Clinical Practice and Cases in Emergency Medicine

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Clinical Practice and Cases in Emergency Medicine

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83-year-old Woman with a Fever and Emesis

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CASE PRESENTATION (DR. BIRCH)

An 83-year-old Filipino woman was brought to the emergency department (ED) by family for chief complaints of fever and emesis. Much of the patient's history and review of systems was obtained through her daughters, as the patient only spoke Tagalog and had severe dementia. The patient lives with her daughters who are her primary caregivers. The patient's husband is deceased. The daughters report that the patient was at her baseline mental status the night before. This morning they found her warm to the touch and obtained an oral temperature of 38.3° Celsius. She was constantly shifting her gaze about the room and appeared mildly distressed. She then had one episode of non-bloody, non-bilious emesis, so the daughters decided to bring her to the ED for evaluation.

The patient primarily communicates through hand gestures, incomprehensible sounds, or nonsensical words. She has not missed any doses of her medications, nor has she had any cough, urinary frequency or incontinence, any changes in her bowel habits, focal weakness, or rashes. Her daughters report no evidence of abdominal pain, dysuria, back or flank pain, chest pain, or lightheadedness.

The patient has a past medical history of hypertension, diabetes mellitus, myocardial infarction with stent placement, and hyperthyroidism. The patient is a retired teacher but has not worked in years. She has not had any surgeries. She does not drink alcohol, smoke cigarettes, or use illicit drugs. Her medication list includes glimepiride, metformin, gabapentin, pioglitazone, hydrochlorothiazide, lisinopril, aspirin, atorvastatin, ticagrelor, potassium supplements, methimazole, risperidone, trazadone, donepezil, escitalopram, and memantine. Her medicines are administered by her daughters and stored in a cabinet by her bed along with some salves, creams, and medicinal oils. The family reports that the patient has no known drug allergies.

Physical examination revealed a thin, frail, elderly Asian woman lying calmly on the stretcher. She had a temperature of 36.8° Celsius**,** a blood pressure of 185/85 millimeters of mercury, a pulse of 112 beats per minute, a respiratory rate of 36 breaths per minutes and an oxygenation saturation of 98% on room air. Her body mass index was 21. Her head was atraumatic and normocephalic. Her extra-ocular movements were intact and her pupils were four millimeters in diameter, round, equal, and reactive to light. Her oral mucosal membranes were moist. She was tachycardic on exam, but there were no audible murmurs, rubs or gallops. She had normal breath sounds bilaterally. She was clearly tachypneic and had mild subcostal retractions. Her abdomen was soft and normal bowels sounds were heard. She was not distended or tender, and she did not have any rebound, guarding or organomegaly. All four extremities were warm, well perfused and without any tenderness to palpation. Neurologically, she was not oriented to self, place, or time, and would only follow simple commands.

Laboratory results are shown in Table 1. Her electrocardiogram is shown in Image 1, and a chest radiograph (CXR) in Image 2.

CASE DISCUSSION (DR. DUBBS)

Geriatric patients with dementia are among the most challenging patients to treat in the ED. They present often with diffuse or non-specific complaints with broad differentials (such as fever or altered mental status). It can be easy to order a litany of labs and imaging, hoping that something comes up positive. Casting a wide net in this situation is not the pitfall; rather, it is encouraged. But the real challenge lies in maintaining a sense of diagnostic vigilance.

What is diagnostic vigilance? Diagnostic vigilance is not settling for a diagnosis of urinary tract infection in an altered 74-year-old woman with 5-10 white blood cells and 25-50 squamous cells per high-power field. Diagnostic vigilance is not settling for a diagnosis of dehydration in an 80-year-old man with acute kidney injury. The word *vigilant* is defined as "alertly watchful especially to avoid danger."[1] When we are presented with a patient who cannot provide much history, as in the case presented here, we must be alertly watchful, searching for clues

Table 1. Laboratory values for 83-year-old woman with fever and emesis.

