease, thyroid dysfunction, nutritional deficiencies (eg, niacin/cobalamin [B12]/folate), substance abuse, encephalitis, and actual parasitic infestations.[5]

Patients should be queried about recent travel history and exposure to known infected individuals with scabies or bed bugs. An objective ED workup that may help elucidate whether an underlying disease process is occurring includes a CBC (for eosinophilia), thyroid stimulating hormone, B12, folate, glucose, urea, liver function tests, urine toxicology, syphilis screen, human immunodeficiency virus testing, and head CT based on the history and physical exam. Lastly, some prescription and illicit drugs that have been associated with delusional parasitosis include ciprofloxacin, corticosteroids, topiramate, ketoconazole, and chronic alcohol (and withdrawal), cocaine and amphetamine use.[5,6,7,8]

Delusional parasitosis is a difficult syndrome to treat given patient noncompliance.[9] Patients typically deny that they have a delusional disorder and will seek multiple doctors and specialists to validate their symptoms.[10] Patients are often lost to follow-up since they commonly mistake their doctor's recommendations as a sign of incompetence or apathy. Therefore, outpatient providers are challenged with the difficult task of establishing a therapeutic and trusting relationship with the patient so that they may eventually accept recommendations and become compliant with treatment.

A systematic review of multiple case series and observational studies showed a 60-100% efficacy rate with antipsychotic medications such as pimozide, olanzapine, or risperidone.[11,12] Randomized controlled trials are difficult to conduct with this patient population, but some retrospective reviews note no difference between first- and second-generation antipsychotics in treatment success for delusional parasitosis.[12] A second-generation antipsychotic may be the more prudent treatment option given the lower rate of extrapyramidal side effects.[13,14]

EPs can play a role in treatment by reassuring these patients that there is no acute illness going on. Reassurance should be supported by negative laboratory and imaging studies. Furthermore, EPs should delicately acknowledge the patient's concern, being careful not to disregard them in a cavalier manner,

CPC-EM Capsule

What do we already know about this clinical entity?
Delusional parasitosis is a difficult diagnosis of exlcusion to make in the emergency department. Treatment and close follow-up are often unsuccessful given the vulnerable patient population.

What makes this presentation of disease reportable?
This presentation of delusional parasitosis is fairly common but outlines common pitfalls in diagnostic work-up and treatment of which many providers are unaware.

What is the major learning point?
An important treatment approach for delusional parasitosis is acknowledgement of a real medical problem, initiation of antispychotic therapy, reassurance, and proper followup with a trusted physician.

How might this improve emergency medicine practice?
A more tactical treatment approach may reduce emergency department repeat visits and wasted resources.

and recommend close follow-up with a physician with whom the patient can develop a trusting relationship.

CONCLUSION

EPs have the difficult task of discerning between psychiatric and organic causes of mental illness. Even when successfully identified, many psychiatric diseases are still challenging to treat due to unfamiliarity with the disorder, vulnerable patient population, and tendency for return visits. Delusional parasitosis represents one rare psychiatric illness that emergency providers may become more familiar with and thus develop better recommendations to ensure treatment compliance and close follow-up. Here we have outlined the appropriate history, physical exam, important differential diagnoses, and treatments that emergency physicians should be familiar with to manage delusional parasitosis.

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

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Difficult Intraoperative Heparinization Following Andexanet Alfa Administration

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Direct oral anticoagulants are now commonplace, and reversal agents are recently becoming available. Andexanet alfa (AnXa), approved by the United States Food and Drug Administration in 2018, is a novel decoy molecule that reverses factor Xa inhibitors in patients with major hemorrhage. We present a case of a 70-year-old man taking rivaroxaban with hemodynamic instability from a ruptured abdominal aortic aneurysm. He received AnXa prior to endovascular surgery, and intraoperatively he could not be heparinized for graft placement. Consideration should be given to the risks and benefits of AnXa administration in patients who require anticoagulation after hemorrhage has been controlled. [Clin Pract Cases Emerg Med. 2019;3(4):390–394.]

INTRODUCTION

Apixaban and rivaroxaban are direct oral anticoagulants (DOAC) that inhibit factor Xa (FXa), thereby preventing the conversion of prothrombin to thrombin and inducing coagulopathy.[1] Based on clinical trials that led to United States Food and Drug Administration (FDA) approval as well as post-marketing experience, DOACs carry a similar risk of life-threatening hemorrhage as compared to vitamin K antagonists such as warfarin.[2-5] Agents for reversal of DOAC-induced coagulopathy have only recently been developed. Andexanet alfa (AnXa) was reported by Lu et al. in 2013 to be a reversal agent for the FXa inhibitors. It is a decoy FXa molecule that avidly binds FXa inhibitors, temporarily inhibiting their anticoagulant effects.[6] It was also noted in vitro to have a binding affinity for heparin-bound antithrombin, thereby suggesting an effect against heparin-induced coagulopathy.[6-8]

AnXa was approved by the FDA in May 2018 under regulations allowing for Accelerated Approval for Biological Products for Serious or Life-Threatening Illnesses (21 CFR 601 Subpart E).[9] Approval was based on efficacy and safety data from the "Andexanet Alfa, a Novel Antidote to the Anticoagulation Effects of FXa Inhibitors Apixaban and Rivaroxaban" (ANNEXA-A and -R, respectively) trials in healthy volunteers, as well as from preliminary data from the "Andexanet Alfa, a Novel Antidote to the Anticoagulation Effects of Factor Xa Inhibitors" (ANNEXA-4) trial in patients with "acute major bleeding." ANNEXA-4 showed that AnXa has a marked, albeit transient, impact on the surrogate outcome of median anti-FXa activity among those on apixaban or rivaroxaban who have clinically significant hemorrhage.[10,11] Notably, ANNEXA-4 excluded patients requiring emergent surgery, and identified a 10% 30-day risk of thromboembolic events.[11] Following FDA approval, guidelines have been published recommending AnXa for the reversal of apixaban and rivaroxaban in patients with major hemorrhage, including those who require emergent surgery.[12,13]

A ruptured abdominal aortic aneurysm (AAA) is a rare but rapidly life-threatening illness, accounting for 6274 U.S deaths in 2017.[14] Survival depends on early identification, effective resuscitation, and rapid access to vascular surgery (VS). Increasingly, ruptured aneurysms are being managed endovascularly.[15] Somewhat counterintuitively, to reduce thrombotic complications these patients receive intraoperative heparinization once proximal control and hemostasis have been achieved.[16] For patients taking a DOAC with a ruptured aneurysm, initial emergency

department (ED) resuscitation may include reversal of anticoagulation. These decisions should be made in consultation with VS and should consider factors such as hemodynamic stability, concern for ongoing bleeding, and time to obtaining proximal control. It is unclear how treatment with AnXa would impact intraoperative anticoagulation and subsequent thrombotic risk.

CASE REPORT

A 70-year-old, 85-kilogram (kg) man with a history of atrial fibrillation on rivaroxaban, a known 4.5 centimeter (cm) infrarenal AAA, and remote three-vessel coronary artery bypass graft was transferred to our academic, tertiary referral center from a community hospital with the diagnosis of a ruptured AAA. He had last taken 20 milligrams (mg) of rivaroxaban the morning of presentation. At 2 PM, he developed abdominal pain associated with intermittent ripping sensations into his back. He presented to the outside hospital somnolent, with a systolic blood pressure of 80 millimeters of mercury (mmHg). Computed tomography with angiogram demonstrated rupture of his AAA with an associated retroperitoneal hematoma. He received two units (U) of packed red blood cells (pRBC) prior to transfer.

The patient arrived in our ED at 9:35 PM, appearing ashen and uncomfortable. He had a new oxygen requirement of four liters (L) by nasal cannula, a respiratory rate of 24 breaths per minute, a heart rate of 103 beats per minute, and a blood pressure of 122/85 mmHg. The VS team was emergently consulted and he was taken to the operating room (OR) for endovascular aortic repair (EVAR) at 10:57 PM. In our ED, laboratory studies showed the following: hemoglobin 13.0 grams per deciliter (g/dL) (13.7-17.5g/dL), creatinine 1.1 mg/dL (0.5-1.2mg/dL), international normalized ratio (INR) 2.6 (0.9-1.1), and partial thromboplastin time 28.0 seconds (s) (25.0-36.5 s).

Due to his hypotension at the outside hospital, poor skin perfusion, supplemental oxygen requirement, and history of recent rivaroxaban dose with elevated INR qualitatively suggesting coagulopathy and ongoing bleeding, the patient was ordered for low- dose AnXa (400 mg intravenous bolus followed by 4 mg/minute infusion) in consultation with VS. Due to the long preparation time, the AnXa was not immediately available in the ED, and it was started at 11:07 PM by the anesthesiologist in the OR. Two additional units of pRBCs were administered at 11:30 PM and 11:44 PM in the setting of persistent tachycardia to the 120s. A timeline of the operative course is outlined in Figure 1.

Intraoperative heparinization (typically 80-100 U/kg) is used to reduce thromboembolic risk during EVAR.[16] Intensity of anticoagulation is monitored by activated clotting time (ACT), with target values greater than 250 seconds This patient's initial ACT by point-of-care assay was 135 at 11:48 PM, approximately 40 minutes after the AnXa bolus and two-hour infusion was started (non-coagulopathic range 80-120 s). After obtaining hemostasis, the VS team proceeded with anticoagulation. The patient was bolused with unfractionated heparin (UFH) 8000 U between 11:49 PM and 12:04 AM, and a repeat ACT at 12:06 AM was minimally changed (144 seconds). Over the next hour an additional 6000 U UFH were given, and the ACT remained well below the 250-second goal. The hematology service was consulted intraoperatively and recommended transfusing a unit of fresh frozen plasma, which was administered at 1:19 AM. For the remainder of the surgery, which ended at 1:46 AM, the ACT never exceeded 152 seconds.

The VS team continued with aortic graft deployment despite subtherapeutic heparinization. The aneurysm extended into the right common iliac artery; therefore, to obtain adequate endovascular seal and exclusion of the aneurysm, the endograft's right limb was extended into the right external iliac artery necessitating coverage of the right hypogastric artery. The left iliac limb terminated just proximal to the left hypogastric artery. Completion angiogram following

CPC-EM Capsule

What do we already know about this clinical entity?
Andexanet alfa is a reversal agent for direct Factor Xa inhibitors. It may be valuable in patients with major hemorrhage requiring operative intervention.

What makes this presentation of disease reportable?
A patient on rivaroxaban with a ruptured aortic aneurysm received andexanet alfa. This complicated his emergent aortic repair by inhibiting necessary heparinization.

What is the major learning point?
Patients requiring endovascular surgery require careful consideration prior to administration of andexanet alfa.

How might this improve emergency medicine practice?
Increased communication with vascular surgeons regarding the use of andexanet alfa in endovascular candidates may help prevent operative complications.

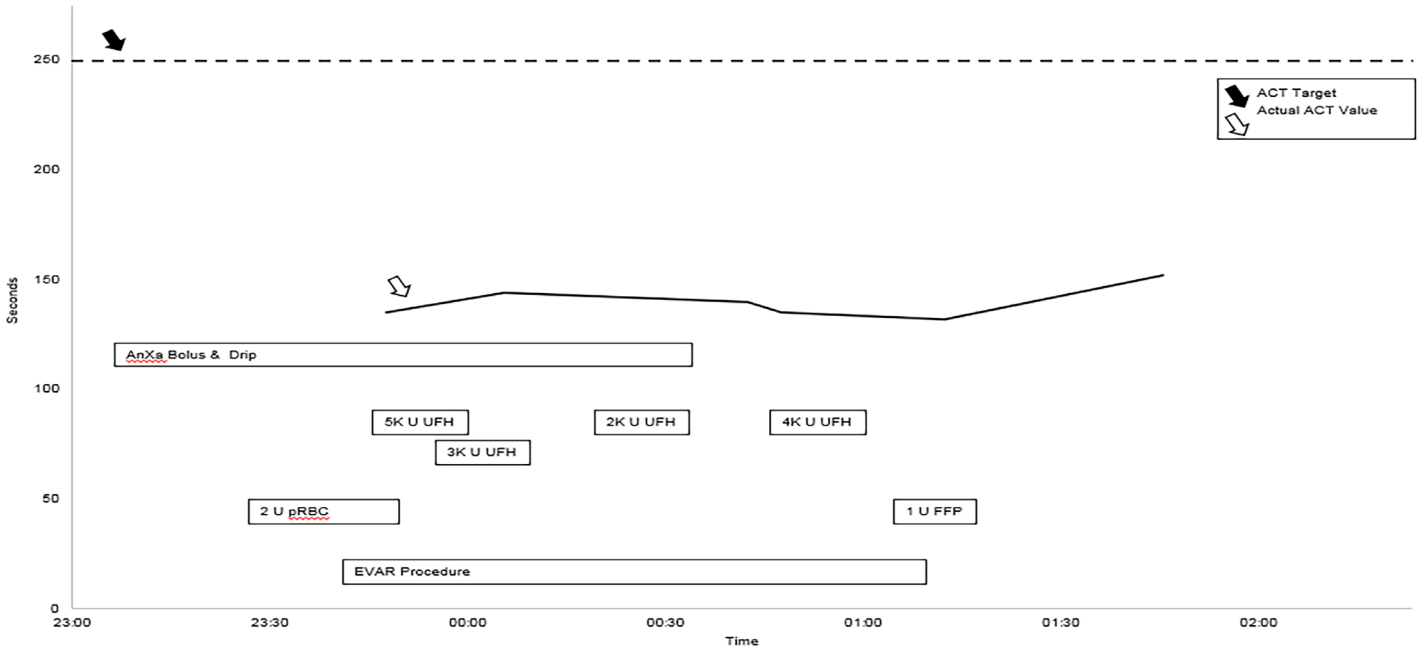

Figure 1. Timeline in operating room. Boxes represent administration of medications and blood products, as well as timing of the surgical procedure. The solid line represents the measured activated clotting time, which never became therapeutic (dashed line). *ACT,* activated clotting time; *AnXa,* Andexanet alfa; *K,* thousand; *U,* units; *UFH,* unfractionated heparin; *pRBC,* packed red blood cells; *FFP,* fresh frozen plasma; *EVAR,* endovascular repair.

deployment revealed adequate exclusion of the aneurysm; however, there was evidence of thrombus in the iliac system including the left hypogastric artery.

Following repair, the patient was admitted to the surgical intensive care unit, where he was monitored for four days. He was discharged with no clinical ischemic complications despite extensive thrombosis of his pelvic vasculature. Interestingly, the patient's INR remained persistently elevated (1.7–2.1) for the remainder of his hospitalization despite remaining off rivaroxaban. The hematology service ordered a mixing study and factor VII (FVII) concentration, which was low at 21% (normal 65-180%), suggesting a congenital FVII deficiency. He was discharged on rivaroxaban 20 mg daily. At hematology clinic follow-up, the patient was well, and his outpatient FVII level remained low (18%) and INR remained high (3.3). This is consistent with congenital FVII deficiency. It is unlikely that the FVII deficiency played a role in the subtherapeutic intraoperative heparinization, as FVII deficiency is a coagulopathy and affects a different point within the coagulation cascade.

DISCUSSION

We have presented the case of a 70-year-old man on rivaroxaban who suffered a ruptured AAA, received AnXa to reverse FXa inhibitor-induced coagulopathy, and during EVAR was difficult to anticoagulate, thus putting him at increased post-operative thrombotic risk. He sustained thrombosis of his pelvic vasculature without any clinical adverse consequences. This case is highly clinically relevant given the novelty of both the agent and the intraoperative complications at hand; in fact, this case is being published within the clinical pharmacy literature as well with a focus on the pharmacology of the heparin-AnXa interaction and the valuable role of pharmacists in making decisions regarding reversal of anticoagulation.[17]

This case highlights emerging considerations regarding the use of AnXa. This patient underwent an endovascular procedure, the safety profile of which is dependent on intraoperative heparinization. While in vitro data demonstrates inhibition of heparin by AnXa, this relationship has not been clinically studied and is not widely known. AnXa reversibly binds to the heparin-antithrombin complex, rendering heparin ineffective. Therefore, when surgical control of hemorrhage is rapidly available, and intraoperative anticoagulation is important, administration of AnXa may not be warranted. Emergency physicians (EP) should engage their surgical consultants to discuss risks and benefits of using this reversal agent preoperatively.

However, if the EP is caring for a similar patient in a facility without definitive surgical capabilities, AnXa administration may still be a valuable part of the initial resuscitative bundle prior to transfer to a referral center. Currently, AnXa is not widely available in all hospitals, but

this is expected to change as manufacturing and distribution improves. AnXa would normalize coagulation parameters during the highest risk period of interfacility transport, while its short half-life would allow it to clear by the time the patient arrives at the referral center. The ANNEXA-A, -R, and -4 studies demonstrated a relative return to expected pre-AnXa anti-FXa activity four hours post-bolus. Ideally, this decision should be made collaboratively between the ED and surgical teams. Notably, if a clinician is faced with needing to anticoagulate a patient after AnXa is administered, a potential strategy would be to use a direct thrombin inhibitor (argatroban or bivalirudin), as the therapeutic effect would be downstream of AnXa in the coagulation cascade. If a patient receives AnXa, it is critical to inform the surgical team, so that they can plan on using one of these alternative agents for intraoperative anticoagulation.

Based on ANNEXA-4, 10% of patients administered AnXa suffered from a thromboembolic event within 30 days of administration.[11] This is somewhat comparable to the venous thromboembolism rate of 5-7% from other anticoagulant reversal strategies such as prothrombin complex concentrates. Patients requiring surgery with a baseline thrombotic risk creates a difficult decision for the EP regarding the use of any reversal agent.

Ultimately, AnXa is a valuable intervention for the reversal of major hemorrhage in the setting of FXa inhibitor use. Less is understood about the role of AnXa in patients with aortic catastrophe for whom definitive management requires surgery with intraoperative heparinization, and for whom there already exists an intra- and postoperative thrombotic risk. Based on this case, the decision to use AnXa should be made with collaboration between the ED, VS, and pharmacy teams, and should take into consideration the patient's acute instability, time to surgical control of the aorta, inability to heparinize in the presence of AnXa, and the baseline operative thrombotic risk.

CONCLUSION

We present a case of a 70-year-old man taking rivaroxaban who suffered a ruptured AAA and received AnXa for stabilization of retroperitoneal hemorrhage, but then could not be adequately heparinized during EVAR. Although it is has been established that AnXa will prevent anticoagulation with heparin, this is not well known within the clinical community. Patients who require heparinization for endovascular surgery may not be appropriate for immediate preoperative AnXa administration.

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

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Ventricular Fibrillation Cardiac Arrest in Young Female from Diffuse Left Anterior Descending Coronary Vasospasm

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This is a case of the most severe and potentially fatal complication of coronary artery vasospasm. We report a case of a 40-year-old female presenting to the emergency department (ED) via emergency medical services with chest pain. The patient experienced a ventricular fibrillation cardiac arrest while in the ED. Post-defibrillation electrocardiogram showed changes suggestive of an ST-elevation myocardial infarction (STEMI). Cardiac catheterization showed severe left anterior descending spasm with no evidence of disease. Coronary vasospasm is a consideration in the differential causes of ventricular fibrillation and STEMI seen in the ED. [Clin Pract Cases Emerg Med. 2019;3(4):395–397.]

INTRODUCTION

Although previously thought to be rare, recent literature suggests that coronary artery vasospasm can lead to ventricular fibrillation.[1] Recognizing this etiology is often difficult. Patients may not experience chest pain and may be younger and with fewer risk factors than those more usually diagnosed with acute coronary syndrome.[2] Patients who experience ventricular fibrillation, often out of hospital, likely will present to the emergency department (ED) after out-of-hospital cardiac arrest or may experience cardiac arrest while in the ED. This is an important consideration to emergency physicians (EP) as this is a more prevalent cause of sudden cardiac death than previously thought.[1,3,4,5] Given the risk of recurrence of arrythmia with spasm in patients who have previously experienced arrest, an EP can make a significant impact on the patient outcome by recognizing this as the causative etiology.[4] Medical therapy with calcium-channel blockers and implanted cardiac defibrillators is the current method of preventing recurrence.[4,5]

CASE REPORT

A 40-year-old female presented to the ED with past medical history of non-ST elevation myocardial infarction (NSTEMI) from proposed coronary artery disease, hypertension, hypothyroidism, anxiety, depression, asthma, and tobacco use. The patient reported chest pain that started at rest and was associated with shortness of breath and radiation to the jaw and left arm. Although previously effective, nitroglycerin tablets did not relieve her pain, which prompted her to call emergency medical services (EMS). EMS administered 324 milligrams of aspirin and three nitroglycerin tablets to the patient. Upon EMS presentation to the ED, she became unresponsive and was noted to be pulseless. Cardiopulmonary resuscitation was initiated, with a heart rhythm of ventricular fibrillation. The patient was successfully defibrillated and had return of spontaneous circulation (ROSC). An electrocardiogram (ECG) (Image 1) immediately post ROSC showed ST elevations in anterolateral and inferior leads.

The patient was taken emergently to the cardiac catheterization laboratory. The cardiac catheterization revealed the following: The left anterior descending artery (LAD) was without atherosclerotic disease; the LAD supplied the apex as well as inferior wall of the heart; and the proximal LAD was normal and large, while the mid and distal LAD were in diffuse spasm (Image 2).

The spasm was not present on the patient's previous cardiac catheterization. The spasm did not respond to intracardiac nitroglycerin. The ejection fraction was reduced to 35%. Post catheterization the patient was started on a diltiazem infusion, and cardiac enzymes began trending down. Trans-thoracic echo two days later reflected a left ventricular ejection fraction of 45-49% with a moderate- sized apical wall motion abnormality with akinesis of the segments. She remained stable and was maintained on diltiazem in an attempt to prevent reoccurrence of the coronary vasospasm. She was fitted with a wearable

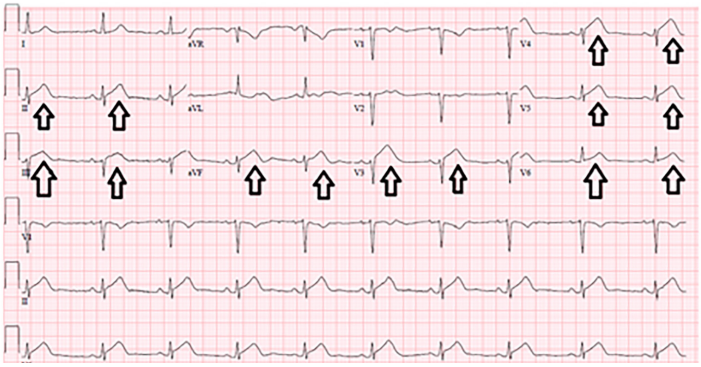

Image 1. Emergency department electrocardiogram showing anterolateral and inferior ST elevation. Arrows indicate ST elevation.

defibrillator and discharged four days after arrival to the ED. Against cardiologist recommendation, the patient chose not to pursue an implantable cardiac defibrillator (ICD).