Complete blood cell count	
	White blood cells 8.7 K/μL
	Hemoglobin 11.6 g/dL
	Hematocrit 35.2%
	Platelets 246 K/μL
Serum Chemistries	
	Sodium 148 mmol/L
	Potassium 4.1 mmol/L
	Chloride 106 mmol/L
	Bicarbonate 23 mmol/L
	Blood urea nitrogen 40 mg/dL
	Creatinine 1.41 mg/dL
	Glucose 106 mg/dL
	Calcium 10.8 mg/dL
	Magnesium 1.7 mg/dL
	Phosphorous 3.8 mg/dL
	Total Protein 8.6 g/dL
	Albumin 4.7 g/dL
	Aspartate Aminotransferase 30 u/L
	Alanine Aminotransferase 25 u/L
	Alkaline Phosphatase 96u/L
	Total Bilirubin 0.5 mg/dL
	Ammonia <9 mg/dL
Coagulation Profile	
	Prothrombin Time 14.0 seconds
	Partial Thromboplastin 28 seconds
	International Normalized Ratio 1.0
Urinalysis	
	Appearance Clear
	pH 7.0
	Ketones Negative
	Bilirubin Negative
	Protein Negative
	Nitrite Negative
	Red Blood Cells 26-50/HPF
	White Blood Cells 0-2/HPF
Venous Blood Gas	
	pH 7.36
	pCO2 30 mmHg
	pO2 35 mmHg
	HCO3 29 mEq/L
	HBO2 70%
	Base Excess -3 mmol/L
Lactate 2.1 mmol/L	

K/μL, kilogram per microliter; *g/dL*, grams per deciliter; *mmol/L*, millimoles per liter; *mg/dL*, milligrams per deciliter; *u/L,* units per liter; *HPF*, high power field; *mmHg*, millimeters of mercury; *mEq/L*, milliequivalents per liter.

in the history, the physical examination, tests, and re-evaluations to uncover the true diagnosis. So, let's approach this case of an 83-year-old woman presenting with fever and vomiting with some healthy diagnostic vigilance.

Dr. Birch paints a picture of a thin, frail, elderly Asian woman who is notably hypertensive, tachycardic, and tachypneic with a respiratory rate in the mid-thirties. I am pleasantly surprised, albeit slightly perplexed, that her oxygen saturation is 98% on room air and that her lungs are clear to auscultation (more on that later). Likewise, her soft and non-tender, non-peritoneal abdomen is reassuring in the light of her episode of vomiting prior to arrival. Very quickly, several life-threatening diagnoses such as heart failure with pulmonary edema, perforated viscous, and acute mesenteric ischemia fall away from the top of the differential list.

The patient's past medical history provides the all-important background on which to visualize our patient. This frail, elderly, retired teacher is on multiple medications for hypertension and coronary artery disease. She is treated with oral medications for diabetes and presumably diabetic neuropathy, takes methimazole for hyperthyroidism, and is on multiple anti-depressants, antipsychotics, and centrally active, Alzheimer's disease medications. On top of this polypharmacy, the patient appears to have access to medicinal oils and salves (medicinal ointment). Could her hyperthermia, hypertension, tachycardia, and altered mentation be due to thyroid storm? Could there be some kind of medication interaction or toxicity at play?

The patient seems to have a very attentive family that cares for her at home. They are clearly concerned about the fever and episode of vomiting that prompted the ED visit. Mention is also made of a change in her baseline mental status – appearing distressed compared to her usual self – which now adds the cognitive framework of altered mental status to the table.

The differential diagnosis for altered mental status is extensive. Initial assessment of patients with altered mental status should look for vital signs indicative of obvious causes such as hypoxia, hypercapnea, hypoperfusion from shock states, hypo- or hyperglycemia, and environmental hypo- or hyperthermia. When moving on to further investigation, it is helpful to categorize the possibilities into a few broad categories: primary neurologic, toxic/metabolic, infectious, and other causes (Table 2). Etiologies to be considered in the primary neurologic category are intracranial hemorrhage, stroke, and seizure. Toxic/metabolic considerations include alcohols and recreational drugs, medication toxicities, carbon monoxide, hyperglycemia, hypo- or hyperthyroidism, electrolyte abnormalities of sodium, potassium, calcium, or others, acidemia, and uremia. Any infection can cause altered mental status as well, especially in the elderly. It is important to consider meningitis, encephalitis, pneumonia, urinary tract infection, bacteremia and sepsis. Finally, psychiatric disorders should be diagnoses of exclusion after organic etiologies are ruled out.

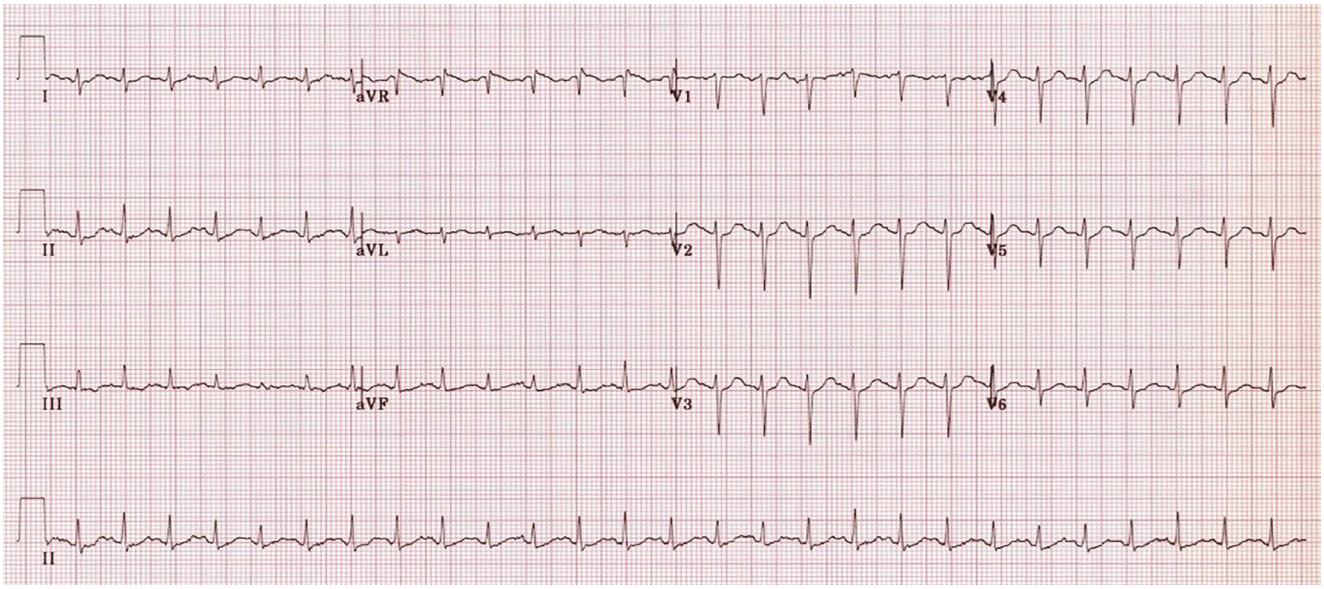

Image 1. Electrocardiogram of an 83-year-old woman with fever and emesis.

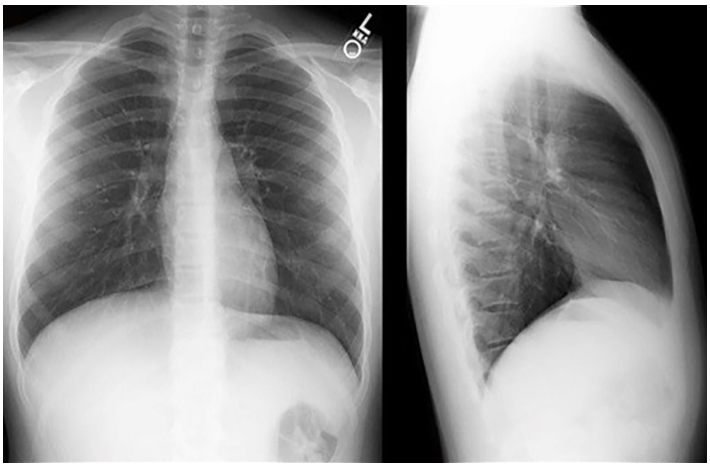

Image 2. Chest radiograph of 83-year-old woman with fever and emesis.