The patient had a follow-up with cardiology at one month, six months, one year and two years. She continued her beta blocker and calcium-channel blocker. Repeat echo at one-month post event showed a preserved left ventricular function with multiple wall motion abnormalities. She had no symptoms of chest pain since her event but did complain of shortness of breath with exertion. She again declined an ICD. The patient reported doing well overall and has optimized her medical management at subsequent follow-up visits.

CPC-EM Capsule

What do we already know about this clinical entity?
Coronary artery vasospasm is a known cause of sudden cardiac death. Current literature indicates it may be a more common cause of cardiac arrest than was once thought.

What makes this presentation of disease reportable?
This patient was not the typical age or gender for sudden cardiac death.

What is the major learning point?
In patients outside of the typical age, gender, and risk-factor profile for sudden cardiac death, coronary artery vasospasm is a possible etiology.

How might this improve emergency medicine practice?
This case highlights an atypical patient at risk for sudden cardiac death, and the electrocardiogram and heart catheterization that demonstrated severe coronary artery vasospasm.

DISCUSSION

Coronary artery vasospasm leading to cardiac arrest was initially thought to be a rare event, but recent reports suggest it might be more common than previously reported.[1,4,6] A recent study showed that coronary vasospasm accounted for 7% of out-of-hospital cardiac arrests.[1,4] Another study has reported a rate as high as 11%.[6]

We identified three case reports in the literature. A 43-year-old had an out-of-hospital ventricular fibrillation arrest, and the initial cardiac catheterization showed nonobstructive disease. This patient developed chest pain one week later, and repeat catheterization showed severe spasm of the right coronary artery.[7] Another case described the presentation of a 68-year-old without chest pain who had two episodes of syncope and then experienced a ventricular fibrillation cardiac arrest secondary to spasm of the right coronary artery.[8] Finally, the literature describes a 46-year-old woman who had an out-of-hospital cardiac arrest secondary to vasospasm.[9] Of note, a year prior she had chest pain; cardiac biomarker elevations and a catheterization showed normal coronary arteries. She was placed on a calcium-channel blocker, yet she experienced an adverse cardiac event one year later. Given the literature, our patient's reported etiology of NSTEMI prior to the event presented here is called into question. It is possible that her prior NSTEMI actually was a presentation of coronary artery vasospasm without sudden cardiac death. Our case is unique in that the cardiac arrest occurred in in the ED.

When treating these patients, the benefit of an ICD should be evaluated. Research suggests that there are no reliable ways to predict risk of recurrence ,and thus ICD placement with medications for vasospasm are advised.[5] In our case, after the event it was highly recommended that the patient receive an ICD, which she declined. Additionally, she was given a wearable defibrillator but was not compliant with wearing it. Research has shown that patients who survive a coronary artery vasospasm that leads to a lethal ventricular arrhythmia have a very high risk of recurrence.[2] Based on a novel risk-stratification system,[10] the patient in our case is considered high risk and has an approximately 13% risk of recurrent adverse cardiac event. One study also suggests that the rate of recurrence continues to rise across the four years after initial coronary vasospasm.[3]

CONCLUSION

This case adds to previously described literature by describing a ventricular fibrillation cardiac arrest secondary coronary artery vasospasm in the ED. Immediately following ROSC, our patient had ECG findings suggestive of STEMI, but with a cardiac catheterization that was suggestive of severe coronary artery vasospasm. Uniquely, this patient was followed for almost two years post-event and remained without neurological sequelae from the cardiac arrest event.

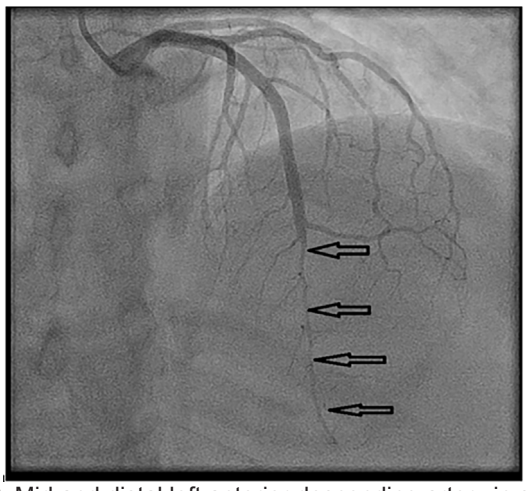

Image 2. Mid and distal left anterior descending artery in severe vasospasm. Arrows indicate left anterior descending vasospasm.

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

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A Rare Oncologic Emergency: Spontaneous Tumor Lysis Syndrome in Metastatic Colon Adenocarcinoma

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Tumor lysis syndrome is an oncologic emergency that can present with variable symptoms and is truly a laboratory-based diagnosis without pathognomonic clinical findings. The classical teaching is to consider this diagnosis in cancer patients undergoing chemotherapy. We present the case of a 66-year-old female with newly diagnosed metastatic liver adenocarcinoma, not on chemotherapy, who was diagnosed with spontaneous tumor lysis syndrome. Cognizance of this syndrome and associated laboratory findings are paramount to diagnosis and rapid intervention. [Clin Pract Cases Emerg Med. 2019;3(4):398–400.]

INTRODUCTION

Tumor lysis syndrome (TLS) is an oncologic emergency seen in patients with rapidly proliferating hematologic malignancies following exposure to systemic chemotherapy. It is a metabolic disorder characterized by hyperuricemia, hyperkalemia, hypocalcemia, and hyperphosphatemia, which can result in acute kidney injury, cardiac arrhythmias, and central nervous system toxicity. Spontaneous TLS (STLS) is rare and occurs in the absence of cytotoxic therapy. It is characterized by spontaneous cellular death in rapidly proliferating malignancies. STLS in patients with solid cancers is associated with a high mortality.[1] We present a case of a woman recently diagnosed with metastatic colon adenocarcinoma who presented with STLS.

CASE REPORT

A 66-year-old female with past medical history of hypertension, hyperlipidemia, and diabetes mellitus presented to the emergency department (ED) with subacute weakness and dyspnea. Five weeks prior to this presentation, she was seen in the ED for abdominal pain and dyspnea and was found to have large, bilateral pleural effusions and anemia. Extensive further work-up revealed a diagnosis of stage IV, poorly differentiated adenocarcinoma of the transverse colon. Imaging revealed diffuse large lymphadenopathy throughout the abdomen, retroperitoneum, and mediastinum with multiple pulmonary nodules. She underwent an open extended right hemicolectomy with ileostomy as well as resection of the jejunum due to tumor invasion. Thoracentesis was performed and she was eventually discharged home to follow up with oncology for discussions about immunotherapy or chemotherapy.

At the current ED visit, the patient's vitals were as follows: blood pressure 121/39 millimeters of mercury, pulse rate of 88 beats per minute, respiratory rate of 20 breaths per minute, and an oxygen saturation of 98% on three liters of oxygen via nasal cannula. Physical examination was pertinent for an ill-appearing female with mild jaundice, abdominal distention with mild tenderness, and decreased breath sounds with rales in the bilateral lower lung fields. A chest radiograph showed bilateral pleural effusions. The complete blood count was unremarkable. The complete metabolic

panel demonstrated hyponatremia, hyperkalemia, uremia, acute kidney injury, and transaminitis (Table). Given the new acute kidney injury and hyperkalemia, there was suspicion for STLS. Additional diagnostics were obtained and revealed hypocalcemia, hypermagnesemia, hyperphosphatemia, and hyperuricemia (Table). Electrocardiogram demonstrated minimal changes in T-wave morphology as well as a slight increase in QRS duration. Review of the patient's chart showed that all of the laboratory abnormalities were new in comparison to four weeks prior.

A diagnosis of STLS was made in this patient with metastatic, poorly differentiated adenocarcinoma, who had not been initiated on systemic chemotherapy. Intravenous crystalloids, rasburicase, and treatment for acute hyperkalemia with calcium gluconate, insulin, dextrose, kayexalate, and sodium bicarbonate were initiated. After consulting the renal and oncology teams, the patient was admitted. Given the poor prognosis, the patient declined dialysis and further invasive measures. Her status was changed to comfort measures only and she died two days later.

CPC-EM Capsule

What do we already know about this clinical entity?
Tumor lysis syndrome (TLS) is an oncologic emergency identified by lab abnormalities: hyperkalemia, hyperphosphatemia, hyperuricemia, hypocalcemia, and acute kidney injury.

What makes this presentation of disease reportable?
TLS may occur spontaneously, but typically it occurs in the setting of hematologic malignancies. Spontaneous TLS in solid tumors is rare.

What is the major learning point?
The classic lab abnormalities of TLS should prompt consideration of this complication in any oncology patient, regardless of concomitant chemotherapy.

How might this improve emergency medicine practice?
Early recognizance of TLS can lead to rapid management, mitigation of complications, and improved emergency medical care.

DISCUSSION

TLS can be therapy-related (cytotoxic therapy) or spontaneous (no exposure to cytotoxic therapy). TLS is more commonly seen in patients with hematologic malignancies compared to solid malignancies, due to increased proliferation rates and sensitivity to cytotoxic therapy.[1-4] Solid tumors with a large tumor burden are at increased risk of TLS. STLS, which occurs in the absence of cytotoxic therapy, is more rare and most often seen in hematologic malignancies with high proliferation rates, such as acute leukemia and aggressive lymphomas, such as Burkitt lymphoma.

One retrospective review found that STLS occurred in 1.1% of patients with hematologic malignancy and acute renal failure.[4] The rate of STLS in patients with solid malignancies is even lower, with the true prevalence remaining uncertain due to a paucity of literature comprised mainly of case reports.[4] It has been described in patients with metastatic and advanced malignancies including small cell lung cancer, cholangiocarcinoma, hepatocellular carcinoma, and only two other cases of colon adenocarcinoma.[1,3-5] The laboratory abnormalities outlined in the Cairo-Bishop definition of TLS include hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia, either outside the reference range or a 25% change from baseline.[2] Potential complications of these metabolic abnormalities include acute kidney injury, cardiac arrhythmias, and seizures.[1-4]

There is a 20-50% mortality rate in TLS occurring in solid tumors if undiagnosed, or diagnosed too late.[6] Spontaneous development of TLS in solid tumors reflects large tumor burden and likely portends an even higher mortality. Timely recognition and intervention following a diagnosis of STLS is vital. Management consists of intravenous hydration, correction of electrolyte abnormalities, and treatment of hyperuricemia, which can lead to obstructive uropathy. Allopurinol can be used prophylactically in patients at high risk for TLS, but should not be used for hyperuricemia in acute TLS.[5] Acute hyperuricemia in TLS or STLS can be treated with rasburicase, a recombinant urate oxidase enzyme that catalyzes uric acid to allantoin, or hemodialysis. Patients should have strict monitoring of urine output as persistent oliguria may be an additional indication for hemodialysis, along with hyperkalemia and hyperuricemia.

Our patient had been recently diagnosed with metastatic colon adenocarcinoma with diffuse metastases. We believe the heavy tumor burden and rapid proliferative rate increased her risk of developing STLS.

CONCLUSION

Consideration of a diagnosis of TLS was historically reserved for patients with the classic lab abnormalities who were undergoing chemotherapy. Reports of STLS in

Table. Laboratory values of patient with tumor lysis syndrome.

Component	Value	Reference range
White blood cells	9.7	4.0-11.0 k/mm^3
Hemoglobin	11.4	11.7-15.5 Gm/dL
Hematocrit	36.9	35.7-45.8%
Platelet count	473	150-460 k/mm^3
INR	1.2	0.9-1.1
Prothrombin time	12.6	9.7-12.2 seconds
Sodium	123	133-145 mmol/L
*Potassium	7.7	3.6-5.2 mmol/L
Chloride	87	98-107 mmol/L
Bicarbonate	16	22-29 mmol/L
Anion gap	20	4-17
Glucose	137	70-99 mg/dL (fasting)
BUN	79	8-23 mg/dL
Creatinine	6.4	0.5-1.0 mg/dL
*Calcium, Ionized	1.02	1.13-1.32 mmol/L
Magnesium	2.4	1.3-1.9 mEq/L
LDH	544	94-250 units/L
Alkaline phosphatase	882	35-104 units/L
AST	81	0-32 units/L
ALT	36	0-33 units/L
Bilirubin, total	5.9	0-1.2 mg/dL
Lactate	1.7	0.5-2.2 mmol/L
Troponin T Quant	<0.01	0.0-0.09 ng/mL
Nt-ProBNP	3,029	0-125 pg/mL
Haptoglobin	334	30-200 mg/dL
*Uric acid	23.9	1.6-7.6 mg/dL
*Phosphorus	10.1	2.5-4.5 mg/dL

*Laboratory abnormalities listed in the Cairo-Bishop definition of tumor lysis syndrome (*TLS*). Creatinine ≥1.5x upper limit of normal meets criteria for clinical TLS, along with the defined laboratory changes (2).
k, kilogram; *mm*^{3}, cubic millimeter; *gm*, gram; *dL*, deciliter; *INR*, international normalized ratio; *mmol*, millimole; *L*, liter; *mg*, milligram; *BUN*, blood urea nitrogen; *mEq*, milliequivalent; *LDH*, lactate dehydrogenase; *AST*, aspartate aminotransferase; *ALT,* alanine aminotransferase; *ng*, nanogram; *mL*, milliliter; *pg*, picogram.

both hematologic and solid malignancies are increasing in the literature. Many of these patients will first present to the ED and it is pertinent for the emergency physician to recognize this oncologic emergency. Striking electrolyte imbalances and acute kidney injury in any cancer patient ought to prompt consideration of STLS, as the associated complications can be life-threatening.

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

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Ketamine Implicated in New Onset Seizure

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Ketamine is used widely in emergency departments for a variety of purposes, including procedural sedation and pain management. A major benefit of using ketamine is the rapid onset and lack of respiratory depression. The known side effects include emergence reactions, hallucinations, hypertension, dizziness, nausea, and vomiting. Recent studies have shown the benefit of ketamine for refractory status epilepticus; however, this application of the drug is still being studied. We present a case where ketamine likely induced a seizure in a patient on whom it was used as a single agent in procedural sedation. Seizure is not a known side effect of ketamine in patients without a seizure history. Given the eagerness over additional uses for ketamine, this novel case of a seizure following procedural sedation with ketamine should be of interest to emergency providers. [Clin Pract Cases Emerg Med. 2019;3(4):401–404.]

INTRODUCTION

Ketamine is a versatile and effective drug used commonly in the emergency department (ED) to aid in the treatment of various pathologies. Although it is now commonly administered in EDs, it was first made available for commercial use in 1970. The initial use of ketamine was as a rapid-acting, dissociating anesthetic.[1] Ketamine, a derivative of phencyclidine, was initially created to reduce the adverse psychotomimetic effect and lessen the abuse potential when compared to its parent drug. Because of its effect profile, phencyclidine was removed from the market in 1978.[2] Ketamine is desirable for anesthetic use in procedural sedations, due to its rapid onset, relatively short half-life, and lack of respiratory depression.[3]

Although initially used as an anesthetic, ketamine has had evolving implications for other therapeutic uses. Dosing and route of administration vary for its evolving clinical uses; however, a dose of 1-2 milligrams per kilogram (mg/kg) is typically administered intravenously (IV) during procedural sedations. Ketamine causes a dissociative sedation, defined as sedation without a complete loss of consciousness but associated with a catatonic state, as well as amnesia. The drug has been used for procedural sedation, treatment of agitated delirium, analgesia and anti-inflammatory effects, treatment of depression and schizophrenia, and more recently for refractory status epilepticus[3,4]; however, like other medications, it is not without side effects. Adverse effects of ketamine that have been well described include the following: psychoactive effects such as hallucinations; visual disturbances including diplopia and ocular nystagmus; potential for abuse; emergence reactions as the medication wears off; dizziness; and nausea and vomiting.[3]

Despite reports of recent uses of ketamine for the treatment of seizures, we describe a case where a 15-year-old autistic girl, without a prior seizure history, received ketamine for a procedural sedation and had a new onset seizure during the process with a prolonged postictal period – a previously unreported adverse effect of the drug.

CASE REPORT

A 15-year-old female with a past medical history of only autism spectrum disorder presented to the ED with complaints of a laceration to her right fourth digit and an abrasion to her right third digit, which were sustained approximately 22 hours prior to ED arrival. Before presenting to the ED, the patient's mother attempted wound care at home; however, secondary to persistent bleeding, the patient was brought to a referral urgent care center that then subsequently sent the patient for evaluation at our ED.

On evaluation in the ED, the patient was at her baseline mental status as per her parents. Physical examination revealed a weight of 53 kg, a temporal temperature of 37

degrees Celsius, a heart rate of 71 beats per minutes, a respiratory rate of 18 breaths per minute, a blood pressure of 114/86 millimeters of mercury, and a room air oxygen saturation of 99%. She was noted to have a small abrasion to the medial aspect of the distal phalanx of the right third finger and a one-centimeter, elliptical-shaped laceration to the medial aspect of the distal right fourth finger that was actively bleeding. She had full range of motion of all 10 of her digits with a capillary refill of less than two seconds on each digit. Of note, her neurologic exam revealed the patient to be at her baseline status as per her parents, awake and alert, and she was symmetrically moving all of her extremities equally. The remainder of her examination was normal.

Due to continued bleeding of the laceration despite other attempts at hemostasis, and after a discussion with the patient's parents regarding increased risk of infection because the wound had been open for 22 hours, they consented to have the laceration repaired with sutures in the ED. Because of the patient's baseline mental status, secondary to her autism spectrum disorder, the decision was made to perform the laceration closure under procedural sedation.

For the procedural sedation, there was no IV access available; thus, the medication was administered intramuscularly. It had been greater than four hours since her last meal, she was able to fully flex and extend her neck, and she was able to fully open her mouth. We chose ketamine as the sedating agent for the procedure at a dose of 3-5 mg/kg, which is in the accepted intramuscular dosage range. Consent for the sedation was obtained from her parents after a full discussion regarding the risks and benefits of sedation and the known adverse effects of ketamine.

Ketamine was administered via a one-time dose intramuscularly at the start of the procedure; the total dose given was 150 mg, which was less than the recommended intramuscular dose. The patient was on a cardiac monitor with pulse oximetry and end-tidal carbon dioxide monitoring throughout the procedure and remained normoxic. The laceration on the right fourth digit was repaired using an aseptic technique after cleansing of the site, with four 4-0 chromic gut sutures. No local anesthetic was administered. As the last stitch was tied and nine minutes after ketamine was administered to the patient, she was observed to have generalized tonic-clonic seizure of one-minute duration that self-resolved without any administration of further medications. Medical toxicology was consulted and advised to evaluate the patient for a new onset seizure, as ketamine was not known to induce seizure activity.

The patient remained postictal in the ED, IV access was established, and a complete blood count, basic metabolic panel, and a computed tomography (CT) without contrast of the head were ordered. Blood analysis was noted to be within normal limits for our institution's normal value range. The head CT was read by radiology to show mild colpocephaly of the ventricles with uncertain significance as there was a present corpus callosum and no evidence of transependymal resorption or other white matter findings.

Pediatric neurology was consulted, and the case, laboratory findings, and imaging findings were discussed. Pediatric neurology recommended inpatient admission if the patient maintained altered mental status and to otherwise follow up with them for an electroencephalography (EEG) due to new onset seizure. The patient continued to be hemodynamically stable in the ED and maintain her airway, but did not return to her baseline and had one episode of vomiting despite observation for four hours after the observed seizure activity. At that time, she was admitted to the pediatric step-down unit for further evaluation and monitoring.

While in the pediatric step-down unit, the patient gradually returned to her baseline mental status; she had no further seizure activity throughout her stay and required no administration of any medications. She was evaluated by neurology and

CPC-EM Capsule

What do we already know about this clinical entity?
Ketamine is a commonly administered sedative used in emergency departments for procedural sedation in pediatric patients, including those with autism spectrum disorder (ASD) and other developmental delays.

What makes this presentation of disease reportable?
We present a patient without a prior history of seizures, known to have ASD and undiagnosed colpocephaly who experienced a seizure after administration of intramuscular ketamine for laceration repair.

What is the major learning point?
Ketamine can have adverse effects such as inducing seizures in patients with undiagnosed colpocephaly or developmental delays as described in this case.

How might this improve emergency medicine practice?
Emergency physicians have multiple different options to consider for procedural sedation and should consider pros and cons to each agent prior to their administration.

underwent a one-hour EEG, which showed no areas of focal slowing, epileptiform discharges, or electrographic seizures. The patient was discharged in stable condition after undergoing the EEG to outpatient follow-up on a course of cephalexin for empiric antibiotic coverage of her repaired finger laceration. The patient was then lost to follow-up.

DISCUSSION

For nearly 50 years, ketamine has proven to be a safe anesthetic drug with potent analgesic properties and a wide range of uses in clinical practice.[2] Emergency physicians have become comfortable with using this drug regularly, for both procedural sedation and management of pain. The benefits, as well as the known side effects, have been well documented. It is considered relatively safe when used with proper precautions in the ED and even in the prehospital setting and has been successfully administered to pediatric patients with autism spectrum disorder for routine ED procedures, such as laceration repair.[4]

One of the newer reported uses for ketamine is for the treatment of refractory status epilepticus. Researchers have shown that during prolonged seizures, the number of activated gamma-aminobutyric acid (GABA)-A receptors on the postsynaptic membrane gradually decreases, whereas the number of inactive GABA-A receptors increases.[5,6] This causes a significant reduction in the efficacy of antiepileptic drugs that target the GABAergic system, including benzodiazepines, propofol, and phenobarbital. In contrast, the number and activities of N-methyl-D-aspartate (NDMA) receptors increases over time.[7] Subsequently, a drug such as ketamine, which is a noncompetitive NDMA receptor antagonist, may play a role in treating status epilepticus. However, as this case clearly illustrates, ketamine may actually induce seizures in certain populations.

Ketamine use can cause excitatory effects on the central nervous system. The U.S. Food and Drug Administration already recommends that ketamine be contraindicated in patients with severe hypertension and allergies to ketamine. It should be used with caution in patients with coronary artery disease, heart failure, glaucoma, atherosclerosis, pulmonary heart disease, pulmonary hypertension, severe intracranial hypertension, pregnancy, history of mental illness, hyperthyroidism, tachyarrhythmia, adrenal pheochromocytoma, and alcoholism.[7] Despite this extensive list of exclusions, there is no caution about administration of ketamine to patients with autism. Additionally, ketamine has been recommended by multiple studies, including in recent literature, as a procedural sedation agent in pediatric patients with autism spectrum disorder for use in laceration repair.[4]

There is no evidence that ketamine is likely to precipitate generalized convulsions, even in patients with both a history of epilepsy and an abnormal EEG.[8] Convulsive activity and other deleterious neurologic sequelae may be seen in ketamine toxicity from overdose. Ketamine toxicity is more commonly seen when ingested recreationally, rather than in clinical practice. During recreational ingestion, ketamine may be laced with other contaminants and may not be a pure drug; thus, it is far from clear whether the clinical effects seen are due to ketamine or an adulterant found within an admix of a street formulation. Furthermore, what we know about ketamine toxicity and overdose is from animal literature and occurs at significantly higher doses than those administered to the patient in the case we have presented here.[9]

CONCLUSION

Notwithstanding a lack of literature to support it, this case suggests that ketamine induced a seizure in a patient with no prior history of epilepsy. As more research emerges on the use of ketamine for refractory status epilepticus in addition to its other broad range of uses in the ED, perhaps the list of its side effects should be expanded more broadly. More research is needed regarding the administration of ketamine to patients with cognitive conditions, such as autism, and neuroanatomical abnormalities such as colpocephaly, as they may have a lower seizure threshold when ketamine is administered. Given the frequency with which ketamine is administered in EDs, emergency physicians should be aware that ketamine could potentially induce seizures.

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

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Postmortem Sperm Retrieval in the Emergency Department: A Case Report and Review of Available Guidelines

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Postmortem sperm retrieval (PMSR) requests and retrievals are increasing in the emergency department (ED) setting. Few EDs have protocols in place, and many emergency physicians (EP) lack the knowledge of how to proceed when such situations arise. We report the case of a 31-year-old male cardiac-arrest victim who expired in the ED, after which his wife requested PMSR. We review the guidelines, procedures, and issues of consent that arise with PMSR. EPs must be aware of their institution's policies and consultant availability should a request for PMSR arise. [Clin Pract Cases Emerg Med. 2019;3(4):405–408.]

INTRODUCTION

Postmortem sperm retrieval (PMSR) involves the retrieval of viable sperm from a recently deceased man for future use in assisted reproductive therapy (ART).[1] The first case of PMSR was reported in 1980, and since then there have been several published reports of PMSR and resultant pregnancies with viable children.[2,3,4] While both requests and retrievals in the United States have been increasing in frequency, controversy about the practice persists.[5,6,7]

Sudden unexpected accidental injury is the most common cause of death during the childbearing years and in situations in which PMSR is requested.[5,8] Few emergency departments (ED) have policies for PMSR, and many emergency physicians (EP) are unaware that PMSR is even a possibility, leaving them ill-prepared to respond to these requests in an informed and timely manner.

CASE REPORT

A 31-year-old male presented to an urban ED in pulseless cardiac arrest after his wife found him obtunded with sonorous respirations at home and began immediate cardiopulmonary resuscitation. He later expired after attempts at resuscitation were unsuccessful. Following a discussion with the decedent's wife regarding his death, she requested we perform PMSR. The EP, having previous knowledge about PMSR in the ED, responded appropriately to the wife's request by obtaining further history and discussing with multiple consultants.

A detailed medical history revealed that the deceased and his wife had been having difficulty conceiving a child and had begun evaluations and fertility planning at a local infertility clinic. The husband had already begun a workup for infertility with the urology group that was on call, and after consultation and further discussion it was confirmed that they follow the Weill Cornell Medicine (WCM), Department of Urology's guidelines for consideration of requests for PMSR.[9]

Concurrent consultation was ongoing with the infertility and reproduction specialist on call from the local academic medical center, who had been seeing the wife as a patient and whose department follows the guidelines published by the Ethics Committee of the American Society for Reproductive Medicine (ASRM) on postmortem retrieval and use of gametes or embryos.[10] Consistent with the WCM and ASRM

guidelines and in discussion with the urology and infertility specialists, this case was deemed appropriate for PMSR based on presumed consent of the deceased and using the family-centered approach as described by Bahm et al. in 2013.[11]

Additional consultation with hospital legal counsel confirmed the wife had the legal rights to the decedent's sperm. The medical examiner granted permission to perform the procedure to harvest the sperm from the body of the deceased as an autopsy was planned. Ice packs were placed in the groin and the body was transferred to the hospital morgue. The next morning the urologist consented the wife and the mother of the deceased for the harvesting procedure, and approximately 15 hours following the time of death sperm was successfully harvested and transported by an embryology technician to the academic medical center's cryopreservation laboratory without complication.

CPC-EM Capsule

What do we already know about this clinical entity?
Postmortem sperm retrieval (PMSR) requests are increasing in frequency in the emergency department (ED) setting, and few EDs and physicians are prepared should such a request arise.

What makes this presentation of disease reportable?
A descriptive case report on PMSR based on available guidelines has not been presented in the emergency medicine literature before.

What is the major learning point?
It is within the emergency physician's (EP) scope of practice to make an informed decision in response to a request for PMSR.

How might this improve emergency medicine practice?
It educates EPs on the issues related to PMSR in the ED, appropriate consultants to involve, and how to find available guidelines.

DISCUSSION

Requests for PMSR are rare but increasing in frequency.[5,6] As in this case, they tend to occur as a request from a spouse after a sudden and unexpected death in an otherwise healthy young person. EPs are more likely than other providers to receive a request for PMSR and must be prepared to respond to this request in an informed manner. The evaluation of the eligibility of a request for PMSR involves many issues that include legal, medical, logistical, ethical, and consent issues. Many of these issues can be made clearer when following an appropriate guideline. Currently there are no national guidelines, regulations, or restrictions related to PMSR.[9] This leaves institutional guidelines, medical society guidelines, and state laws, which vary considerably on the topic of PMSR, to guide decision-making.

Available Guidelines

The first guideline on PMSR was published in 2003 when Tash et al. described the effects of instituting an exclusionary institutional guideline, which had been developed at the WCM by a cross-functional panel of experts that included the urology, reproduction and infertility, law, psychology, and ethics departments.[12] Of the 22 families that sought PMSR after implementation of the guideline only four men were found to be candidates and underwent successful PMSR. Of these four cases, only one wife (of the deceased) attempted in vitro fertilization after an appropriate bereavement period, but no pregnancy occurred as a result.[12]

The number of institutions with guidelines now in place for PMSR has also continued to rise. Batzer et al. suggested 10 core elements for the development of a protocol for PMSR and recommended considering the interests of five key stakeholders to include the deceased, the requesting party the resultant child the physician, and society.[1]

In 2013 Bahm et al. contacted 40 institutions and evaluated nine full PMSR protocols.[11] They identified six major components present in most protocols and described two major approaches to policy structure: an institutional, limited-role approach, and a more family-centered approach.[11] In the limited-role approach, institutions require written consent before the death of the donor, specification of who can receive the sperm, and relinquishment of all responsibility after sperm retrieval. In the family-centered approach, the recipient of the sperm is allowed to use substituted judgment in the absence of written consent, but requires a period of psychological counseling before the sperm can be used in ART. The protocols evaluated varied along a spectrum between these two approaches.[11]

Legal Issues

Two major legal issues may arise in PMSR regarding the retrieval and/or the use of tissue in posthumous reproduction: 1) Does the wife have the legal right to the decedent's sperm for use in reproduction; and 2) are children of the deceased legally recognized as offspring?[10] More recent case law has raised the debate of whether or not treating tissue retrieved from PMSR similarly to organ donation tissue under the

Uniform Anatomical Gift Act of 2006 is appropriate.[13] Jurisdictions and case law vary in regard to the interpretation of these laws and the answers to these questions. In our case, legal consultation confirmed the wife's right to the decedent's sperm. However, our institution did not have a guideline in place. It was quickly confirmed that our urology group followed the WCM guidelines, while the reproductive and infertility group followed the ASRM guidelines.[9,10]

Ethical Issues

According to both the WCM and ASRM guidelines requests for PMSR should only be considered when requested by the wife of the deceased and she should be the only person for whom the sperm could be used for procreation. Requests from other family members or next-of-kin should not be considered.[9,10]

Issues regarding consent of the deceased, or lack thereof, are the most common reason not to proceed with a request for PMSR. The gold standard would be to have expressed written consent from the deceased to proceed with PMSR and ART in the case of his death. This is a rare occurrence, but does occur in instances of planned ART in which the consent forms specifically dictate the disposition of obtained gametes after the death of the individual from whom they were contributed.[10] The goal of presumed consent in PMSR is to ensure that the deceased had previously expressed a desire to conceive or was already attempting to conceive with his wife. Providers should consider "stated, written, or acted on wishes prior to death" when making decisions regarding PMSR.[9]

Logistical Issues

In cases that will require an autopsy, permission to perform PMSR should be obtained from the medical examiner. Additionally, the medical history of the deceased should confirm that there are no medical conditions that would have prevented sperm production and there should be no evidence of communicable disease, which may require additional testing.[9] Several procedural methods can be used to perform PMSR, with the least-invasive methods being the most preferred.[12] The WCM guidelines suggest retrieval be performed within 24 hours of death, while other sources cite up to 36 hours as appropriate to retrieve viable sperm.[14]

Transfer of the body to the morgue after expiration and harvesting of the sperm in less than 24 hours results in a high likelihood of viable sperm (86%, with a mean time to retrieval of 20.4 hours after death) and chances similar to ART of a resultant pregnancy.[14] Discussions with consultants can be done in a less urgent manner while the body is in the morgue, as in any other death in the ED, and will not use further nursing or departmental resources after this point. PMSR requires a cryopreservation facility to be available and nearby for immediate processing of the specimen.

The WCM and ASRM guidelines agree that PMSR programs are not ethically obligated to perform ART even though they may allow for PMSR to occur at their institution. They are also in agreement that PMSR programs should require a period of time for the normal bereavement process and psychological counseling to occur before the wife has the ability to use the sperm in ART.[9,10] The WCM guideline specifically requires a one-year quarantine period, which is used to evaluate the wife's family support system and understanding of the implications of raising a child in the absence of the biological father.[9] The observations from WCM indicate that the majority of women reconsider their decision and do not proceed with ART after they have had time to grieve and receive appropriate counseling.[9]

CONCLUSION

It is within the EPs scope of practice to make an informed decision in response to a request for PMSR. EPs must understand the issues surrounding consent, ethics, logistics, legality, and the various institutional practice guidelines and consultants available to them should such a request arise.

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

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Gestational Trophoblastic Disease-induced Thyroid Storm

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In the United States, gestational trophoblastic diseases (GTD), including molar pregnancies, occur in 121 out of 100,000 pregnancies.[1] Many patients with GTD may develop hyperthyroidism. GTD-induced thyroid storm is a rare but life-threatening complication of GTD.[2] Once patients are hemodynamically stable, the mainstay of definitive treatment is evacuation of the mole.[3] We present a case of molar pregnancy-induced thyroid storm presenting as vaginal bleeding, fever, and tachycardia. [Clin Pract Cases Emerg Med. 2019;3(4):409–412.]

INTRODUCTION

Vaginal bleeding at 6-16 weeks gestation is the most common presentation of hydatidiform moles.[1] Molar pregnancy-induced hyperthyroidism is a rare but potentially life-threatening condition. Presentations can range from subclinical hyperthyroidism to thyrotoxicosis and thyroid storm. We describe a case of a patient with thyroid storm induced by molar pregnancy.

CASE REPORT

A 39-year-old gravida 7 para 3033 female, with a history of three prior therapeutic abortions, presented to our emergency department (ED) with seven hours of heavy vaginal bleeding and crampy, lower abdominal pain. The patient reported her last menstrual period was four weeks prior to presentation. She also complained of one week of non-specific symptoms including intermittent nausea, vomiting, malaise, nasal congestion, and non-bloody diarrhea. She was found to be febrile to 102.2 degrees Fahrenheit and had a heart rate of 137 beats per minute (bpm). Her blood pressure was 124/86 millimeters of mercury, and oxygen saturation was 99% on room air.

The patient had a positive urine human chorionic gonadotropin (hCG) test. Ultrasound showed an empty uterus (Image), but her quantitative hCG was 117,495 milli-international units per milliliter (m[IU]/mL). The obstetrics (OB) team was consulted and evaluated the patient in the ED, and agreed that there was concern primarily for molar pregnancy versus septic abortion.

The patient was persistently tachycardic (to a maximum heart rate of 150 bpm), despite fever control with acetaminophen and intravenous fluid resuscitation. Electrocardiogram and cardiac monitor showed sinus tachycardia. Her white blood cell count was 16.1 thousands per cubic millimeter, and her hemoglobin was 11.3 grams per deciliter (g/dL). Thyroid stimulating hormone (TSH) was then sent and resulted as 0.009 m[IU]/mL. Total thyroxine (T4) was 14.7, micrograms (ug)/dL and free T4 was 1.82 nanograms (ng)/dL (Table), making the clinical picture concerning for thyroid storm.

A Burch-Wartofsky score, used for early detection of thyroid storm, was calculated at 55, which was highly

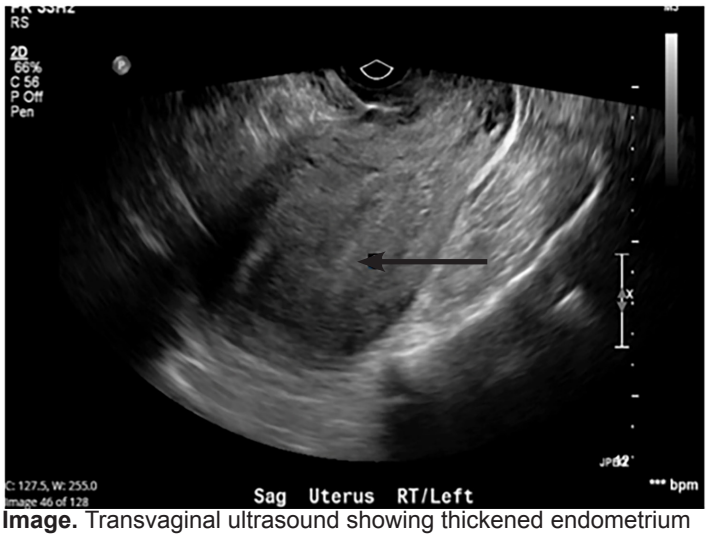

Image. Transvaginal ultrasound showing thickened endometrium (arrow) with no intrauterine pregnancy.

suggestive of thyroid storm. At this point, the intensive care unit (ICU) team was also consulted. The patient was treated with propranolol 1 milligram (mg) followed by an additional 2 mg dose, propylthiouracil (PTU) 500 mg and solumedrol 80 mg, with improvement in tachycardia to 110 bpm. She was then taken to the operating room with obstetrics (OB) for a dilation and evacuation. She was treated for possible septic abortion with cefoxitin 2 grams (g), doxycycline 100 mg and metronidazole 500 mg. After operative intervention, the patient was admitted to the ICU for continued care.

After the procedure, the patient's fever, leukocytosis and vaginal bleeding resolved. Postoperatively, her hemoglobin dropped to 7.8 g/dL and then remained stable. Endocrinology was consulted and thought the abnormal TSH was likely secondary to gestational hyperthyroidism, but could not rule out thyroid storm; they recommended checking thyroid stimulating immunoglobulin (TSI) to evaluate for Graves disease and discontinuation of steroids and PTU. The patient was transferred to the floor on postoperative day (POD) one and discharged home on POD two. TSI was negative. Prior to discharge, she received medroxyprogesterone acetate for contraception.

Pathology ultimately showed chorionic villi with marked hydropic changes consistent with complete hydatidiform mole and marked trophoblastic hyperplasia with cytologic atypia. She was recommended to have repeat beta hCGs checked weekly until negative for three weeks, and then monthly for six months, as well as thyroid function tests every four to six weeks until normal. She followed up with OB on POD eight and repeat hCG was 632 m[lU]/mL and was negative when she followed up four months later. She was lost to follow-up for endocrine and subsequent OB visits.

CPC-EM Capsule

What do we already know about this clinical entity?
Patients with gestational trophoblastic disease (GTD) may develop hyperthyroidism.

What makes this presentation of disease reportable?
GTD-induced thyroid storm is a rare but life-threatening complication of GTD.

What is the major learning point?
GTD-induced thyroid storm should be considered in any female patient of childbearing age with signs and symptoms of thyrotoxicosis.

How might this improve emergency medicine practice?
A high level of suspicion may help with faster diagnosis and initiation of treatment in this potentially life-threatening condition.

DISCUSSION

Gestational trophoblastic disease (GTD) results from the proliferation of placental trophoblast cells. In the United States, GTDs are associated with about 121 per 100,000 pregnancies.[1] GTD includes hydatidiform moles (complete and partial), invasive moles, choriocarcinomas, and placental site trophoblastic tumors. Hydatidiform moles occur when abnormal fertilization results in proliferative trophoblastic tissues and vesicular swelling of placental villi; these changes lead to a characteristic "grapelike" appearance.[4] Most complete moles are 46,XX due to the fertilization of an empty ovum by a duplicated haploid sperm or two sperm. Most partial moles are caused by the fertilization of a normal ovum by two sperm and result in a triploid (69,XXY) karyotype.

Vaginal bleeding at 6-16 weeks gestation is the most common presentation of complete hydatidiform moles, occurring in 80-90% of cases.[1] Other common symptoms include uterine enlargement greater than expected for gestational age and hyperemesis. Patients have elevated beta-hCG levels compared to normal pregnancies (often>100,000 m[IU]/mL) and absent fetal heart tones. Many cases initially present as a suspected missed or incomplete abortion. On ultrasound, a complete mole has a "snowstorm" granular appearance, although only 30-50% of hydatidiform moles are visualized by ultrasound.[3,5] Once the diagnosis is established, it is important to evaluate patients for medical complications, such as anemia, preeclampsia, and hyperthyroidism.

Hyperthyroidism has been reported in many patients with GTD. The beta subunit of β-hCG is structurally similar to TSH, allowing it to bind to the TSH receptor on thyroid follicular cells.[5] Of patients with hydatidiform mole 25-64% have been reported to have increased thyroid function, but only about 5% have clinical signs of hyperthyroidism.[6] It has been reported that β-hCG levels of greater than 200,000 m[IU]/ml sustained for several weeks are required to induce clinical hyperthyroidism.[6] It is estimated that for every 10,000 m[lU]/ml increase in serum hCG, TSH decreases by 0.1 mlU/ml and free T4 increases by 0.1 ng/dL.[5] In normal pregnancy, the elevated β-hCG concentration induces a weak hyperthyroid state. This is heightened in molar pregnancy due to higher concentrations of β-hCG. Additionally, the molecular variants of the β-hCG present in molar pregnancy have increased thyrotropic activity, likely due to its decreased sialic acid concentration.[7] The degree of increased thyroid hormone

Table. Select laboratory values on admission and discharge from hospital.

Test	Admission value	Discharge value	Reference range
WBC	16.1 K/mm^3	7.4 K/mm^3	4.0 – 11.0 K/mm^3
HGB	11.3 g/dL	7.8 g/dL	12.3 – 15.3 g/dL
TSH →	0.009 mIU/mL	0.006 mIU/mL	0.360-3.740 miU/mL
Total T4 →	14.7 ug/dL	N/A	4.5 - 10.9 ug/dL
Free T4	1.82 ng/dL	1.75 ng/dL	0.89 – 1.76 ng/dL
Total T3	108.10 ng/dL	N/A	60.00 – 181.00 ng/dL
Quantitative hCG →	117,495 mIU/mL	44,479 mIU/mL	<5 mIU/mL

→ Decreased TSH and elevated total T4 on admission; → Elevated quantitative hCG at admission, decreased by time of discharge.
WBC, white blood cell count; *HGB*, hemoglobin; *TSH*, thyroid stimulating hormone; *T4*, thyroxine; *T3*, triiodothyronine; *hCG*, human chorionic gonadotropin; *K*, thousand; *mm^3*, cubic millimeter; *g*, gram; *dL*, deciliter; *mIU*, milli-international unit; *mL*, millileter; *ug*, microgram; *N/A,* not applicable; *ng*, nanogram; *g*, gram.

varies based on the level of β-hCG, the amount of desialylation of the β-hCG, and the duration of the molar pregnancy.

Presentations can range from subclinical hyperthyroidism to thyrotoxicosis and thyroid storm. GTD-induced thyroid storm is a rare but life-threatening complication of GTD.[2] Thyroid storm is highly lethal, with a mortality of 10-30%, associated with tachycardia, fever, agitation, and altered mental status.[8,9] Patients are often profoundly tachycardic (>140 bpm), and have limited response to calcium-channel blockers, beta blockers, and intravenous fluids. A high level of suspicion and early diagnosis is crucial to prevent the complications of thyroid storm such as stroke, dysrhythmia, heart failure, rhabdomyolysis, liver dysfunction, and death.[4]

The emergent management of thyroid storm involves decreasing hormone synthesis and release, blocking the action of thyroid hormone, reversing systemic decompensation, and removing the precipitating event. PTU or methimazole can be used to block hormone synthesis, and iodine or lithium can decrease hormone release. Iodine therapy should be initiated at least one hour after PTU to avoid reflex thyroid hormone release.[6] In addition, PTU, beta blockers, and glucocorticoids all can help decrease peripheral conversion of T4 to T3. Beta blockers also help decrease the peripheral effects of thyroid hormone.[3,9]

Once patients are hemodynamically stable, the mainstay of definitive treatment is evacuation of the mole.[3] OB evaluation should be obtained early in these patients' clinical course. Suction and curettage is usually the preferred method of evacuation. Alternatively, hysterectomy is sometimes performed in patients who do not desire further pregnancy.[1] The evacuation of the mole results in rapid reduction in thyroid hormone levels.[6] Post-evacuation follow-up with serial quantitative β-hCG measurements is crucial to evaluate for persistent molar tissue or development of choriocarcinoma. These complications develop in about 15-20% of patients with complete mole and 1-5% of patients with partial mole.[1]

CONCLUSION

GTD-induced thyroid storm is a rare but potentially life-threatening condition. A high level of suspicion and early diagnosis in the ED is critical to obtain appropriate treatment. This diagnosis should be considered in any female patient of childbearing age with signs and symptoms of thyrotoxicosis.