Now, let's take this broad, altered mental status differential diagnosis and apply the particulars of this patient. What are the pertinent positive and negative findings on her physical examination and work-up? For the initial assessment, we know that she is not hypoxic. She is confused but alert and has a high respiratory rate, so hypercapnea is not likely. Her level of alertness also makes hypoglycemia unlikely, but a point-of-care glucose would be helpful. Finally, her temperature is not indicative of hypo- or hyperthermia.

Do any of her findings point to a primary neurologic cause? She does not have focal neurologic deficits that would indicate an ischemic or hemorrhagic stroke. She is awake and able to follow simple commands, so seizure is not the diagnosis either.

Do any of her findings point to a primary toxic or metabolic cause? Alcohols could certainly be at play. She does have a small anion gap, which could be caused by a toxic alcohol such as methanol, ethylene glycol, or propylene glycol. Isopropanol presents with profound inebriation/coma, cerebellar signs, and hemorrhagic gastritis, which our patient does not have. Recreational drugs are not impossible, but are less likely given this patient's social situation; plus she does not have pupillary or other exam findings consistent with classic, drug-ingestion toxidromes. Carbon monoxide is also less likely, as it seems that family members, who reside with her, are asymptomatic. When it comes to medications, the patient has many that could be responsible for her altered mental status. Notably, her tachycardia, hypertension, vomiting, and altered mental status could be caused by escitalopram toxicity. Similar symptoms could be seen as well with thyrotoxicity if she stopped taking her methimazole. Aspirin toxicity may also present with the same tachycardia and tachypnea. Finally, labs effectively rule out major electrolyte abnormalities and uremia. In reviewing the venous blood gas for acidemia as a metabolic cause of the mental status, I find it to be surprisingly normal in light of the patient's slight anion gap metabolic acidosis and mildly elevated lactate of 2.1. Could there be a secondary process going on there?

Do any of her findings point to a primary infectious cause? With reported fever and the abnormal vital signs in this elderly patient, a sepsis workup is absolutely indicated. I would examine her for meningismus and strongly consider a lumbar puncture to look for central nervous system (CNS) infection if no more-convincing diagnoses are found. Her urine does not appear infected, and there are no signs of pneumonia on her chest radiograph. Could the vomiting be a sign of abdominal infection? Her abdominal exam is reassuring, but that is never

Table 2. Approach to altered mental status.

Initial Assessment
Hypoxia
Hypercapnea
Hypotension/Shock
Hypoglycemia
Hypothermia
Hyperthermia
Secondary assessment
Primary neurologic
Intracranial hemorrhage
Stroke
Seizure
Toxic/Metabolic
Alcohols (ethanol & toxic alcohols)
Recreational drugs
Medication toxicities
Carbon monoxide
Hypo- or hyperglycemia
Hypo- or hyperthyroidism
Electrolytes (sodium, potassium calcium, etc.)
Acidemia
Uremia
Infectious
Meningitis
Encephalitis
Pneumonia
Urinary tract infection
Bacteremia
Sepsis
Other
Psychiatric

guaranteed in the elderly population.

Do any of her findings point to a primary psychiatric cause? Again, psychiatric causes are a diagnosis of exclusion.

Several possible diagnoses came to the surface by going through the altered mental status differential. To review, these are toxic alcohols, escitalopram (selective serotonin reuptake inhibitor) toxicity, thyrotoxicosis, aspirin toxicity, intra-abdominal infection, and CNS infection.