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

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Atypical Presentation of Median Arcuate Ligament Syndrome in the Emergency Department

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Celiac artery compression syndrome, also called median arcuate ligament syndrome (MALS), is a rare condition in which the diaphragmatic crura compresses the celiac axis. This results in a constellation of primarily gastrointestinal (GI) symptoms including nausea, vomiting, postprandial abdominal pain, and weight loss. It is typically a diagnosis of exclusion and may be detected via several imaging techniques including ultrasound and computed tomography angiography. We present an atypical case of MALS detected in the emergency department (ED). We review the symptomatology, diagnostic workup, and treatment options here, as well as discuss implications concerning revisits to the ED for recurrent GI symptoms. [Clin Pract Cases Emerg Med. 2019;3(4):413–416.]

INTRODUCTION

Abdominal pain is one of the most common reasons patients present to the emergency department (ED) and carries a myriad of possible underlying pathologies. While it is a relatively benign condition, median arcuate ligament syndrome (MALS) may mimic life-threatening causes of abdominal pain. This case describes this patient's unique presentation, reviews current diagnostic and treatment modalities for this condition, and discusses its place in the broader context of revisits to the ED.

CASE REPORT

A 59-year-old male presented to the ED with complaints of crampy, diffuse abdominal pain with associated nausea and watery diarrhea ongoing for the prior four days. He had been evaluated in the ED two days prior to presentation with similar complaints, at which time his workup was significant only for diverticulosis on computed tomography (CT) of the abdomen and pelvis with intravenous contrast. He had been discharged home in stable condition with oral dicyclomine 10 milligrams (mg) and ibuprofen 600 mg. The patient followed up with his primary care physician the next day, at which time he was prescribed hydrocodone/acetaminophen 5/325 mg for pain control. Due to progressively worsening abdominal pain not relieved with these medications, he returned the ED for further evaluation.

The patient's past medical history was significant for Barrett's esophagus, chronic kidney disease, diverticulosis, gastroesophageal reflux disease, gout, hyperlipidemia, and hypertension. He had undergone esophagogastroduodenoscopy (EGD) for GI complaints in the past. He was compliant with his home amlodipine, atorvastatin, lisinopril, and omeprazole. Social history was significant for a 45-pack per year smoking history and weekly, moderate alcohol consumption.

Upon arrival to the ED, the patient's vitals revealed a temperature of 97.9 degrees Fahrenheit, pulse 71 beats per minute, respiratory rate 20 breaths per minute, blood pressure 150/89 millimeters of mercury (mmHg), and pulse oximetry 98% on room air. Physical examination revealed a nontoxic patient, mildly uncomfortable appearing, but in no acute distress. He did have mild, diffuse abdominal tenderness to palpation without distention, rebound, or abdominal bruit. The remainder of his physical examination was unremarkable. Initial diagnostic workup including complete blood count, complete metabolic panel, lipase, urinalysis, urine drug screen, lactate, electrocardiogram, troponin I, and chest radiograph was unremarkable.

Upon reevaluation, the patient's symptoms had somewhat improved, but he then reported some tenderness to palpation in the midline of his back. A CT angiogram

of the chest and abdomen with three-dimensional reconstructions was obtained and revealed stenosis at the origin of the celiac artery with characteristic "hooked" appearance, raising concern for MALS (Images 1 and 2). The patient was then reassessed and reported spontaneous relief of his symptoms. He was informed of his imaging findings and discharged home in stable condition with referral to general surgery to explore possible treatment options. He subsequently followed up with general surgery and was referred to the interventional radiology (IR) service at a tertiary care center.

Prior to recommending IR angioplasty and stenting, the patient underwent EGD, colonoscopy, and cholescintigraphy, which were normal. The patient then had an abnormal stress test as part of cardiac clearance for surgery, leading to subsequent cardiac catheterization and percutaneous coronary intervention. At follow-up with his primary care physician 10 months after the initial ED visit, the patient was still exhibiting similar GI symptoms and was awaiting cardiology clearance to proceed with IR angioplasty and stenting.

CPC-EM Capsule

What do we already know about this clinical entity?
Median arcuate ligament syndrome (MALS) is a rare condition that typically produces chronic gastrointestinal (GI) symptoms.

What makes this presentation of disease reportable?
This case was detected via imaging in the emergency department (ED) rather than the outpatient setting. Additionally, this patient did not fit the typical demographics for this condition.

What is the major learning point?
MALS may be detected in the ED. Patients with imaging suspicious for this condition will require GI and surgical referral for further diagnostic and therapeutic management.

How might this improve emergency medicine practice?
Readers will include MALS in their differential for patients with multiple ED visits for GI complaints, and will know appropriate disposition if this condition is suspected.

DISCUSSION

MALS, also called celiac artery compression syndrome or Dunbar syndrome, is a rare condition in which low-lying fibers from diaphragmatic crura compress the celiac artery or ganglion.[1] Typically, the ligament lies anterior to the aortic hiatus, uniting fibers from either side of the diaphragmatic crura superior to the celiac axis. However, in up to 24% of the population, the ligament crosses anterior to the celiac artery and in some individuals may compromise blood flow, leading to concurrent symptomatology.[2] The condition was first described in 1963 by Harjola in a case report in which a patient presented with epigastric bruit and postprandial abdominal pain and was found to have narrowing at the celiac axis secondary to local fibrotic tissue.[3] Compression or stenosis in this area is thought to cause postprandial foregut ischemia, resulting in a constellation of GI symptoms including postprandial epigastric pain, nausea, vomiting, diarrhea, bloating, and unintentional weight loss secondary to food aversion.[1-4] Physical examination may also reveal an audible abdominal bruit best heard over the epigastrium, which is detected in up to 83% of cases.[5,6]

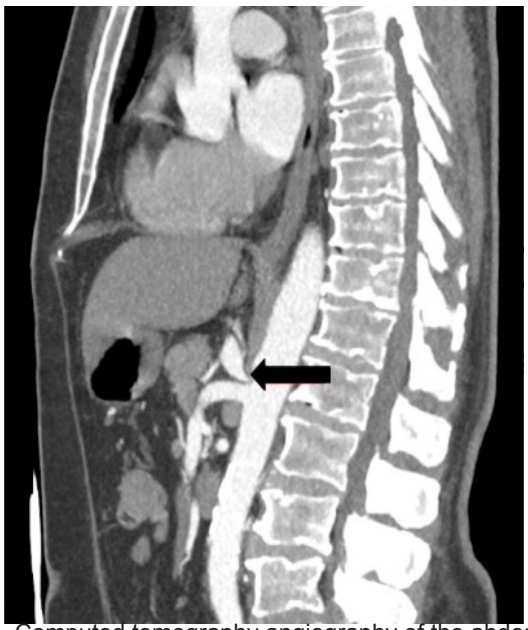

Image 1. Computed tomography angiography of the abdomen and pelvis sagittal view demonstrating characteristic "hooked" appearance (arrow) of celiac artery, origin off the abdominal aorta.

While celiac axis compression and subsequent foregut ischemia is the most widely accepted understanding of MALS pathophysiology, this is debatable as multiple studies have suggested the symptoms are secondary to irritation of the celiac ganglion as opposed to intestinal ischemia.[7,8] A significant portion of the general population may have some

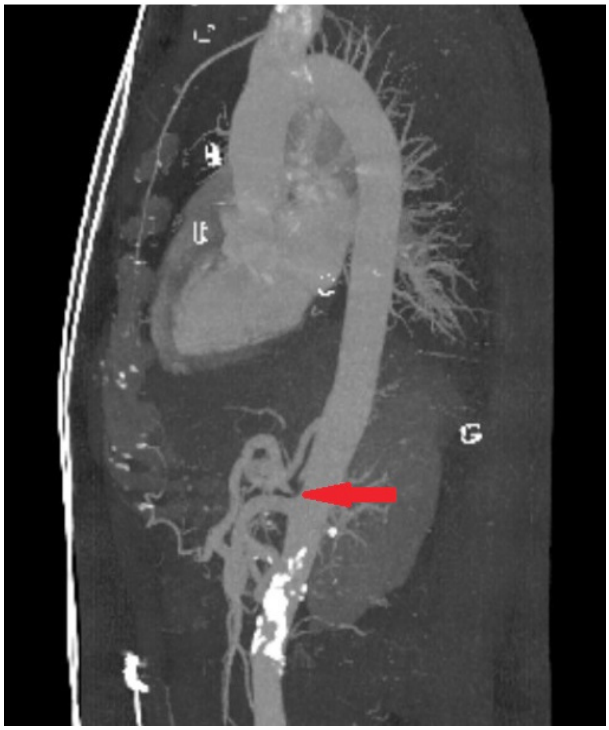

Image 2. Three-dimensional reconstruction of computed tomography angiography of the abdomen and pelvis sagittal view demonstrating characteristic "hooked" appearance of celiac artery (arrow), origin off the abdominal aorta.

degree of median arcuate ligament compression without symptomatology, with estimates varying from 3.42% in a study of asymptomatic patients with compression noted on CT to 10-24% in a comprehensive literature review of MALS cases.[4,9]

Conversely, the incidence of MALS is estimated at approximately two per every 100,000 patients.[10] The majority of MALS patients are relatively young women between the ages of 20-50 years old with thin body habitus who have been extensively worked up for various intra-abdominal pathology.[1,6] As MALS is a diagnosis of exclusion, initial gastroenterology referral may be required if patients have not had prior extensive workup to investigate other possible causes of functional abdominal pain. Once other causes have been ruled out, the diagnosis can be confirmed with imaging including duplex ultrasound, CT angiography, and magnetic resonance angiography revealing compression or stenosis of the celiac artery.[6,8] Percutaneous diagnostic celiac ganglion blockade, in which lidocaine is injected into splanchnic nerves feeding into the celiac plexus, can also be performed by general surgery or IR to predict symptomatic improvement with surgical intervention.[10-12]

Treatment historically consists of surgical techniques, although due to the rarity of the syndrome there is no single accepted treatment algorithm and options should be considered in the context of an individual patient's age and severity of symptoms. General surgeons may perform minimally invasive techniques such as laparoscopic or robotic median arcuate ligament release, while vascular surgeons may perform percutaneous transluminal angioplasty, primary reanastomosis, or celiac artery bypass grafting in refractory cases.[6,13-17] IR may also perform angioplasty with stenting.[14,18] The success rate of immediate postoperative symptom relief has been reported at up to 85%, with sustained symptom relief for years following surgery reported between 53-80% with surgical decompression alone and over 80% relief with both decompression and other vascular intervention.[19,20] Patients should thus be counseled that multiple interventions may be necessary for the best chance of long-term symptom relief.

The presentation of MALS discussed in this case report is unusual in that it was in a moderately obese, slightly older male. His physical examination findings were nonspecific, and his history lacked the characteristic postprandial food aversion seen in many MALS cases. Furthermore, this condition is primarily detected in the outpatient setting following years of chronic GI symptoms as opposed to acutely in the ED. While MALS is not life-threatening, it may mimic the symptoms of mesenteric ischemia and may initially be worked up similarly in the ED, as was done in this case.[21] Therefore, this diagnosis should be considered in any patients presenting to the ED with recurrent GI complaints with previously negative workups. If MALS is suspected based on imaging obtained in the ED, providers should refer patients to gastroenterology for further workup and exclusion of other causes of chronic abdominal pain, with general or vascular surgery referrals if the patient records demonstrate a history of extensive workup for GI symptoms.

CONCLUSION

Median arcuate ligament syndrome is a rare cause of recurrent abdominal pain and is typically a diagnosis of exclusion. While a significant portion of the United States population may have some underlying compression of the celiac axis, a much smaller percentage of these individuals may develop symptoms of MALS. The above case brings awareness of this uncommon disease to ED professionals, and demonstrates that it may occur in patients who do not fit its typical epidemiological demographics. Its presentation may mimic that of mesenteric ischemia and should be considered in any patient presenting to the ED multiple times for abdominal pain with previously unremarkable workups. Early recognition of atypical vascular causes of abdominal pain with proper outpatient referral may decrease costly ED revisits and help patients with chronic GI complaints ultimately find lasting relief.

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

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A Rare Case of Hemorrhagic Shock: Morel-Lavallée Lesion

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Hemorrhage is a major cause of death among trauma patients. Controlling the bleeding is essential but can be difficult when the source of bleeding remains unidentified. We present a 67-year-old healthy male with a hypovolemic shock after a suicide attempt by jumping from a height. Apart from a bilateral pneumothorax with multiple rib fractures, a femur fracture and spine fractures, computer tomography (CT) revealed a closed, degloving injury of the back, also known as a Morel-Lavallée lesion. Hemodynamic instability due to hemorrhage caused by a Morel-Lavallée lesion in the lumbar region is very rare and easily overlooked. This case demonstrates the importance of clinical signs of Morel-Lavallée, and illustrates the need for total body CTs to exclude other locations of bleeding and to detect contrast extravasation. This report also discusses the possible treatment options for Morel-Lavallée lesions. [Clin Pract Cases Emerg Med. 2019;3(4):417–420.]

INTRODUCTION

Hemorrhage is the most common cause of shock in trauma patients and a major cause of death among them.[1] Failure to provide the correct treatment to control the bleeding contributes to preventable trauma death. A structural and uniform initial assessment according to Advanced Trauma Life Support (ATLS) guidelines is essential for early identification of bleeding and hemostasis. A timely log roll could be warranted, especially when the focus of hemorrhage is unclear. Massive soft tissue injury is an underestimated cause of hypovolemic shock. We present a trauma patient with a rare case of hemorrhagic shock due to a Morel-Lavallée lesion in the lumbar region. We also discuss the management of this injury.

CASE REPORT

A 67-year-old male, with no known health issues prior to presentation at the emergency department (ED), arrived at the ED via ambulance after a suicide attempt by jumping from a height. The patient was assessed according to the ATLS guidelines. On arrival, his breathing was spontaneous and he was able to speak.

The patient had the following vital signs: respiratory rate of 18 breaths per minute, 100% oxygen saturation on 15-liter (L) non-rebreathing mask, heart rate of 108 beats per minute, blood pressure: 86/40 millimeters of mercury, and a temperature of 36.5° Celsius. The Glasgow Coma Scale (GCS) was 14 (motor: four, verbal: six, eyes: four) and pupils were symmetric and reactive to light. On physical examination his lungs were bilaterally clear to auscultation. The abdomen was soft with no abdominal tenderness and the pelvis was clinically stable. The left femur appeared shortened and rotated. His neurological exam did not demonstrate any localized neurological deficit. Motor strength was 5/5 in the upper extremities and -/5 in the lower extremities due to a femur fracture. Sensory testing was normal and reflexes were symmetrical low with a normal Babinski reflex.

A back examination revealed a large, boggy swelling on the lower back. The patient received two L of warmed saline and four units of packed red blood cells after which the circulatory status normalized. Additional radiological imaging was performed. Chest radiography showed multiple rib fractures. A focused assessment sonography for trauma (FAST) showed no free intraperitoneal fluid. No pericardial effusion was detected.

As we did not yet have a clear explanation for the ongoing shock, computer tomography (CT) was performed. CT imaging included the head, cervical-, thoracic- and lumbar spines, chest, abdomen, pelvis and femora. This CT showed unstable fractures of the first to the fourth lumbar vertebrae, bilateral multiple rib fractures with a small bilateral pneumothorax, a left proximal femur fracture, and an extensive Morel-Lavallée lesion in the lumbar region with minor contrast extravasation (Image 1). There were no signs of a spinal cord lesion.

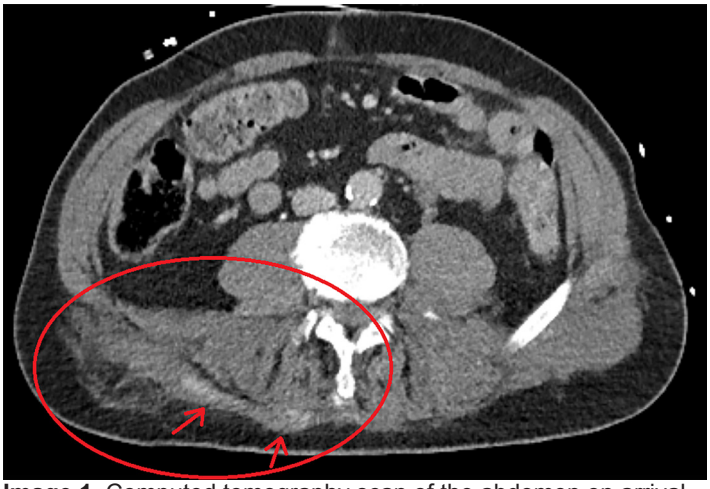

Image 1. Computed tomography scan of the abdomen on arrival at the emergency department showing the early signs of a Morel-Lavallée lesion in the lumbar region. The oval shows the main lesion. The arrows indicate the contrast extravasation.

CPC-EM Capsule

What do we already know about this clinical entity?
A Morel-Lavallée lesion can occur in the lumbar region but hemodynamic instability is rare. There are different treatment modalities such as surgical debridement or percutaneous drainage.

What makes this presentation of disease reportable?
In this case the Morel-Lavallée lesion in the lumbar region caused significant hemodynamic instability and the total body computed tomography scan showed significant contrast extravasation.

What is the major learning point?
A Morel-Lavallée lesion can cause significant hemodynamic instability and a full body examination is important. Pressure of the patients own body weight can be a sufficient treatment.

How might this improve emergency medicine practice?
Compression of the patients own body weight can be a considered treatment in patients with a Morel-Lavallée lesion in the lumbar region to regain hemodynamic stability.

The patient was transferred to the intensive care unit for further resuscitation. Temporary skeletal traction was applied for the proximal femoral fracture. Conservative treatment of the Morel-Lavallée lesion was initiated, allowing for spontaneous tamponade of the bleeding due to compression from the patient's own body weight. In-line spinal immobilization was maintained.

A CT angiography was repeated eight hours after admission because of an ongoing need for inotropic support. A persistent, active bleeding in the Morel-Lavallée lesion was observed (Image 2). Because of the accompanying unstable fractures of the lumbar vertebrae, surgical treatment or coiling was deemed high risk, and therefore conservative treatment was continued. During the following 24 hours the hemodynamic status normalized and inotropic support was discontinued. After one week, a successful spondylodesis of the lumbar spine as well as an osteosynthesis of the proximal femur fracture was performed. The patient recovered well without any complications. He was discharged to a rehabilitation center on day 64.

DISCUSSION

A Morel-Lavallée lesion is a closed, degloving injury first described by the French surgeon Victor Auguste Francois in 1863.[2] It is also known as a post-traumatic, soft tissue cyst or a chronic expanding hematoma. In most cases, this lesion is the result of severe injury due to high-energy trauma. It is also reported in contact sports and in postoperative complications.[3,4] Severe shearing forces during trauma cause separation between the loose skin, subcutaneous fat, and the relatively immobile underlying deep fascia. This results in hemolymphatic fluid collection originating from the disrupted blood vessels and lymphatic vessels. Morel-Lavallée lesions can involve only soft tissue or they can occur in combination with fractures. Apart from local pain and a soft boggy swelling, hypoesthesia in the affected region is common due to damage to cutaneous nerve branches.[2,3]

Little is known about the epidemiology of Morel-Lavallée lesions. Vanhegan et al. reviewed the location of 204 Morel-Lavallée lesions in 29 papers. The lesions are most commonly found at the following locations: greater trochanter or hip–30.4%; thigh–20.1%; pelvis–18.6%; knee–15.7%; gluteal–6.4%; lumbosacral–3.4%; abdominal wall–1.5%; calf or lower leg–1.5%; head–0.5%; and unspecified–2.0%. Only a few published case reports describe a Morel-Lavallée lesion in the lumbar region, and they are mainly chronic lesions in non-shock patients.[3] Only three cases of hemorrhagic shock have been described.[5,6,7]

Morel-Lavallée lesions often evolve over a few hours to days after an injury. However, up to 30% of Morel-Lavallée lesions develop over months following the initial injury, of which

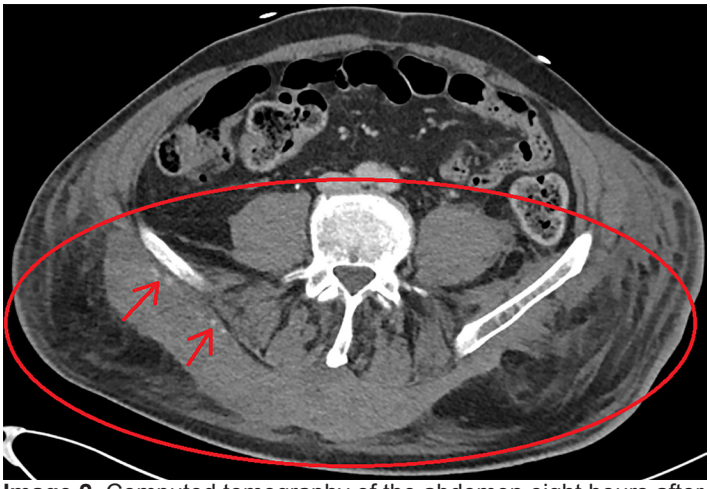

Image 2. Computed tomography of the abdomen eight hours after admission with a Morel-Lavallée lesion in the lumbar region. A mass is visible on the dorsal side of the pelvis. The oval shows the main lesion. The arrows indicate the contrast extravasation.

30% remain undiagnosed.[8,9] These lesions are often missed or mistaken for tumors (sarcoma), soft tissue hematomas, fat necrosis, pseudo-lipoma, abscesses or a bursitis. This often causes a delay in treatment.

Several diagnostic imaging modalities can help to diagnose a Morel-Lavallée lesion. These include CT and magnetic resonance imaging (MRI).[9,10] Ultrasound has also proved to be effective for diagnosing, monitoring and follow-up, but due to the stages of internal blood product degeneration (seroma, subacute hematoma, and chronic organized hematoma), their presentation can vary over time and these lesions can be difficult to detect.[11]

A CT is usually easier to perform in the acute situation. Early lesions typically demonstrate the CT characteristics associated with hematomas. Only a third of the lesions show active contrast extravasation at the time of the initial scan. McKenzie et al. stated that the possibility of a closed, soft tissue degloving injury should be raised in the clinical setting of a high-energy trauma in combination with a fluid collection in the subcutaneous tissues overlying the deep fascia, with sparing of the overlying skin and internal fat globules on CTs. The clinical setting is highly important while examining the CT, otherwise a Morel-Lavallée lesion can often go unrecognized.[12] Despite this recent research by McKenzie et al., who considered CT imaging to be a reliable detection method, MRI is still the gold standard in diagnosing Morel-Lavallée lesion at its different stages.[10] In its early stages, the hemolymphatic fluid appears hyperintense on T2 images. At a later stage, hemoglobin in the hemolymphatic fluid transforms to methemoglobin, which causes an increased intensity on T1 images. In the final stage, a surrounding capsule develops that will be visible on MRI images.[13, 14]

Traditionally, surgical open debridement has been the preferred treatment of a Morel-Lavallée lesion. Over time, less invasive methods with better aesthetic outcomes have been developed, including (imaging-guided) percutaneous drainage, compressive bandaging and injection of sclerosing agents such as doxycycline, fibrin glue, and alcohol.[15,16] Sclerodesis is the favored method in patients without fractures. When the lesion is older and encapsulated, surgical excision is more commonly used.

Even though a Morel-Lavallée lesion is a closed injury, potential complications include soft tissue or deep bone infection, wound dehiscence, and skin necrosis. Separation of the vasculature due to the shearing mechanism can result in skin necrosis. This may also be a result of the mass effect of the fluid collection, which further compresses the supplying vascular plexus, causing pressure-related ischemia.[8]

To date, only three cases of a patient in shock due to a Morel-Lavallée lesion have been reported. Mao et al. reported a lesion located at the upper thigh, which was treated by surgical drainage after a few days.[7] Hefny et al. reported a case of an extensive hematoma at the flank without contrast extravasation on the CT.[5] In this case, percutaneous suction drainage was performed. Yumoto et al. reported a lesion of the lower back. An initial CT detected contrast extravasation, and transcatheter arterial embolization was successfully performed.[6]

In our case the Morel-Lavallée lesion was situated at an uncommon location. The lesion developed in a very short time and it caused hemodynamic instability. Per ATLS guidelines there was, at minimum, class III shock based on tachycardia, hypotension, and confusion.[17] In trauma patients, the most likely cause of shock is bleeding unless there is an obvious alternative cause. In addition to the swelling on the back, the patient had a femur fracture, and multiple rib and spinal fractures, which could also have caused bleeding. In light of the fact that we applied traction on the femur, the ongoing shock was, in our opinion, the result of the Morel-Lavallée lesion on the patient's back. There was no sign of cardiogenic shock or cardiac tamponade and there was no tension pneumothorax. Although only three cases have been reported, Morel-Lavallée lesions can contribute to or be responsible for hemorrhagic shock in trauma.

The traditional screening in trauma patients involving chest and pelvic radiographs and FAST cannot detect all bleeding sites. Physicians should check for occult bleeding in patients with hemorrhagic shock with an unknown focus. In hemodynamically unstable patients, contrast-enhanced CT may be needed to find the source of bleeding. However, particularly in patients with clinical signs of a Morel-Lavallée lesion, a contrast-enhanced CT is advised to exclude other foci of bleeding. In our case, the vertebral fractures made it difficult to use one of the recommended treatments as described above. In the two similar cases of Hefny et al. and Yumoto et al. they chose to drain the lesion. We believe that external compression is a suitable alternative treatment, especially if the lesion is located in the lumbar region and accompanied by unstable fractures. Compression can be achieved by using the patient's

own body weight. This seems to be a practical approach that is not always considered.

CONCLUSION

Hemodynamic instability due to hemorrhage caused by a Morel-Lavallée lesion in the lumbar region is very rare and easily overlooked. In hemodynamically unstable patients a contrast-enhanced CT is advised to detect the bleeding focus. When a Morel-Lavallée lesion is situated in the lumbar region, conservative treatment by compression using the patient's own body weight can be an appropriate therapy.

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

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Colpocephaly Diagnosed in a Neurologically Normal Adult in the Emergency Department

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Colpocephaly is a form of congenital ventriculomegaly characterized by enlarged occipital horns of the lateral ventricles with associated neurologic abnormalities. The diagnosis of colpocephaly is typically made in infancy. Its diagnosis in adulthood without associated clinical symptoms is exceptionally rare. We report a case of colpocephaly diagnosed incidentally in an adult without neurologic abnormalities in the emergency department. To our knowledge, this is only the ninth reported case in an asymptomatic adult and the first to be described in the emergency medicine literature. [Clin Pract Cases Emerg Med. 2019;3(4):421–424.]

INTRODUCTION

Colpocephaly is a rare form of congenital ventriculomegaly often associated with partial or complete agenesis of the corpus callosum. Diagnosis is typically made in infancy due to associated neurological and neurodevelopmental disorders.[1,2] Initial discovery in adulthood is exceedingly rare.[3-9] When identified incidentally in adults, colpocephaly may be misdiagnosed as hydrocephalus.[4,7,9] We report a case of colpocephaly in an adult of normal neurological development discovered in the emergency department (ED).

CASE REPORT

A 29-year-old male with no pertinent past medical history presented to our ED with two weeks of intermittent headaches. His headache was described as throbbing in character, localized to the bitemporal region, non-radiating, and non-positional. The headache occurred daily, lasting a few minutes to hours, with no particular exacerbating factors. His symptoms improved with acetaminophen, which he used sparingly. He reported no associated vomiting, gait abnormalities, vision changes, confusion, urinary changes, or other neurologic abnormalities. He had been treated at four different EDs in the two weeks prior to presentation for the headaches, but no imaging studies had been performed. The patient had no psychiatric history. His highest level of education was a high school diploma, and he was unemployed.

On arrival, the patient was afebrile with pulse, blood pressure, and respiratory rate all within the normal range. Physical examination revealed an anxious male who was alert, oriented, and in no acute distress. His head was normocephalic with no evidence of trauma. His pupils were equal, round, and reactive to light. His neurological examination did not reveal any cranial nerve deficits, speech abnormalities, muscle weakness, or loss of sensation. His reflexes were intact and symmetrical. His coordination was normal. His gait was stable with balanced cadence, and he exhibited a negative Romberg test. His visual acuity was 20/20 in both eyes. The remainder of the examination was unremarkable.

Laboratory values of complete blood count and complete metabolic panel were unremarkable. The serum carboxyhemoglobin level was within the normal range. Due to the patient's headache not being fully consistent with a primary headache and his multiple visits to the ED without

a history of imaging, computed tomography (CT) of the head was ordered to assess for a possible anatomic cause of his symptoms. The CT was notable for marked enlargement of the occipital horns of the lateral ventricles with agenesis of the corpus callosum, consistent with colpocephaly (Images 1 and 2).

The patient was evaluated by the neurology service in the ED. It was their opinion that his headaches were primary in nature and not associated with the incidental finding of colpocephaly. His headache resolved after receiving 10 milligrams (mg) of intravenous metoclopramide and 50 mg of oral diphenhydramine, and he was discharged home with neurology follow-up. The patient returned to the ED one month later for an unrelated complaint and did not report a headache at that time.

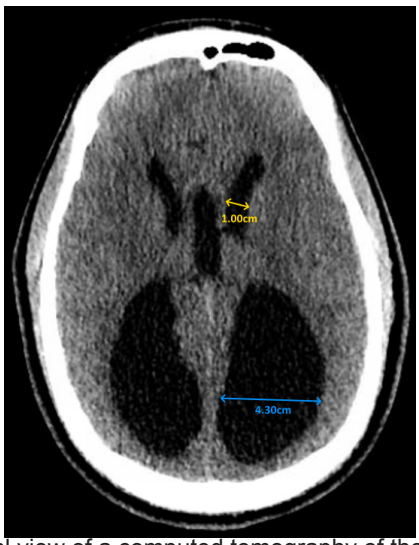

Image 1. Axial view of a computed tomography of the head demonstrating ventriculomegaly, consistent with colpocephaly. Lines compare the maximal width of the occipital horns (blue line) to that of the anterior horns (yellow line) of the lateral ventricle, with an occipital-to-anterior horn ratio of 4.3.

CPC-EM Capsule

What do we already know about this clinical entity?
Colpocephaly is a congenital form of ventriculomegaly. Diagnosis is typically made in infancy due to associated neurologic abnormalities.

What makes this presentation of disease reportable?
This is only the ninth reported case of colpocephaly diagnosed in an asymtomatic adult and the first to be described in the emergency medicine literature.

What is the major learning point?
While exceptionally rare, colpocephaly may be present in asymptomatic adults. It may be misdiagnosed in adults as normal pressure hydrocephalus.

How might this improve emergency medicine practice?
Knowledge of the clinical and radiographic differences between colpocephaly and normal pressure hydrocephalus will help avoid unnecessary diagnostic and therapeutic procedures.

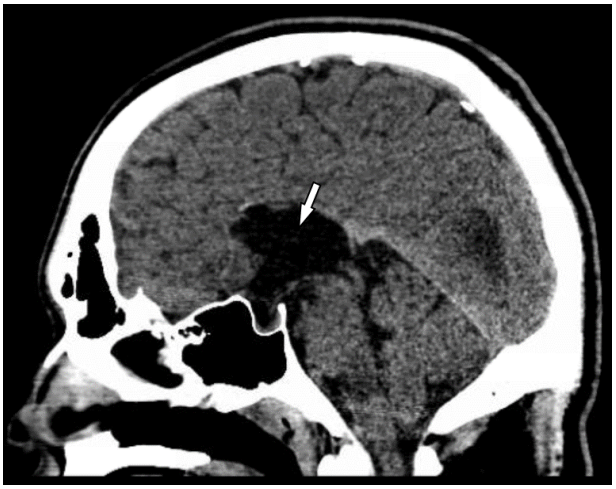

Image 2. Sagittal view of computed tomography of the head demonstrating complete agenesis of the corpus callosum (arrow).

DISCUSSION

First described by Benda in 1940, colpocephaly is a rare congenital brain malformation in which the occipital horns are disproportionately larger than the anterior horns of the lateral ventricles.[10] Colpocephaly can be associated with partial or complete agenesis of the corpus callosum, Chiari malformations, lissencephaly, and microcephaly.[9] The abnormal ventricular enlargement in colpocephaly is believed to be secondary to the developmental arrest

Table 1. Previous reported cases of colpocephaly diagnosed incidentally in adulthood.

Author	Year	Patient	Reason for imaging
Wunderlich G, et al.[6]	1996	60-year-old female	New onset partial complex seizures
Cheong J, et al.[7]	2012	67-year-old female	Four months of headache and dizziness - ultimately diagnosed with meningioma
Esenwa C, et al.[3]	2013	60-year-old female	Headache after minor head trauma
Ciurea R, et al.[9]	2014	28-year-old female	Longstanding, intermittent headaches and vertigo
Brescian N, et al.[8]	2014	88-year-old male	New onset left hand apraxia
Nasrat T, et al.[4]	2014	66-year-old female	One month of declining mental status - ultimately diagnosed with paraspinal abscess
Bartolome E, et al.[5]	2016	67-year-old female	Syncopal episode
		60-year-old female	Confusion with fever - ultimately diagnosed with an upper respiratory infection

of white matter formation that occurs during fetal development.[7] Various etiologies have been proposed, including chromosomal abnormalities, intrauterine infection, perinatal anoxic-ischemic encephalopathy, intrauterine growth retardation, and maternal toxin exposure.[1,3]

Colpocephaly is typically discovered in infancy due to associated intellectual disability, seizures, motor abnormalities, or visual abnormalities.[1,2] Discovery in adulthood is remarkably uncommon and has only been reported eight times previously (Table 1).[3-9] Colpocephaly can be identified radiographically by measuring the maximal width of the anterior and occipital horns of the lateral ventricles. An occipital-to-anterior horn ratio of greater than 3 is highly specific for colpocephaly, although it has relatively low sensitivity.[3,11]

The identification of colpocephaly in adulthood is a phenomenon that has only recently been described. Colpocephaly discovered in adulthood may be misdiagnosed as normal pressure hydrocephalus, a much more common cause of ventriculomegaly in adults.[3,5] Knowledge of the clinical and radiographic differences between these two conditions is needed to avoid unnecessary diagnostic and therapeutic procedures (Table 2). Colpocephaly discovered incidentally in asymptomatic adults requires no specific treatment.

CONCLUSION

Colpocephaly discovered in asymptomatic adults is exceedingly rare. It may be misdiagnosed as normal pressure hydrocephalus in the ED. It is important to differentiate between these two conditions to avoid unnecessary interventions.

Table 2. Clinical and radiographic characteristics of colpocephaly and normal pressure hydrocephalus.

	Colpocephaly	Normal pressure hydrocephaly[3,12]
Clinical characteristics	Typically diagnosed in infancy due to associated neurological abnormalities	Typically diagnosed after age 50 years
	Diagnosis in asymptomatic adults is exceptionally rare	Symptoms include varying degrees of the classic triad of gait disturbance, urinary incontinence, and dementia
Radiographic characteristics	Disproportionate dilation of the occipital horns of the lateral ventricles, often associated with full or partial agenesis of the corpus callosum	Ventriculomegaly marked by dilation of the anterior and occipital horns of the lateral ventricles
Treatment	No treatment is indicated when diagnosed in asymptomatic adults	CSF shunting procedures lead to symptom improvement in approximately 60% of cases

CSF, cerebral spinal fluid.

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

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Pseudogout Diagnosed By Point-of-care Ultrasound

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A 71-year-old male presented to the emergency department (ED) for worsening right knee pain for the prior 3-4 weeks. Point-of-care ultrasound (POCUS) of the right knee showed a pseudo-double contour sign. Subsequent ultrasound-guided arthrocentesis of the knee joint was performed, and fluid studies showed the presence of calcium pyrophosphate crystals, which was consistent with pseudogout. Ultrasound for detection of calcium pyrophosphate crystals in pseudogout and chondrocalcinosis has sensitivity of 86.7% and specificity of 96.4% making POCUS a valuable tool for diagnosing crystalline-induced arthropathy in the ED. [Clin Pract Cases Emerg Med. 2019;3(4):425–427.]

CASE PRESENTATION

A 71-year-old male presented to the emergency department with worsening right knee pain and swelling for the prior 3-4 weeks. Past medical history was significant for gout treated with colchicine. The patient was afebrile. Physical exam demonstrated a swollen right knee, mild erythema, and limited range of motion. Point-of-care ultrasound (POCUS) of the right knee showed the findings depicted in Images 1 and 2**.** A POCUS-guided arthrocentesis was performed and confirmed the diagnosis suggested by POCUS.

DISCUSSION

In this case, POCUS suggested the diagnosis of pseudogout by demonstrating the pseudo-double contour sign. The joint aspirate contained calcium pyrophosphate crystals, 161 white blood cells per cubic millimeter, no organisms on gram stain,

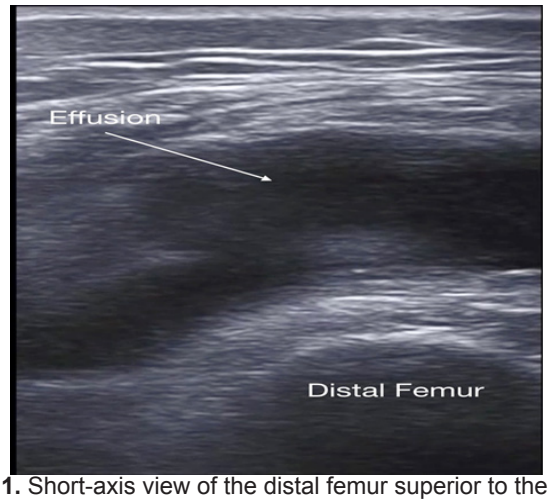

Image 1. Short-axis view of the distal femur superior to the patella using a linear high-frequency transducer. A large knee joint effusion is shown anterior to the distal femur.

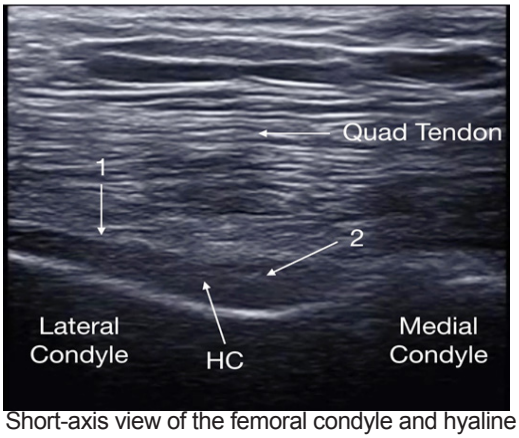

Image 2. Short-axis view of the femoral condyle and hyaline cartilage (HC) using a high-frequency linear transducer. Mild hyperechoic enhancement of the superficial layer (arrow 1) and hyperechoic thickening within the intermediate layer of the HC (arrow 2) are visible-the so-called pseudo-double contour sign.

and negative cultures. The pseudo-double contour sign is formed when calcium pyrophosphate crystals deposit in the hyaline cartilage[1] (depicted in Image 2). This is opposed to the double contour sign seen in gout (Image 3), where monosodium urate crystals deposit on the surface of the articular cartilage causing hyperechoic enhancement of the superficial margin.[1,2] Image 4 depicts a normal knee for comparison.

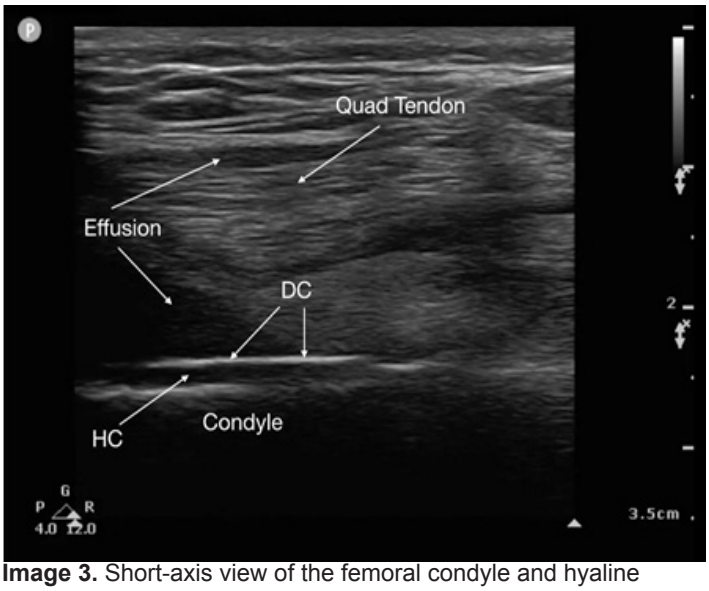

Image 3. Short-axis view of the femoral condyle and hyaline cartilage (HC) using a high-frequency linear transducer, which shows intense hyperechoic enhancement of the superficial layer of the HC, the double contour (DC) sign. Thickening and hyperechoic enhancement of the intermediate layer is absent.

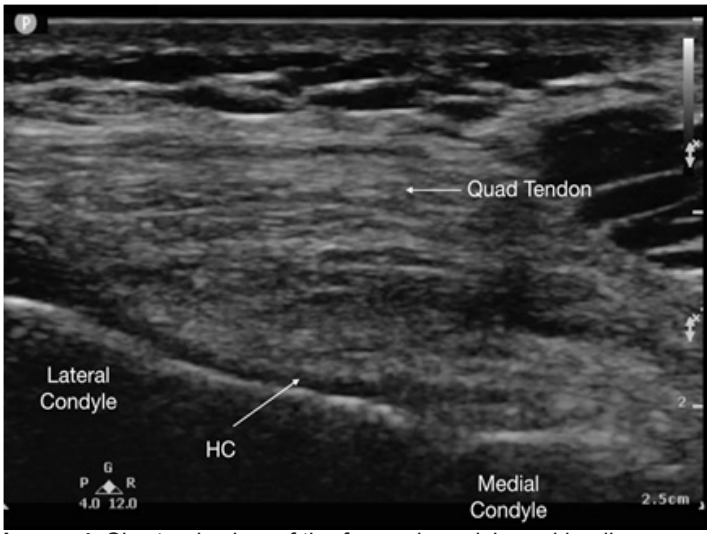

Image 4. Short-axis view of the femoral condyle and hyaline cartilage (HC) of a normal knee using high-frequency linear transducer. Note the absence of hyperechoic enhancement of the superficial layer of the HC and absence of hyperechoic enhancement and thickening of the intermediate layer of the HC.

CPC-EM Capsule

What do we already know about this clinical entity?
Ultrasound detection of calcium pyrophosphate crystals in pseudogout has high sensitivity and specificity per rheumatology literature.

What is the major impact of the image(s)?
These point-of-care ultrasound (POCUS) images will help emergency physicians diagnose and differentiate pseudogout and gout.

How might this improve emergency medicine practice?
Identifying pseudogout or gout on POCUS may decrease the need for aspiration and risk of infection in patients with previously diagnosed crystalline arthropathies.

Ultrasound for detection of calcium pyrophosphate crystals in pseudogout and chondrocalcinosis has sensitivity of 86.7% and specificity of 96.4%.[3] The double contour sign for gout has a reported sensitivity of 57% and 100% specificity for knees.[4] POCUS is a valuable tool in diagnosing crystalline-induced arthropathy.[5] Emergency physicians should consider using POCUS as an aid in diagnosing microcrystalline disease and guiding joint aspiration, as it may reduce the need for aspiration in the patient previously diagnosed with gout or pseudogout.

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

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Acquired Pediatric Right Diaphragmatic Hernia Following Automatic Implantable Cardioverter-defibrillator Placement

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Diaphragmatic hernias are an uncommon occurrence in the pediatric population; however, they can cause significant morbidity and mortality if the diagnosis is missed or delayed. This case discusses the radiographic and clinical exam findings of a one-year-old patient with this pathology. [Clin Pract Cases Emerg Med. 2019;3(4):428–429.]