I introduced the concept of diagnostic vigilance at the beginning of this section. Through the process of considering this patient's presentation within the framework of an altered mental status differential diagnosis, I strived to maintain that vigilance, remaining alertly watchful as would an investigator searching for clues at a crime scene or finding inconsistences in the stories of suspects. A couple aspects of the case were noticeably unexpected in this patient's presentation. I was surprised that she was not hypoxic and had clear lungs and a negative CXR despite being very tachypneic. I was surprised that her venous blood gas was within normal range when I expected to see acidemia from the anion gap and lactate. Could these two things that both seem like inconsistencies be made consistent, if related? Yes.

The patient has a primary anion gap metabolic acidosis, which is not reflected in the venous blood gas because she also has a primary respiratory alkalosis. One single diagnosis explains these findings, as well as her tachycardia, fever, and altered mental status. That diagnosis is aspirin overdose, also known as salicylate toxicity. The patient is on aspirin for her cardiovascular disease and has potential access to additional salicylate in the creams, salves, and oils stored at her bedside. Therefore, the diagnostic study that I would perform is a serum salicylate level to confirm the diagnosis of salicylate toxicity.

CASE OUTCOME (DR. BIRCH)

The patient was found to have a salicylate level of 45 milligrams per deciliter (mg/dL). Shortly after the level returned, another family member arrived with an empty bottle from the patient's medicine cabinet that had been full the day before (Image 3). Our patient had ingested approximately three-quarters of a 100-milliliter (ml) bottle of methyl salicylate camphor and methyl salicylate oil. This was calculated to be more than 18 grams of salicylate and the patient was diagnosed with acute salicylate poisoning. She was started on a bicarbonate drip and admitted to the hospital, where her peak salicylate level was found to be 53.7 mg/dL.

CASE DISCUSSION

In the 1960s and 1970s salicylate toxicity was the leading cause of fatal overdoses. Salicylate toxicity has declined since then, especially in children, due to increased public awareness about Reye's syndrome and child-resistant packaging.[2] Yet the entity is still very relevant to emergency medicine as over 24,000 salicylate overdoses occurred in 2014.[3]

The term "salicylates" refers to a large group of medications. For example, salicylic acid is a topical medication used to treat acne or to eliminate warts, but it is too irritating to the gastric mucosa to be ingested directly.[2] Methyl salicylate is a formulation that is almost always used topically to alleviate aches and pain. Salicylate containing compounds include oil of wintergreen (a topical oil solution), Bengay®, salicylic acid (a wart remover) and even Pepto Bismol®, (a common medicine for indigestion) which contains bismuth subsalicylate 262 mg per 15 ml. Luckily, salves are very poorly absorbed cutaneously, so it takes coating oneself in a copious amount of ointment to achieve toxic levels. However, there was a 2002 toxicology case report of a naturopath who used methyl salicylate to treat psoriasis, which caused systemic toxicity.[4] In this case absorption was enhanced due to the large body surface area used with an occlusive dressing.

Image 3. Empty container of Efficascent Oil, a topical pain-reliever containing camphor and methyl salicylate.

There are also combined medicines that include aspirin with other oral medicines such as Percodan® and Excedrin®.

Salicylates act through three main mechanisms: inhibition of the cyclo-oxygenase (COX) enzymes, stimulating chemoreceptors in the brain and alteration of cellular metabolism.[5] The COX-1 and COX-2 enzymes catalyze platelet aggregation and prostaglandin synthesis. Aspirin and related salicylates cause nausea and vomiting by inhibiting these enzymes, resulting in decreased prostaglandins, which protect the gastric mucosa.[6] COX inhibition also causes platelet dysfunction, which theoretically increases a patient's risk of bleeding, though this is rarely a clinical presentation of salicylate toxicity.[7] The nausea and vomiting caused by the decreased prostaglandin synthesis is augmented by stimulation of the chemoreceptor zone in the medulla.[8] Salicylates also activate the respiratory center, causing a respiratory alkalosis.[9] Aspirin and other salicylates uncouple oxidative phosphorylation, which causes a metabolic acidosis through an increase in lactic acid and organic acids.[10]