CASE PRESENTATION

A one-year-old male with a past medical history of prolonged QT syndrome and past surgical history of a sternotomy for automatic implantable cardioverter-defibrillator (AICD) and pacemaker placements presented to the emergency department (ED) in moderate respiratory distress with tachypnea, nasal flaring, and subcostal retractions. The father reported the patient had been irritable, coughing, vomiting, and diaphoretic over the prior day.

Chest radiograph (CXR) demonstrated infiltrate versus atelectasis in the right lung base with a large right-sided diaphragmatic hernia demonstrated by infiltration of the large bowel into the right hemithorax with apparent bowel gas in the chest cavity (Image 1). CXR from six months prior showed no evidence of diaphragmatic hernia (Image 2). Prior to transfer to higher level of care, the patient received fluid resuscitation, supplemental oxygen, nebulized albuterol, and antibiotics for presumed pneumonia. His tachypnea and hypoxia subsequently improved. During operative intervention, findings included herniation of the small bowel, colon, and omentum through the right-sided diaphragmatic defect in the anterior subxiphoid region, with the liver displaced posteriorly. The pacemaker coil leads were wrapped within this hernia.

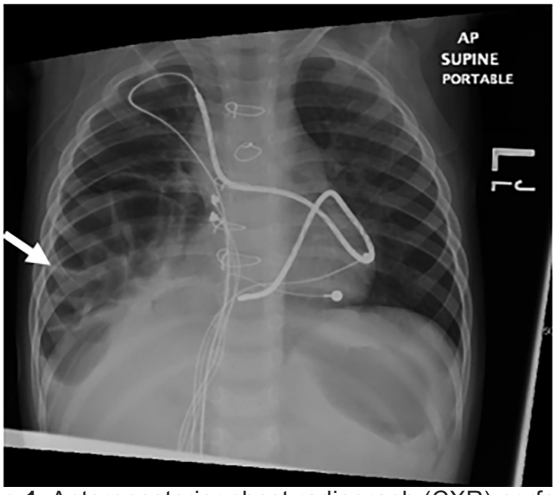

Image 1. Anteroposterior chest radiograph (CXR) performed on a child who presented to the emergency department in respiratory distress; CXR demonstrated significant elevation of the right hemidiaphragm with colonic interposition and right lung base infiltrate (arrow).

DISCUSSION

In this case, the right-sided diaphragmatic hernia was likely a sequelae of the initial surgical placement of the AICD. As seen on CXR (Image 3) from approximately one month prior to presentation in the ED, the diaphragmatic hernia slowly increased in size and demonstrated colonic interposition. The patient presented with nausea and vomiting but was asymptomatic from a respiratory standpoint; therefore, no

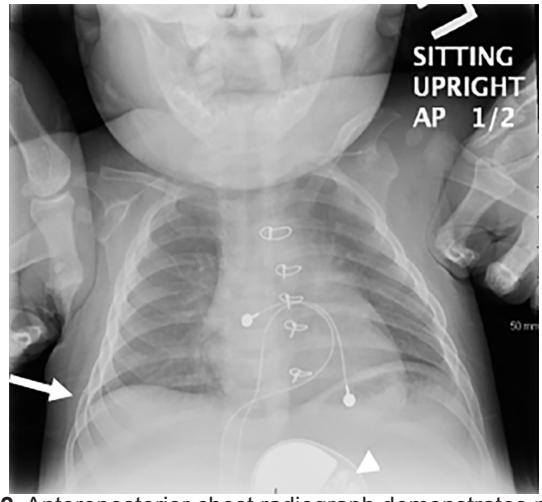

Image 2. Anteroposterior chest radiograph demonstrates normal right-sided costophrenic angle and prior stemotomy without any elevation of the diaphragm (arrow). Also visualized is the pulse generator placed in the abdomen (arrowhead).

intervention was pursued as he had close follow-up with an outside facility. The hernia eventually progressed, causing respiratory distress and ventilation-perfusion mismatch. This manifested as an increased respiratory rate, evidence of auto-peep, and feeding difficulties.

While uncommon in children, patients with diaphragmatic hernias may present with respiratory distress, unspecified thoracoabdominal pain, or symptoms of gastrointestinal obstruction such as nausea and vomiting. Herniation of abdominal contents into the chest cavity may also be identified by auscultation of bowel sounds in the chest.[1,2] The patient should be radiographically examined quickly upon presentation to avoid the morbidity and mortality associated with delayed diagnosis and management.[1,2]

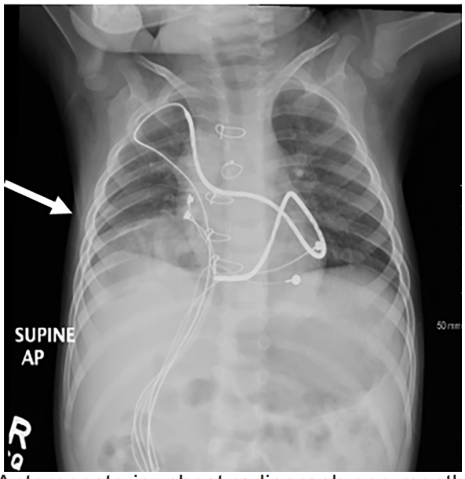

Image 3. Anteroposterior chest radiograph one month prior to image 1, demonstrating right colonic interposition with probable associated atelectasis (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Right-sided diaphragmatic hernias are difficult to diagnose in children, and diagnostic delay contributes to morbidity and mortality.

What is the major impact of the image(s)?
Diagnosis of right-sided diaphragmatic hernia, a likely sequela of automatic implantable cardioverter-defibrillator (AICD) placement, was made via chest radiograph.

How might this improve emergency medicine practice?
Children presenting with an AICD presenting respiratory distress should warrant suspicion of right diaphragmatic hernia and be radiographically examined to avoid mortality.

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

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Cardiac Standstill With Intracardiac Clot Formation

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This case describes and depicts cardiac standstill with thrombosed blood within the chambers of the heart. This was likely due to stasis of blood from a prolonged no-flow state. After viewing this ultrasound finding, the decision was made to halt resuscitative efforts in this case of a patient in cardiac arrest. [Clin Pract Cases Emerg Med. 2019;3(4):430–431.]

CASE PRESENTATION

An 88-year-old female with gastric cancer presented to the emergency department (ED) in cardiac arrest. She was at an outpatient clinic when she lost pulses. Cardiopulmonary resuscitation (CPR) was initiated by emergency medical services upon their arrival after a downtime of several minutes without chest compressions. Upon arrival in the ED, CPR was in progress. During a rhythm and pulse check, transthoracic echocardiography was performed demonstrating cardiac standstill, as well as a collection of echogenic material within the ventricles. This finding represented thrombosed blood (Image 1-2 and Video). After visualizing cardiac standstill with intracardiac clots, the decision was made to stop resuscitation.

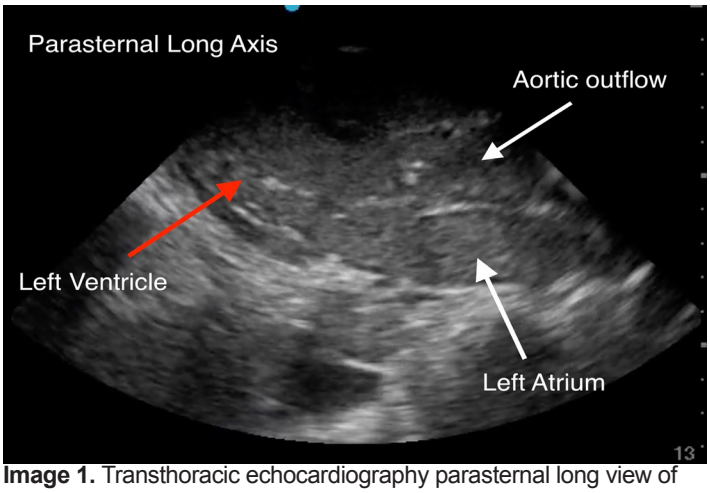

Image 1. Transthoracic echocardiography parasternal long view of the heart demonstrating echogenic (thrombosed) blood within the left atria, left ventricle, and aortic outflow.

DISCUSSION

Cardiac standstill is defined as the lack of movement of the valves and free wall of the heart. When visualized on ultrasound during cardiac arrest assessment it has been discussed as a potential endpoint to CPR. Cohort studies have shown that cardiac standstill was associated with a very low rate of survival from 0-0.6%.[1,2] In contrast, cardiac activity on ultrasound during cardiac arrest is strongly associated with return of spontaneous circulation and survival, and therefore provides prognostic information that may guide resuscitation efforts.[3]

This case also demonstrates clot formation within the heart, which is hypothesized to be from stasis of blood after a prolonged downtime prior to initiating CPR. Due to the initial lack of chest compressions, it is possible that there was a period without blood flow through the heart causing coagulation. This patient's history of cancer also may have contributed to a hypercoagulable state. There are no case reports discussing intracardiac clots in cardiac arrest, but this type of echocardiography pattern has been described to be fine, speckled, and with a uniform appearance.[4] Perhaps with further investigation, this echocardiography pattern

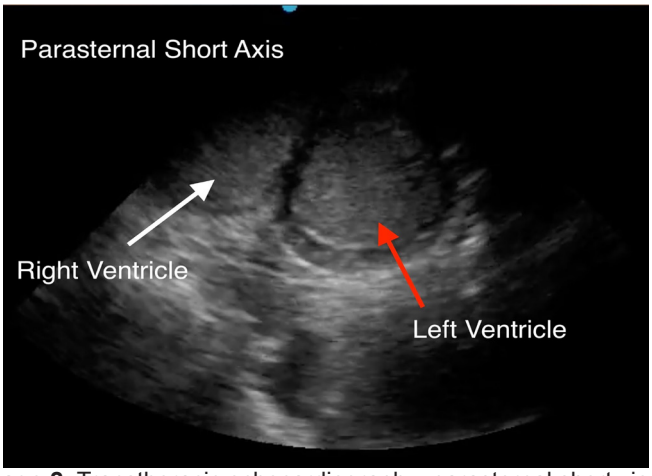

Image 2. Transthoracic echocardiography: parasternal short view of the heart demonstrating echogenic (thrombosed) blood within the left and right ventricles.

could be used to identify cardiac arrest patients with a prolonged downtime and potentially shed light on a prognosis if extensive intracardiac clots were found with or without ongoing cardiac activity.

CPC-EM Capsule

What do we already know about this clinical entity?
Increased clot burden can be caused by a low-flow state, create an even lower flow state, and can increase strain on the myocardium.

What is the major impact of the image(s)?
Rarely do you see this amount of clot burden inside both ventricles of a patient with a witnessed arrest and cardiopulmonary resuscitation. It provides a clear illustration.

How might this improve emergency medicine practice?
The presence of significant clot burden in both ventricles could give the physician an indication of prolonged downtime.

Video. Transthoracic echocardiography: parasternal long view of the heart demonstrating cardiac standstill and thrombosed blood within the heart chambers.

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

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An Elderly Woman With Gibbus Deformity and Physiologic Shock

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Physiological shock requires prompt diagnosis and treatment in the emergency department. We present a case of physiological shock in a 91-year-old woman resulting from obstruction of the left atrium and inferior vena cava by a giant esophageal hiatal hernia, identified using computed tomography imaging. The patient's age and history, including diet and eating behavior (namely needing to lie down immediately after a meal), and kyphotic posture were important factors to consider in establishing the differential diagnosis. While rare, a giant esophageal hiatal hernia should be considered in the differential diagnosis of obstructive shock. [Clin Pract Cases Emerg Med. 2019;3(4):432–433.]

CASE PRESENTATION

A 91-year-old Asian woman was admitted to our emergency department with complaints of nausea and dyspnea. One month prior to admission, the patient had become bedridden, and could maintain an upright sitting position for only 30 minutes. Moreover, she reported having to lie down quickly after meals. The patient, who was 143 centimeters tall and weighed 35 kilograms, had a significant thoracic gibbus deformity (extreme thoracic kyphosis from vertebral collapse and wedging).

Vital signs were as follows: a blood pressure of 89/52 millimeters of mercury, pulse of 123 beats per minutes, respiration of 32 breaths per minutes, and oxygen saturation (on ambient air) of 91%. Physical examination revealed epigastric tenderness, cool extremities, dry mouth, and livedo reticularis of both lower limbs. In addition to these physical findings, the laboratory evaluation was significant for a metabolic acidosis and hyperkalemia. There was no ST-T change on the electrocardiogram, and the transthoracic echocardiography was of poor quality, because of the patient's short stature and kyphotic posture. We then performed chest radiography (CXR) and computed tomography (CT) (Image).

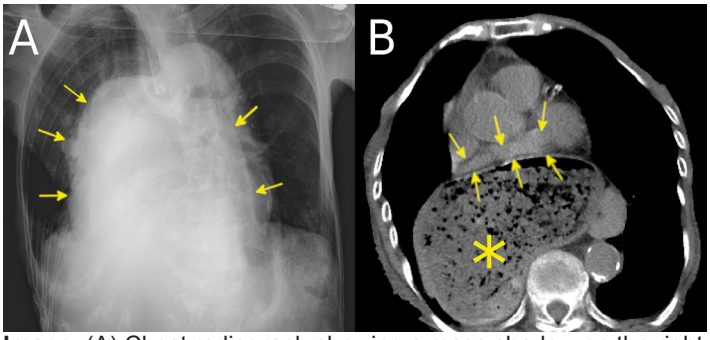

Image. (A) Chest radiograph showing a mass shadow on the right side of the mediastinum (arrows). (B) Computed tomography of the chest in axial view showing displacement of the left atrium (arrows) by a giant esophageal hiatal hernia (stars).

DISCUSSION

The CXR showed a mass shadow on the mediastinum. CT showed the left atrium being compressed by a hiatal hernia. Obstructive shock, secondary to a giant esophageal hiatal hernia, was diagnosed. Both nasogastric tube and endoscopic examination failed to remove the stomach

contents. The patient was not deemed to be a surgical candidate because of her advanced age and poor overall condition. After conservative treatment, she died with non-occlusive mesenteric ischemia on the following day.

The risk factors of esophageal hiatal hernia include female sex, aging, and gibbus.[1] A hiatal hernia rarely leads to death. Only two previous similar cases have previously been reported in the literature and indicate that surgical management is necessary.[2,3] We suggest that obstructive shock should be considered in elderly women, particularly in those with a gibbus deformity.

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

CPC-EM Capsule

What do we already know about this clinical entity?
A hiatal hernia is well known as a common disease of aged people and it also causes reflux esophagitis.

What is the major impact of the image(s)?
Obstructive shock is a major type of shock, caused by pulmonary embolism, cardiogenic tamponade, and tension pneumothorax. However, a hiatal hernia can also cause obstructive shock.

How might this improve emergency medicine practice?
The obstructive shock caused by hiatal hernia should be considered in elderly women, particularly in those with a gibbus deformity.

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Retained Catheter in the Aorta

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Due to the recent increase in endovascular procedures, retained foreign bodies such as stents and catheters in vasculature have become a common and serious complication. Treatments for these complications vary depending on the acuity and stability of the foreign body in the vessel. We discuss a rare case of an adult found to have an incidental retained umbilical artery catheter in the aorta. [Clin Pract Cases Emerg Med. 2019;3(4):434–435.]

CASE PRESENTATION

A 25-year-old female with no past medical or surgical history presented to the emergency department with a complaint of intermittent epigastric and abdominal pulsation sensation that she had been experiencing for the prior year. An abdominal aorta ultrasound showed no evidence of aneurysm or dissection, but a 3.9-centimeter, echogenic tubular structure was found in the distal abdominal aorta consistent with a retained catheter fragment (Images 1 and 2). The patient reported that she was born premature at seven months at an outside hospital. Given her history and the ultrasound findings, there was high suspicion for a retained fragment of an umbilical catheter in her aorta. Vascular surgery was consulted. The patient was seen in clinic but was later lost to follow-up before further imaging and treatment.

DISCUSSION

This case demonstrates an incidental finding of a retained umbilical artery catheter fragment in an adult patient's aorta. There are several case reports of retained umbilical artery catheter fragments in neonates and infants, which resulted in thrombosis, infection, and embolization.[1,2] This is the first known finding of a retained umbilical artery catheter found in an adult patient. Given the recent increase in endovascular procedures, retained foreign bodies such as stents, coils, guidewires, and catheters in vasculature have become a common and serious complication.[3] The recommended treatment for acute and nonadherent foreign body is endovascular retrieval, which has a high success rate with minimal mortality. However, in the case of stable intravascular foreign bodies, which are adherent to the vessel wall, the benefits of removal such as decreasing the risk of thrombosis and further migration should be weighed against the risks. In those cases, leaving the foreign body in place is an option.[4]

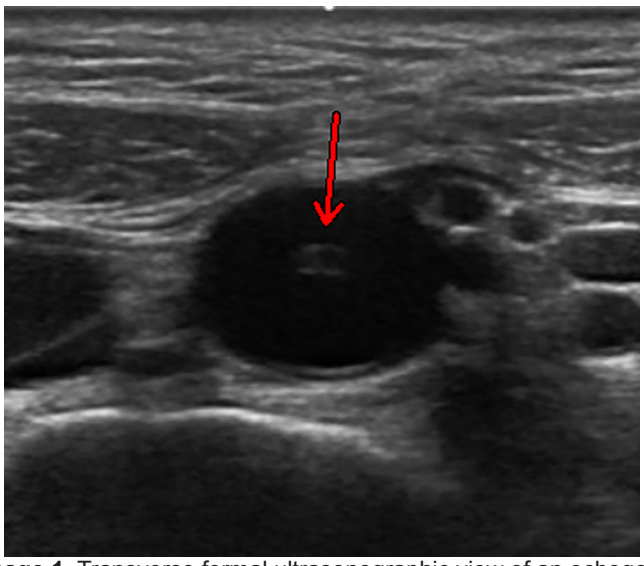

Image 1. Transverse formal ultrasonographic view of an echogenic tubular structure (arrow) in the aorta.

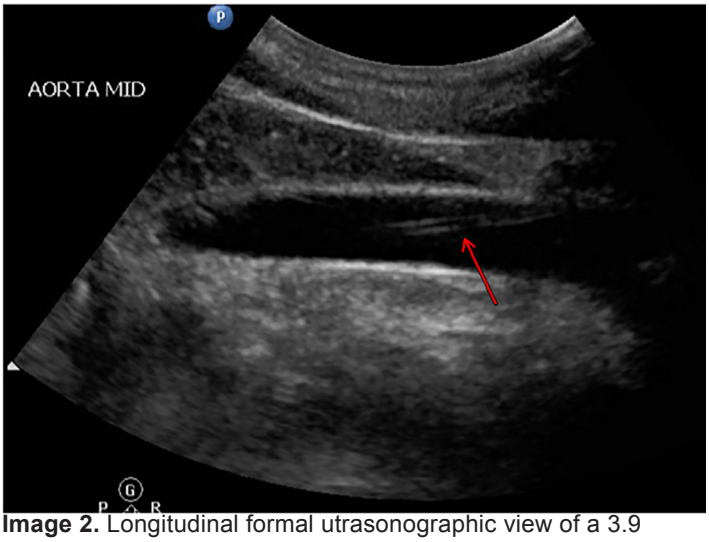

Image 2. Longitudinal formal utrasonographic view of a 3.9 centimeters (cm), echogenic tubular structure with the distal tip located 1.7 cm from the bifurcation (arrow).

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

CPC-EM Capsule

What do we already know about this clinical entity?
Intravascular retained foreign bodies as a result of endovascular procedures can have many complications, including thrombosis and infection.

What is the major impact of the image(s)?
This is the first known case of retained umbilical artery catheter fragment found in an adult.

How might this improve emergency medicine practice?
Incidental findings of stable intravascular foreign bodies in the emergency department can be managed with observation and referral to vascular surgery.

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Monocular Vision Loss: A Rare Cause

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A 62-year-old woman with a history of metastatic breast cancer and known meningioma presented with unilateral vision loss associated with anisocoria and an afferent pupillary defect. On magnetic resonance imaging we found the cause to be optic nerve compression by a right frontal meningioma. Monocular vision-loss etiologies are anatomically localized to structures anterior to the optic chiasm. This case serves as a reminder that cerebral structures in this location must not be forgotten in the differential. [Clin Pract Cases Emerg Med. 2019;3(4):436–437.]

CASE PRESENTATION

Our patient had a past medical history significant for right frontal lobe meningioma and metastatic breast cancer who awoke from sleep approximately 20 hours prior to presentation with acute, painless, right eye vision loss and enlarged right pupil. Her physical exam consisted of visual acuities of 20/100 right, 20/25 left, right pupillary enlargement with afferent pupillary defect, and normal ocular pressure bilaterally. Magnetic resonance imaging (MRI) demonstrates pre-chiasmatic optic nerve compression secondary to frontal lobe mass (Images 1 and 2).

DISCUSSION

The visual pathway requires that causes of monocular vision loss sit anterior to the optic chiasm.[1] One must be mindful of the cerebral anatomy within the cranium, where the frontal lobe sits anterior to the optic chiasm. Therefore, mass effect must be considered in the differential for monocular vision loss. Prior to MRI, we obtained a computed tomography for definitive imaging secondary to previously known meningioma measuring 1.7 centimeters by 2 centimers.

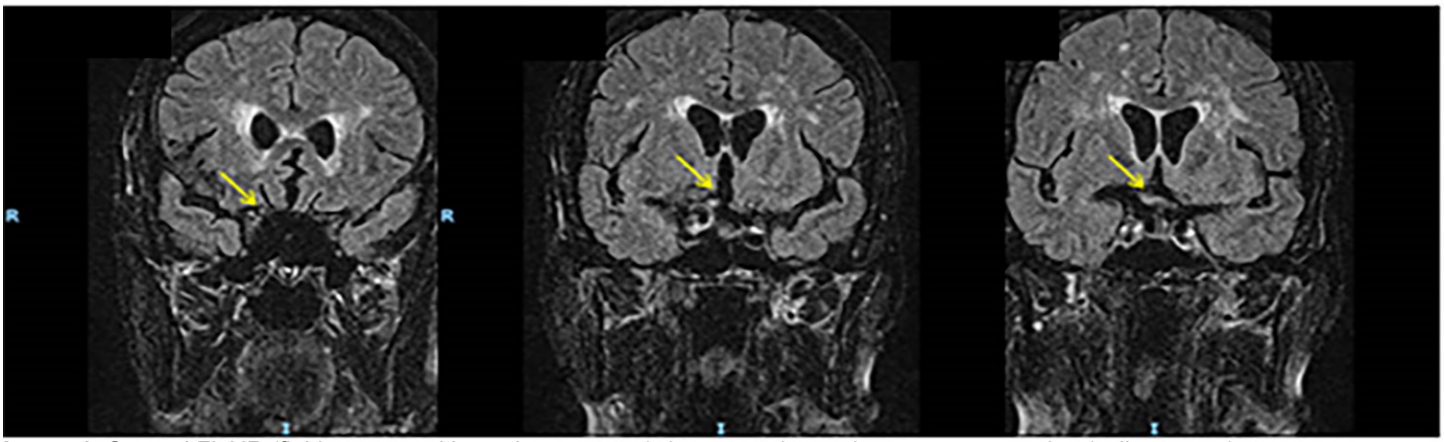

Image 1. Coronal FLAIR (fluid-attenuated inversion recovery) demonstrating optic nerve compression (yellow arrow).

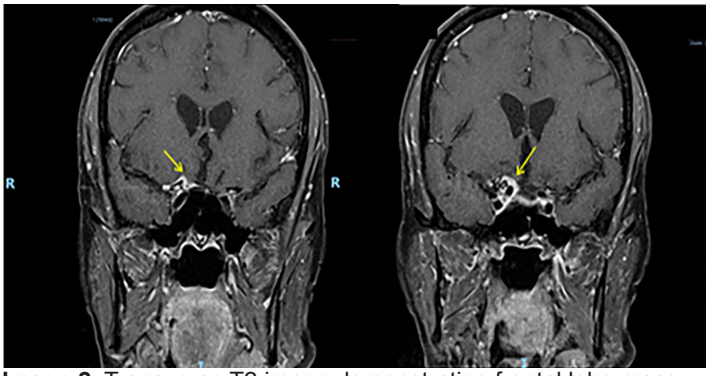

Image 2. Transverse T2 image demonstrating frontal lobe mass abutting the optic nerve (yellow arrow).

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

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CPC-EM Capsule

What do we already know about this clinical entity?
Acute vision loss requires prompt evaluation with a careful history and physical examination. Due to the nerve and vascular supply to the eye, the etiologies of monocular vision loss are most commonly secondary to vascular, intra-ophthalmic, or inflammatory pathologies.

What is the major impact of the image(s)?
The magnetic resonance image in this report demonstrates a frontal lobe mass that impinges on the optic nerve, a rare cause of monocular vision loss.

How might this improve emergency medicine practice?
This case report can remind emergency physicians that, in addition to ophthalmologic and vascular causes, monocular vision loss can be caused by central nervous system lesions that are located anterior to the optic chiasm.[2]

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Sonographic Detection of Cutaneous Myiasis

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Cutaneous maggots are occasionally encountered in the emergency department. We present a case in which maggots were visually identified and ultrasound was used to detect additional maggots below the skin crevices in a patient with elephantiasis nostras verrucosa. [Clin Pract Cases Emerg Med. 2019;3(4):438–439.]

CASE PRESENTATION

A 47-year-old male with super morbid obesity, bilateral lower extremity dermatitis and lymphedema, presented to our emergency department with redness, pain, and potential maggots in the left lower extremity. One year prior he had burned his left leg with boiling water but never sought medical treatment. On exam, he had chronic skin changes of venous stasis, lymphedema, and burn scarring. The skin was lichenified with deep crevices (Image). This condition, seen in severe chronic lymphedema, is called elephantiasis nostras verrucosa.

Two maggots were identified on the skin surface. Ultrasound with linear transducer was used to determine whether or not gas was present in the tissues. Rather than identify gas in the tissues, sonography revealed additional maggots within the crevices of the skin. These appeared as oblong-shaped structures with a hypoechoic rim and a hyperechoic center. This structure is similar to criteria for the presence of myiasis suggested by Bouer et al.[1] In this case, some of the larvae borrowed beneath the folds of the patient's skin and would intermittently "pop-up" toward the ultrasound probe (Video). The maggots were removed with forceps, and the wounds were thoroughly cleansed and disinfected. There remained concern for bacterial superinfection; therefore, the patient was admitted to the medicine service, and an infectious disease consult was obtained. Patient received seven days of intravenous piperacillin-tazobactam and was discharged home on an additional seven days of oral amoxicillin clavulanate.

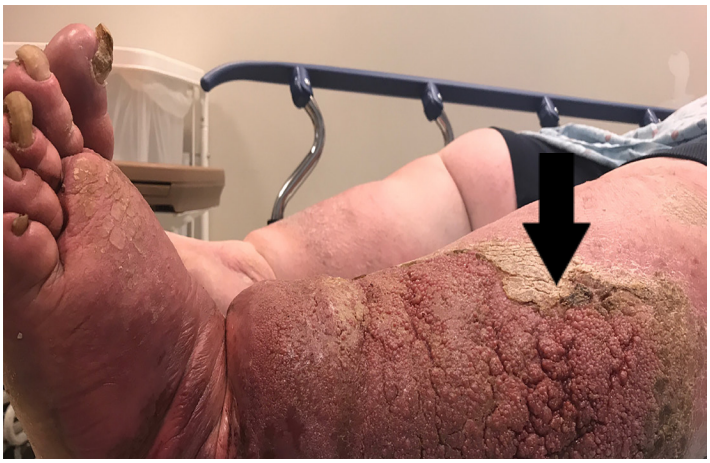

Image. A photograph of the patient's left lower extremity showing lichenfication of the skin (arrow) and crevices.

DISCUSSION

Myiasis is infection with fly larvae, usually occurring in tropical or subtropical areas. Accidental myiasis (also called pseudomyiasis) is caused by flies that have no preference or need to develop in a host but do so on occasion.[2] Transmission in this case occurred through the deposit of eggs on the patient's lichenified skin surface. The larvae of the housefly then feed on both live and necrotic tissue causing myiasis to develop. Myiasis is often misdiagnosed because its symptoms are non-specific. Clues include travel to an endemic area (which our patient did not have), one or more non-healing lesions on the skin, pruritis, movement under the skin, or pain.[3] While this patient did have a few scattered maggots on his legs, ultrasound was able to readily reveal the presence of more maggots below the most superficial skin surface. Mechanical removal of fly larvae is usually curative unless

bacterial superinfection has occurred, in which case antibiotics would be indicated.

Video. Note the maggot on the left of the image (arrow) which briefly appears above the skin surface and subsequently burrows beneath the skin surface.

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

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CPC-EM Capsule

What do we already know about this clinical entity?
Cutaneous myiasis is occasionally encountered in the emergency department. Most of the time the diagnosis is immediately visible to the examiner.

What is the major impact of the image(s)?
This case of a patient with elephantiasis nostra verrucosa illustrates the ability of the examiner to use ultrasound to identify additional maggots which were not readily visible on physical exam.

How might this improve emergency medicine practice?
Ultrasound may be able to detect myiasis in other chronic skin conditions where maggots may not be immediately visible to the examiner.

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Opioid Overdose With Parkinsonian Features

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A 54-year-old man presented to the emergency department with confusion and Parkinsonian features after suspected heroin snorting. He had magnetic resonance imaging of the brain demonstrating isolated symmetric bilateral globus pallidus (GP) restricted diffusion and edema consistent with hypoxic ischemic encephalopathy. In contrast to other anoxic/ischemic insults, where the GP is preferentially spared, autopsy reports on intravenous heroin users have found the GP to be specifically affected, often demonstrating symmetric bilateral lesions. Opioid toxicity should be considered in patients presenting with Parkinsonian features on examination or pallidal lesions on imaging, especially in younger adults where infarction is less common. [Clin Pract Cases Emerg Med. 2019;3(4):440–441.]

CASE PRESENTATION

A 54-year-old man presented to the emergency department (ED) after receiving naloxone for a suspected opioid overdose. He was found in bed unresponsive, hypoxemic to SpO_2 83%, with sonorous respirations and presumed heroin powder on his face and bedside table. On arrival to the ED he was awake but confused and noted to have bilateral upper and lower extremity rigidity along with sustained clonus, hyperreflexia in upper and lower extremities, and complained of "foot cramping." A non-contrast computed tomography of the brain was normal (Image 1)**.** Brain magnetic resonance imaging obtained 15 hours later demonstrated isolated symmetric bilateral globus pallidus (GP) restricted diffusion and edema (Image 2).

DIAGNOSIS

In autopsies on patients with heroin use disorder, bilateral GP lesions were noted to be frequently encountered.[1] This finding is atypical compared to other anoxic-ischemic insults, where the GP is preferentially spared as opposed to the caudate.[2] This GP insult was once thought to be unique to carbon monoxide poisoning, but its causal etiology has expanded to include such conditions as methanol poisoning, cyanide poisoning and, increasingly recognized, opioid overdose. The mechanism behind this finding is not understood, although it has been speculated to be secondary to recurrent episodes of cerebral hypoxia as opposed to a direct neurotoxic substance. Lesions to the GP typically cause akinetic-rigid and/or dystonic syndromes such as noted in this patient.

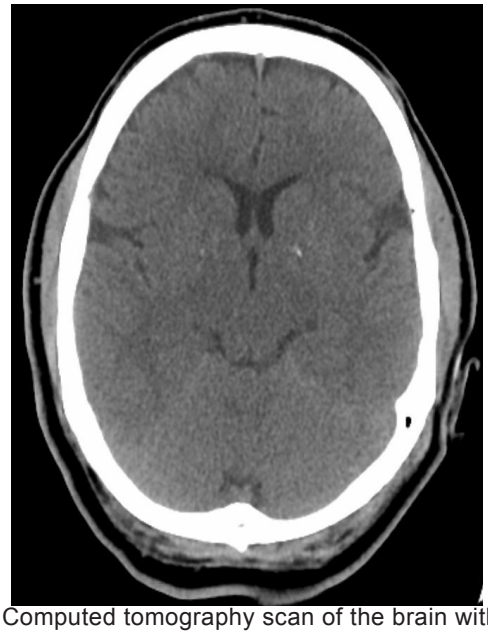

Image 1. Computed tomography scan of the brain without contrast obtained hours earlier without evidence of globus pallidus ischemia.

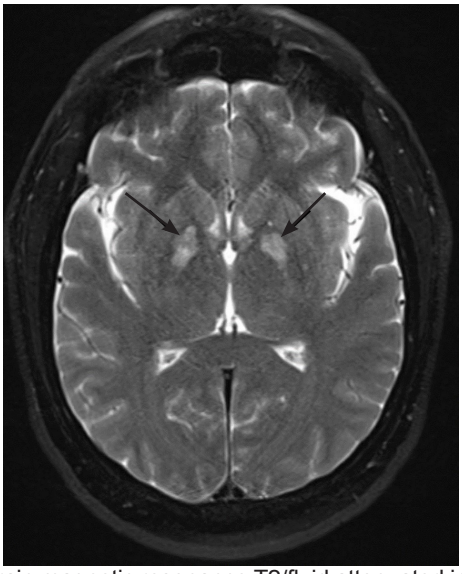

Image 2. Brain magnetic resonance T2/fluid attenuated inversion recovery with evidence of bilateral globus pallidus ischemia (arrows).

The patient was admitted to the hospital where he admitted to snorting heroin powder. He had resolution of his symptoms by hospital day five. Hypoxic ischemic insult, including opioid toxicity, should be considered in patients with Parkinsonian features on exam and/or isolated pallidal lesions on imaging without history of overt cardiorespiratory arrest, especially in adolescents and younger adults where infarction is less common.

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

CPC-EM Capsule

What do we already know about this clinical entity?
Bilateral Globus Pallidus (GP) lesions have been noted in autopsies of patients with heroin use disorder. The etiology is unclear as anoxic-ischemic insults typically affects other brain areas.

What is the major impact of the image(s)?
Toxin induced Globus pallidus injury is a recognized finding in carbon monoxide, methanol, and cyanide poisoning. This magnetic resonance imaging finding should prompt a differential that includes opioid use.

How might this improve emergency medicine practice?
Recognition of GP lesions may identify a patient at high risk of future opioid related injury or death; who would benefit from counseling, substance use referral, and other resources.

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Gastric Outlet Obstruction Due to Malposition of Replacement Gastrostomy Tube

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A 78-year old male presented to the emergency department after accidental dislodgement of his chronic gastrostomy tube. A replacement gastrostomy tube was passed easily through the existing stoma and flushed without difficulty. Confirmatory abdominal radiography demonstrated contrast in the proximal small bowel, but the patient subsequently developed epigastric pain and refractory vomiting. Computed tomography revealed the tip of the gastrostomy tube terminating in the pylorus or proximal duodenum. This case highlights gastric outlet obstruction complicating the replacement of a gastrostomy tube and the associated radiographic findings. [Clin Pract Cases Emerg Med. 2019;3(4):442–443.]

CASE PRESENTATION

A 78-year-old male with a history of stroke presented to the emergency department after accidental dislodgement of his chronic gastrostomy tube approximately five hours prior. The patient offered no other complaints and denied abdominal pain, nausea, or vomiting. On examination, he appeared comfortable with unremarkable vital signs. His abdomen was non-tender and demonstrated a patent, mature gastrostomy stoma. A replacement gastrostomy tube was passed easily through the existing stoma and flushed without difficulty. Confirmatory abdominal radiography revealed contrast in the duodenum and proximal jejunum, but no portion of the stomach was outlined (Image 1). Shortly after, the patient developed epigastric pain, nausea, and refractory vomiting. Subsequent computed tomography revealed the tip of the gastrostomy tube terminating in the pylorus or proximal duodenum (Image 2). The balloon was deflated, and the tube was retracted several centimeters with complete resolution of symptoms. The patient was discharged home with no further complications on follow-up.

DISCUSSION

Gastric outlet obstruction related to gastrostomy tubes is rare.[1-2] Mechanical obstruction of the pylorus results in abdominal cramping and intermittent vomiting that resolve with tube repositioning.[1,3] In the above case, the replacement gastrostomy tube was re-inserted directly into the gastric outlet; however, gastric outlet obstructions have been reported more in chronic indwelling catheters wherein dislodgement of the external bumper allows the tube to advance further into the stomach.[3] Substitution of Foley catheters for true gastrostomy tubes confers a greater risk of gastric outlet obstruction, and this practice is discouraged.[1,4]

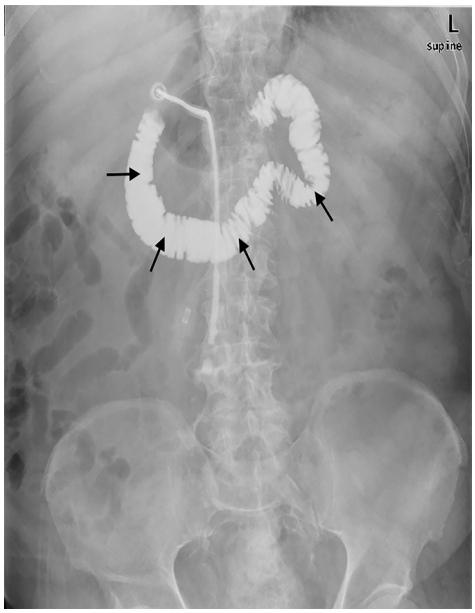

Image 1. Contrast-enhanced abdominal radiography outlining the duodenum/proximal jejunum (arrows).

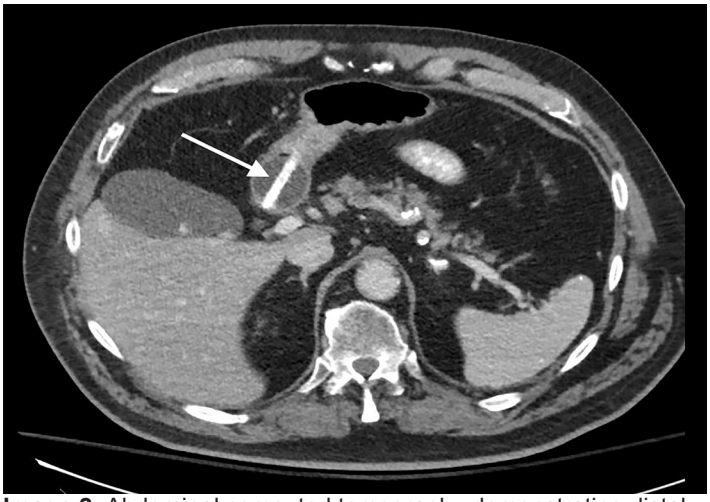

Image 2. Abdominal computed tomography demonstrating distal portion of replacement gastrostomy tube (arrow) in the pylorus/proximal duodenum.

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

CPC-EM Capsule

What do we already know about this clinical entity?
Dislodged gastrostomy tubes are commonly repositioned or replaced in the emergency department. Gastric outlet obstruction is a potential, albeit rare, complication.

What is the major impact of the image(s)?
Emergency physicians should be familiar with the appearance of gastric outlet obstruction due to gastrostomy tube malposition on contrast-enhanced plain radiography.

How might this improve emergency medicine practice?
Early identification of gastric outlet obstruction on plain radiography can prevent patient discomfort and the need for additional advanced imaging.

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An Unusual Case of Unilateral Papilledema

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Neuroretinitis from neurosyphilis is an uncommon finding in previously healthy young individuals. A 37-year-old presented with three days of painless, unilateral vision loss with an associated diffuse erythematous non-pruritic truncal rash. Physical exam demonstrated vision loss in the left eye. Fundoscopic exam showed unilateral peripapillary hemorrhage, papilledema and venous engorgement. Labs showed positive syphilis antibody qualitative. Magnetic resonance imaging demonstrated 12 millimeters of high right frontal lobe cerebrospinal fluid density. The patient was treated with benzylpenicillin and within 18 hours had improvement of his vision. [Clin Pract Cases Emerg Med. 2019;3(4):444–445.]

CASE PRESENTATION

A 37-year-old male presented to the emergency department complaining of three days of painless left eye vision changes. He described the changes as a "white out." He also noted a four-week-old diffuse, erythematous, nonpruritic truncal rash. Visual exam findings were notable for oculus sinister of 20/25. Oculus dexter was 20/20. There was no presence of ptosis. Bilateral fluorescein stain and slit lamp exam were unremarkable. A fundoscopic exam of the left eye revealed unilateral papilledema (UP) and bilateral retinal hemorrhage (Image 1). Ocular pressures were unremarkable.

Labs were significant for reactive hepatitis B, antinuclear antibody screen, rapid plasma reagin test with reflex, fluorescent treponemal antibody absorption test, sedimentation rate Westergren automated test, and c-reactive protein of 3.1 milligrams per deciliter (mg/dL) (0.0-3.1 mg/dL). Venereal disease research laboratory on cerebrospinal fluid (CSF) was nonreactive. Lyme antibody screen and Bartonella antibody panel were both negative. Syphilis antibody qualitative was positive. Rapid human immunodeficiency virus (HIV) test was negative. Syphilitic and UP findings prompted a lumbar puncture to rule out neurosyphilis, which subsequently revealed elevated lymphocytes 100% (40–80%) and protein 65.0 mg/dL (15.0–45.0 mg/dL).

A magnetic resonance imaging of the brain revealed 12 millimeters of high right frontal lobe CSF density (Image 2). The patient was given benzylpenicillin with subsequent vision improvement within 18 hours of administration, indicative of painless vision loss secondary to neurosyphilitic neuroretinitis.

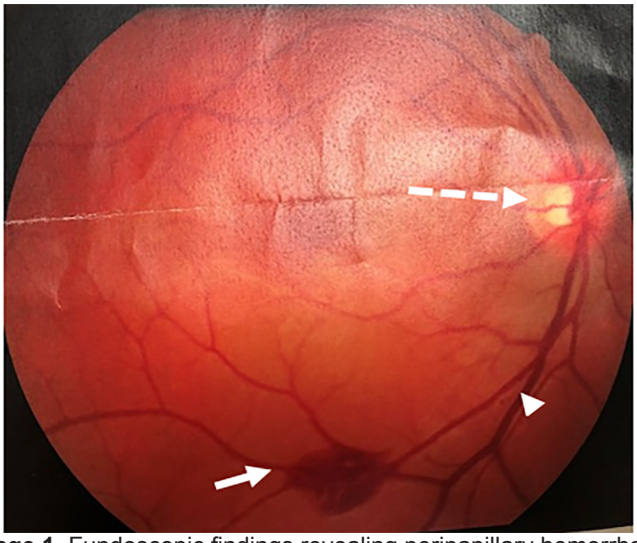

Image 1. Fundoscopic findings revealing peripapillary hemorrhage (white arrow), papilledema (dashed arrow), and venous engorgement (arrowhead).

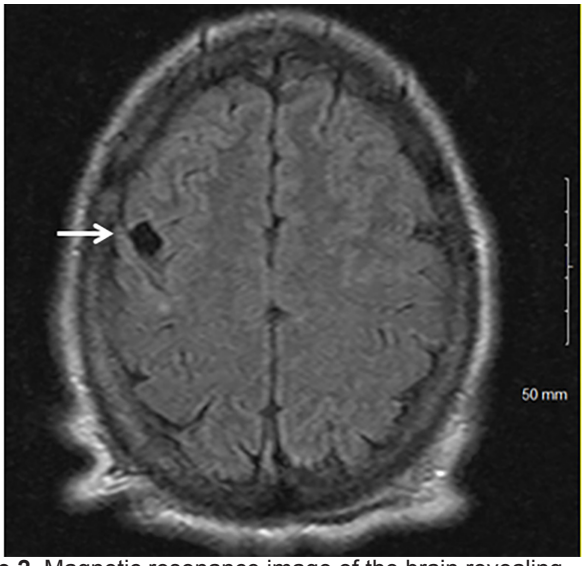

Image 2. Magnetic resonance image of the brain revealing 12-millimeter right frontal lobe cerebrospinal fluid density (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Neuroretinitis is most commonly found in patients with cat scratch disease.

What is the major impact of the image(s)?
This case demonstrates a rare presentation of unilateral papilledema in an otherwise healthy patient.