In its mild toxicity – levels of 40-60 mg/dL – there may be tinnitus caused by sensorineural deafness (from salicylates' neurotoxic effects) and possibly mild tachypnea, nausea and vomiting.[10,11] At moderately toxic levels of 60-80 mg/dL, the toxidrome presents with respiratory distress and CNS dysfunction, which can manifest as lethargy or, more commonly, agitation.[10] In addition to the directly stimulated tachypnea and hyperpnea, dyspnea may be caused by salicylate-induced pulmonary edema (SIPE). SIPE occurs due to the impaired permeability

In chronic toxicity, patients usually appear more ill, have fewer classic presentations and lower salicylate levels. Patients may appear to be septic, in diabetic ketoacidosis, or rarely in decompensated heart failure.[16,17] For such patients you should have a lower threshold to dialyze.

Understanding absorption and excretion of salicylates is key to understanding the clinical manifestations and treatment of their toxicity. Normally, therapeutic doses of aspirin are immediately absorbed in the stomach and converted to salicylate.[18] Approximately 90% of salicylate is metabolized through hepatic conjugation, which can be saturated, and only 10% of total elimination is achieved renally. At therapeutic doses, peak concentrations occur in one hour, with a half-life of two to four hours.[2] In contrast, large doses of salicylates can delay peak levels and delay absorption due to bezoar formation, enteric-coated or extended-release versions, and pylorospasm[19,20] Once hepatic mechanisms of metabolism are saturated, the rate-limiting step for excretion is the much slower renal route; peak levels may not occur for 30 hours.[21] Because of this, it is very important to repeat any positive level until it is downtrending twice to ensure the clinician does not miss a delayed peak level.

Non-polar molecules diffuse easily over cell membranes. Because salicylate is a weak acid, it can easily take an uncharged (pronated) form, allowing it to cross cellular barriers such as the blood-brain barrier or the renal tubule. When salicylate is charged (deprotonated), it stays in the plasma and is not able to damage organs as easily. The main determinant of whether salicylate is charged is the pH of its environment. Salicylates will become pronated (and thus uncharged) at lower pHs, which is part of the toxicity itself. Once the metabolic acidosis develops, more of the salicylate is in its protonated form and able to create significant toxicity.[22] This principle is important for understanding both why we order certain labs and how we treat salicylate toxicity.

Important labs to order while evaluating someone with possible salicylate toxicity are a salicylate level and a blood gas, which allow assessment of the toxicity severity and response to therapy. It is also important to measure potassium to monitor for hypokalemia, which would interfere with one of the mainstays of therapy: alkalinization of the urine. Hypokalemia promotes the resorption of potassium in the distal tubule via a potassium-hydrogen ion pump.[22] As the kidney tries to retain potassium, the urine becomes more acidotic and does not favor urinary excretion of the salicylate. Glucose is important to measure because neuroglycopenia can happen at normal glucose level,[14,15] and it is preferable for a toxic patient to be normo- to hyperglycemic.

Other important studies include creatinine to evaluate for kidney function, coagulation studies to assess liver function, anion gap, and imaging (CXR and head computed tomography) to determine other possible causes of the patient's presentation.

When treating these patients we must first, of course, address the patient's airway and breathing. The most important principle to remember regarding intubation in salicylate toxicity is to avoid it if at all possible.[23] In even the small amount of time the patient is apneic, respiratory acidosis results and more of the salicylate is protonated, allowing it to enter the CNS. When addressing the patient's circulation, the clinician must realize that many patients with salicylate toxicity will be dehydrated due to vomiting and insensible losses secondary to their tachypnea.[10] Patients will likely require aggressive fluid resuscitation. This should only be delayed in cases of cerebral or pulmonary edema.