How might this improve emergency medicine practice?
Unilateral papilledema in younger populations presenting without pain should prompt a thorough medical and physical examination with a widened differential diagnosis.

DISCUSSION

Common causes of UP and vision loss include anterior ischemic optic neuropathy (AION) and optic neuritis (ON).[1] AION is often seen in patients older than 50 years with associated comorbidities, making it an unlikely cause of this patient's vision loss.[1] ON typically affects females between 20-35 years of age.[1] ON in 90% of cases has associated headaches, eye pain or both, whereas 19% of AION cases have associated pain.[1] Neuroretinitis is uncommon, typically characterized by optic disc edema and subsequent formation of a macular star figure. The underlying pathophysiology involves increased permeability of disc vasculature, but is not fully defined.[2]

Our patient's exam and symptoms were most consistent with neuroretinitis. Most cases of neuroretinitis are reported in ophthalmology literature and in association with cat scratch disease (CSD). Only about 1% of the 12,000 yearly cases of CSD present with neuroretinitis. Those who have reported cases of ophthalmological complaints not neuroretinitis specifically are individuals who are HIV positive.[3] Our specific case demonstrates an uncommon presentation of unilateral papilledema in a healthy patient. Painless unilateral papilledema in younger patients should raise concern for an insidious process and prompt thorough investigation.

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

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Disseminated Intravascular Coagulation With Purpura Fulminans Presentation of Acute Promyelocytic Leukemia

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A 47-year-old male presented to the emergency department with 12 hours of nausea, vomiting, abdominal pain, and a widespread skin eruption with mottled, irregular, purpuric lesions with subsequent rapid decompensation. Laboratory analysis revealed thrombocytopenia, bandemia, elevated metamyelocytes, abnormal coagulation panel, decreased fibrinogen, elevated fibrin split products, renal dysfunction, bacterial rods, dohle bodies, and toxic granulation. Acute promyelocytic leukemia (APML) was confirmed via bone marrow biopsy, flow cytometry, and fluorescence in situ hybridization analysis. Disseminated intravascular coagulation (DIC) may be the initial presentation of APML. When treated promptly, APML can achieve high remission rates; however, conditions such as DIC continue to increase mortality requiring early recognition to improve survival rates. [Clin Pract Cases Emerg Med. 2019;3(4):446–448.]

CASE PRESENTATION

A 47-year-old male with no past medical history presented with nausea, vomiting, diffuse abdominal pain, and a widespread rash that began 12 hours prior to arrival. Physical examination revealed an ill-appearing male with diffuse abdominal tenderness as well as diffuse purpuric, mottled skin (Image). Vital signs revealed a blood pressure of 93/74 millimeters of mercury, heart rate of 120 beats per minute, respiratory rate of 30 respirations per minute, and a rectal temperature of 103.7 degrees Fahrenheit with pulse oxygenation saturation of 94%. His course necessitated fluid boluses, vasopressors, broad-spectrum antibiotics, steroids, and intubation.

His laboratory findings revealed bandemia, thrombocytopenia, acute kidney injury, elevated coagulation markers, decreased fibrinogen, elevated fibrin split products, elevated lactate dehydrogenase, and a lactic acidosis as shown in the Table, along with an unremarkable computed tomography of his abdomen and pelvis without contrast. The presumptive diagnosis was the development of acute promyelocytic leukemia (APML) with disseminated intravascular coagulation (DIC), an uncommon skin presentation called purpura fulminans (PF), and septic shock.

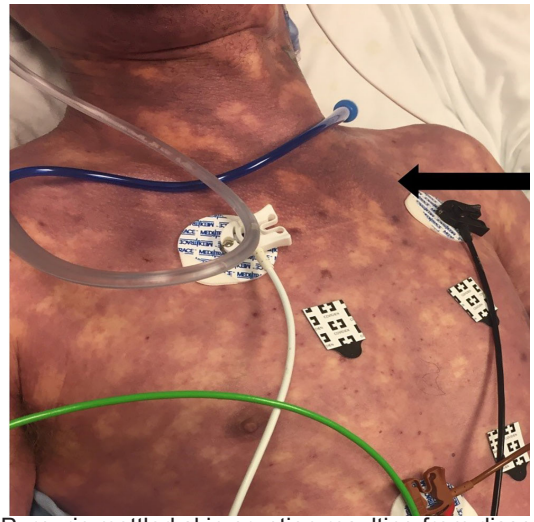

Image. Purpuric mottled skin eruption resulting from disseminated intravascular coagulation (arrow).

DISCUSSION

The patient was transferred to a tertiary care center and underwent bone marrow biopsy, flow cytometry, and fluorescence in situ hybridization analysis confirming APML. APML constitutes 10% of all adult acute myelogenous leukemia and has a high incidence of life-threatening hemorrhage secondary to fibrinolytic-type DIC, which has been attributed to both the development of APML as well as sepsis secondary to APML.[1,2] There is a high misdiagnosis and delayed treatment rate secondary to the varied and complex presentation. APML results from chromosomal abnormalities that involve a balanced translocation between chromosomes 15 and 17 with an incidence of 600-800 cases annually in the United States.[3] The average age at time of diagnosis is between 20 and 50 years of age.

APML should be considered in those with presentations of bleeding, DIC, or infections with laboratory abnormalities of thrombocytopenia, anemia, and subnormal to elevated white blood cell count. In cases of DIC, scoring algorithms use platelet count, level of fibrin markers such as d-dimer and fibrin split products, coagulation studies, and fibrinogen levels to arrive at a diagnosis. Additionally, with DIC, PF may also occur. PF is a poorly understood entity; it occurs secondary to endovascular thrombotic events and endothelial damage, which contribute to the characteristic purpuric appearance.[4] The mainstay of treatment is aggressive supportive care as well as identifying and treating the underlying coagulopathy and infections, in addition to induction therapy with all-trans-retinoic-acid and chemotherapeutic agents in consultation with a hematologist.[5]

CPC-EM Capsule

What do we already know about this clinical entity?
Acute promyelocytic leukemia (APML) is associated with a high incidence of fibrinolytic-type disseminated intravascular coagulation (DIC).Treatment is supportive.

What is the major impact of the image(s)?
The image reinforces what purpura fulminans looks like and highlights the occurrence of DIC in APML and the importance of early recognition and treatment.

How might this improve emergency medicine practice?
Keeping a broad differential with knowledge of this emergent condition will yield earlier coordination of care and treatment of APML, which carries a high misdiagnosis and mortality rate.

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

Table. Abnormal laboratory findings in patient diagnosed with acute promyelocytic leukemia.

Laboratory findings	Values	Reference ranges
White blood cell	9.9x10^3 x 1000/mm^3	4-11 x 1000/mm^3
Hemoglobin	16.1 g/dL	12-18 g/dL
Platelets	27 x10^3 x 1000/mm^3	150-450 x 1000/mm^3
Manual differential	26% bands, 6% metamyelocytes, as well as many bacteria rods, Dohle bodies, and evidence of toxic granulation.	Segmented neutrophils: 50%–70%. Band forms: 0–5%. Lymphocytes: 30%–45%. Monocytes: 0–6%. Basophils: 0–1%. Eosinophils: 0–3%
Coagulation panel	Prothrombin time: 24.9 seconds, partial thromboplastin time: 151 seconds, INR: 2.1	Prothrombin time: 11-13 seconds, partial thromboplastin time: 25-35 seconds, INR: 0.8-1.2
Fibrinogen	<80 mg/dL	200-400 mg/dL
Fibrin split products	>40 ug/ml	<10 ug/ml
Creatinine	3.2 mg/dL	0.7-1.3 mg/dL
Lactate dehydrogenase	868 u/L	80-225 u/L
Lactic acid	11.5 mmol/L	0.7-2.1 mmol/L

mm^3, cubic millimeters; *g/dL,* grams per deciliter; *INR,* international normalized ratio; *mg/dL,* milligrams per deciliter; *ug/ml,* micrograms per milliliter; *mmol,* millimoles; *L,* liter; *u,* unit.

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Young Man With Suspected Foreign Body Ingestion

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As United States emergency departments (ED) and hospitals continue to contend with increasing numbers of patients presenting with complications of substance abuse, emergency physicians should also be aware of patients who may be smuggling illicit drugs. We report the case of a 26-year-old man who was transported to the ED for suspected drug smuggling. Abdominal computed tomography was notable for the presence of multiple tubular foreign bodies throughout the colon that were later identified as packets containing heroin. Body-packing patients present a high-risk clinical scenario that may result in massive, inadvertent drug exposure. [Clin Pract Cases Emerg Med. 2019;3(4):449–450.]

CASE PRESENTATION

A 26-year-old man was transported from the airport to the emergency department (ED) in police custody after being detained for suspected drug smuggling. The patient had no medical complaints and denied any ingestions. On arrival, he appeared anxious but readily consented to ED evaluation and treatment. He had normal vital signs and an unremarkable physical examination. His abdomen was soft, nontender, and nondistended without palpable mass. No signs of a recognizable toxidrome were present. We obtained abdominal computed tomography (CT) with oral and intravenous contrast for further evaluation (Image 1).

DIAGNOSIS

Body Packing

Abdominal CT revealed multiple radiopaque foreign bodies present throughout the colon without evidence of bowel obstruction. Whole bowel irrigation with four liters of oral polyethylene glycol was administered in the ED over approximately three hours. The patient was admitted to the intensive care unit (ICU) for observation, and over the following 24 hours he passed a total of 26 packets that contained heroin (Image 2). He was subsequently discharged in good condition in police custody.

"Body packing" refers to the ingestion of prepared packets of illicit drugs as a means of transport while avoiding detection.[1] Providers must have a high degree of suspicion based on the available history as over 50% of patients will be asymptomatic on presentation.[2] Abdominal radiographs have a

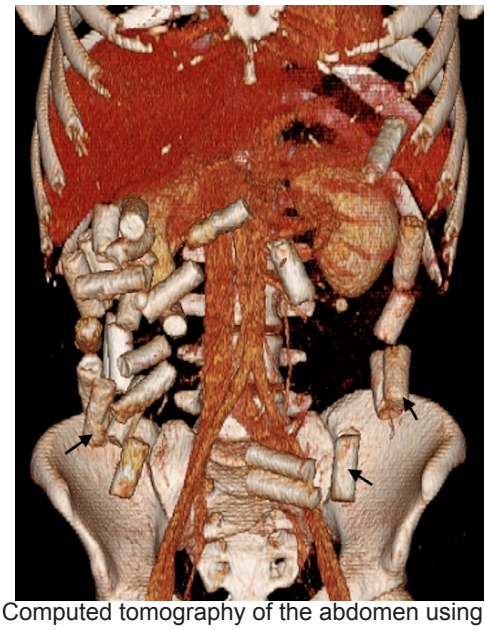

Image 1. Computed tomography of the abdomen using three-dimensional reconstruction showing extensive tubular foreign bodies present throughout the colon (arrows).

Image 2. Multiple tubular, plastic-wrapped packets of heroin subsequently passed by the patient.

specificity of 99% for packet detection, but their use is limited by reported sensitivities of 70%. Contrast CT imaging has a similar specificity but sensitivities of 99-100%.[3] The patient should be monitored closely in the ICU for signs of systemic toxicity and obstruction, and treated with whole bowel irrigation until clear rectal effluent is achieved with passage of all packets. Although most patients have an unremarkable hospital course, in cases of packet rupture, mortality rates of over 50% have been reported.[4] Management of symptomatic patients is based primarily on the type of substance ingested,

CPC-EM Capsule

What do we already know about this clinical entity?
Available literature describes body packing as a means to transport illicit drugs and discusses the best imaging modality to detect these illicit substances.

What is the major impact of the image(s)?
This image is novel in its demonstration of 3-dimensional computed tomography reconstruction imaging used to detect extensive tubular drug packets throughout the colon.

How might this improve emergency medicine practice?
The recognition and diagnosis of body packing in asymptomatic patients is important, as missed ingestion could lead to potentially fatal complications.

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

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Man With Bilateral Leg Swelling

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A 52-year-old man without known medical history presented with painful, progressive, bilateral lower extremity edema over a two-week period. An abdominal exam noted a firm left upper quadrant mass. Emergency department (ED) point-of-care ultrasound (POCUS) revealed a hyperechoic, heterogeneous structure in the inferior vena cava that was determined to represent a tumor thrombus extending from a primary renal cell carcinoma. This case demonstrates how POCUS was valuable in rapidly diagnosing this rare cause of lower extremity edema and its usefulness in directing the initial ED management of this patient. [Clin Pract Cases Emerg Med. 2019;3(4):451–452.]

CASE PRESENTATION

A 52-year-old man with no medical history presented to the emergency department (ED) with painful, progressive, bilateral lower extremity edema over a two-week period. The patient also had the following complaints: difficulty with ambulation; blood in his urine; dull, left lower quadrant abdominal pain for one month; and an 80-pound weight loss over two years. Vital signs were within normal limits. Palpation of the abdomen revealed a large, firm mass in the left upper quadrant and 2+ pitting edema in the bilateral lower extremities up to the mid-shin. Point-of-care ultrasound (POCUS) of the heart and lower extremities were unremarkable. Abdominal POCUS revealed a hyperechoic, heterogeneous structure in the inferior vena cava (IVC) (Image 1 and Video).

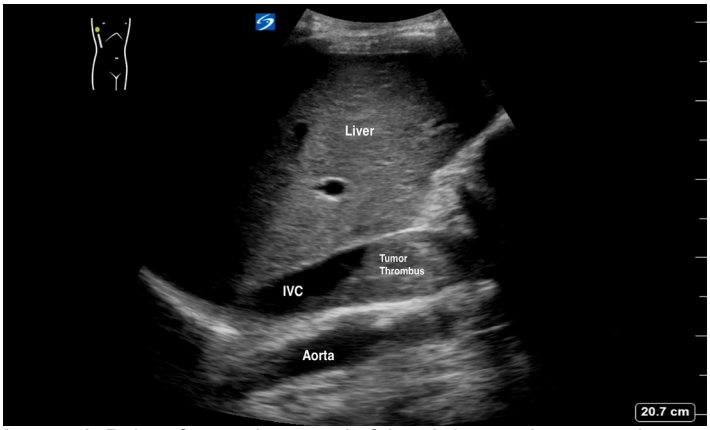

Image 1. Point-of-care ultrasound of the abdomen demonstrating a hyperechoic, heterogeneous structure in the inferior vena cava.

DISCUSSION

ED POCUS revealed a hyperechoic, heterogeneous structure in the IVC representing a tumor thrombus, which often results after intravascular extension of a primary tumor. This occurs most commonly in the setting of the following: renal cell carcinoma (RCC); hepatocellular carcinoma; Wilms tumor; and adrenal cortical carcinoma.[1] Computed tomography demonstrated continuity between a large heterogeneous left kidney mass and tumor thrombus in the IVC (Image 2). The patient was ultimately diagnosed with high-grade clear cell RCC with tumor thrombus extension into the IVC likely resulting in his bilateral leg swelling.

To date, no previous images in the literature have demonstrated POCUS identification of an IVC tumor thrombus in the ED. Radiology literature dating back to the 1980s attempted to characterize known IVC tumor thrombi with ultrasound.[2] In emergency medicine literature, two case reports describe the detection of IVC thrombosis during an ED POCUS exam, although neither was associated with extension from intra-abdominal malignancy.[3,4] In this case, POCUS allowed clinicians to quickly identify the likely etiology of this patient's lower

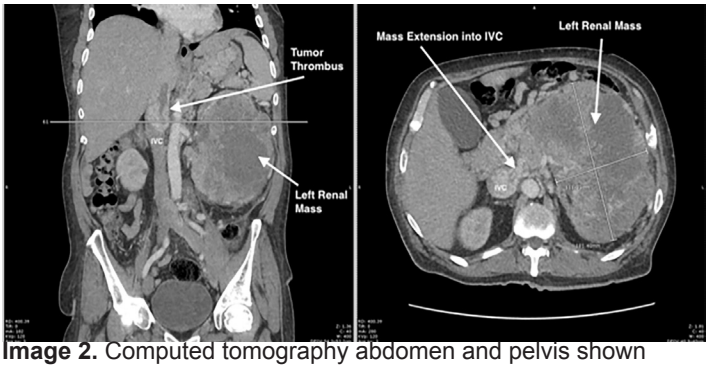

Image 2. Computed tomography abdomen and pelvis shown in the coronal (left) and axial (right) view demonstrating a large heterogeneous structure measuring approximately 20 x 14 x 19 centimeters and extending into the inferior vena cava.

extremity swelling, and guided initial clinical management and additional advanced imaging decisions.

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Video: Point-of-care ultrasound of the abdomen.

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

CPC-EM Capsule

What do we already know about this clinical entity?
No known reports to date have published images depicting an inferior vena cava (IVC) tumor thrombus detected on emergency department point-of-care ultrasound (POCUS).

What is the major impact of the image(s)?
This case describes how POCUS was used to rapidly identify an IVC tumor thrombus as the etiology of bilateral lower leg swelling, which in turn guided initial management.

How might this improve emergency medicine practice?
The case emphasizes that POCUS is an effective and versatile tool to make rapid diagnoses in the emergency department.

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Acute Auricular Perichondritis With an Effusion

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A 62-year-old man presented to the emergency department with acute, atraumatic, swelling of his left ear. Incision and drainage revealed serous fluid without blood or purulence. He was diagnosed with acute perichondritis with an effusion and managed with oral antibiotics. Perichondritis must be recognized and treated promptly to avoid necrosis of the underlying avascular cartilage and auricular deformity. [Clin Pract Cases Emerg Med. 2019;3(4):453–454.]

CASE PRESENTATION

A 62-year-old man presented to the emergency department with one day of painful left ear swelling. He denied preceding trauma or recent instrumentation, but expressed concern that he might have been bitten by a spider while cleaning cobwebs in his basement two days prior. He denied systemic symptoms. The area of swelling was tender and fluctuant with mild overlying erythema (Image 1). Point-of-care ultrasound revealed an avascular anechoic fluid collection within the cartilaginous layer of the ear (Image 2).

Incision and drainage revealed serous fluid without blood or purulence. This fluid was sent for culture, and the incisional wound was closed with non-absorbable sutures. A xeroform bolster was then sutured through-and-through the contours of the antihelix with gauze buttressed behind the ear to prevent formation of an auricular hematoma. The patient was

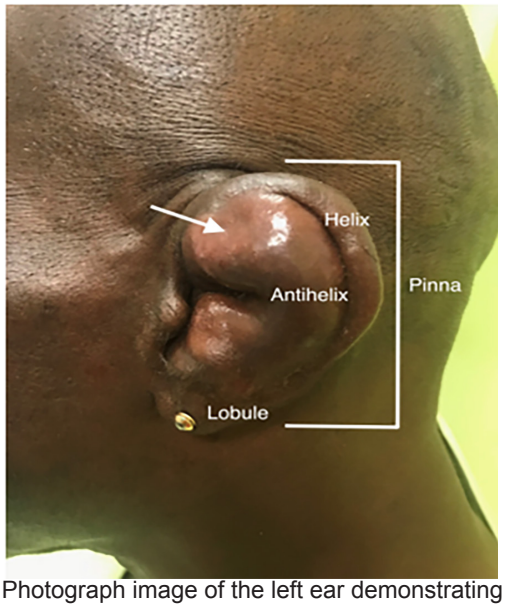

Image 1. Photograph image of the left ear demonstrating swelling of the pinna (arrow) with mild overlying erythema.

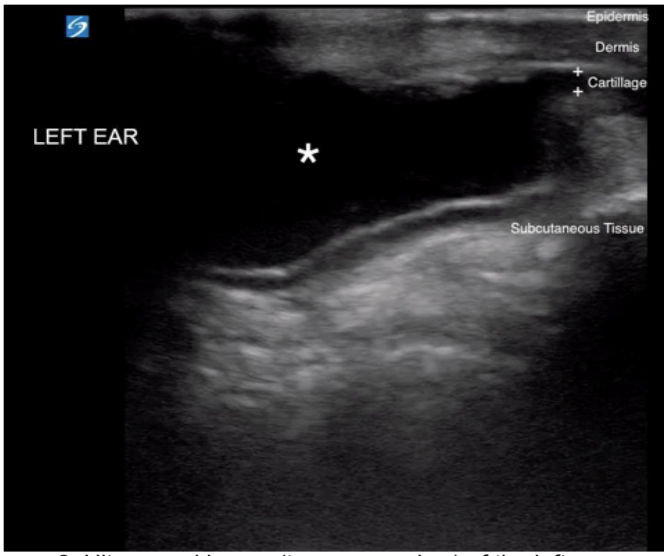

Image 2. Ultrasound image (transverse view) of the left ear demonstrating an anechoic fluid collection (asterisk) within the cartilaginous layer (plus sign) of the pinna.

diagnosed with acute auricular perichondritis with an effusion and discharged with amoxicillin-clavulanate.

The patient's culture grew methicillin-sensitive *Staphylococcus aureus*. He completed the course of antibiotics and had complete resolution of his symptoms and the fluid collection by his second otolaryngology follow-up appointment 14 days later.

DISCUSSION

Acute auricular perichondritis is an infection of the pinna that involves the cartilage and subcutaneous tissue but spares the lobule. Most cases of perichondritis result from minor trauma, often after piercings or insect bites. *Pseudomonas aeruginosa* is the most common organism isolated but *Staphylococcus aureus* can also be causative, primarily after piercings.[1-3] In the absence of infection, perichondritis – particularly recurrent perichondritis may herald underlying immunosuppression.[4,5]

Perichondritis must be recognized and treated promptly to avoid necrosis of the underlying avascular cartilage and auricular deformity, better known as cauliflower ear.[1,3] Treatment includes incision and drainage, auricular bolster placement and oral antibiotics with *Pseudomonas* coverage. All patients should follow up with otolaryngology for repeat wound evaluation and to ensure appropriate infection control.

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

CPC-EM Capsule

What do we already know about this clinical entity?
Acute auricular perichondritis is an infection of the external ear that may result in auricular deformity and requires treatment with incision and drainage followed by oral antibiotics.

What is the major impact of the image(s)?
In addition to the typical external exam findings of auricular perichondritis, the ultrasound image is a unique example of how an auricular effusion appears sonographically.

How might this improve emergency medicine practice?
Sonographic evaluation of suspected auricular perichondritis may serve as a diagnostic adjunct when investigating if an auricular infection has an associated effusion requiring drainage.

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