The main principles of treatment after attending to airway, breathing, and circulation are decontamination and alkalization of the blood and urine.[24] Immediately decontaminating with one gram/kilogram of activated charcoal, orally, up to 50 grams is a critical first step.[25] Ideally, this should be given within two hours of ingestion; however, it should always be given due to the possibility of bezoar formation, enteric-coated tablets, and pylorospasm.[2] After the initial dose, activated charcoal can be given 25 grams orally every two hours for three doses or 50 grams orally every four hours for two doses.

As discussed previously, the charged form of salicylates will more readily stay in the plasma and be amenable to excretion, whereas the uncharged form will more easily cross into cells to interfere with metabolism and the brain and cause damage. Because the ion will retain its uncharged form in an alkaline environment, the mainstay of treatment is to "trap" the salicylate ion in the plasma and urine by alkalizing them. Alkalization is achieved with a bolus (1-2 milliequivalents per kilogram [meq/kg]) and infusion (100-150 meq in dextrose 5% sterile water) of sodium bicarbonate with titration to a urine pH of 7.5 to 8.[22] As previously discussed, potassium levels must be monitored and normokalemia maintained to effectively keep the urine basic. Urine alkalization to a pH of 7.5-8.0 increases urinary excretion of salicylates significantly.[26] It is important to also monitor blood gases in order to avoid severe alkalemia (arterial pH >7.6). Indications for hemodialysis include volume overload preventing sodium bicarbonate therapy, pulmonary edema, cerebral edema, kidney failure that impairs salicylate elimination, salicylate levels over 80 mg/dL, and severe acidemia.[10] Consultation with nephrology is essential even when patients do not appear to immediately require hemodialysis, as it will help facilitate treatment in case the patient decompensates.

The importance of this elusive diagnosis is highlighted by the reflections of two physicians:

"Twenty-five years ago, a lady jumped off a bridge, and survived her trauma. She was admitted and appeared to go into sepsis with high fever but later died of an aspirin overdose which had not been checked for. She was trying to end her life for sure and it worked."[27]

"I had an unfortunate lady a few years ago who presented in status epilepticus. Airway obtained early with Ativan[™] for seizures, blood gas obtained with pH around 7.0. Boyfriend provided history of missing bottle of aspirin as she proceeded to arrest."[28]

FINAL DIAGNOSIS

Acute salicylate toxicity.

KEY TEACHING POINTS

1. Cast a wide diagnostic net including ingestion when confronted with elderly patients, especially those with dementia.
2. When clinically suspicious of salicylate toxicity, do not accept a "therapeutic level;" always repeat until the level is declining for two serial values.
3. Salicylate toxicity causes fever, metabolic acidosis and respiratory alkalosis by uncoupling oxidative phosphorylation and stimulating the respiratory centers in the medulla.
4. Decontamination with charcoal should be initiated rapidly, and intubation should be avoided as long as possible.
5. Keep the patient hyperglycemic, as the CNS may be hypoglycemic, and alkalize the patient's serum and urine with sodium bicarbonate.
6. Speak to nephrology early in the patient's care because dialysis can be lifesaving.

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 Pair of Testicular Torsion Medicolegal Cases with Caveats: The Ball's in Your Court

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In this article, we present two medicolegal cases illustrating medical and diagnostic pitfalls that can lead to litigation for missed testicular torsion. Testicular torsion (TT) is a urologic emergency with potentially devastating consequences and costs, for providers and patients alike. TT occurs in approximately 4.5 per 100,000 males under the age of 25. While uncommon, TT is the third most common cause of medical malpractice suits in this demographic. As a consequence of varying presentations and physical exam findings, and diagnostic imaging subject to individual interpretation, this time-sensitive diagnosis may be missed by emergency department providers. Delays in diagnosis significantly increases the morbidity associated with TT, and 31.9%-41.9% of such cases result in testicular loss. The average reported settlement for TT malpractice litigation is $60,000. This article discusses two actual malpractice cases involving TT and provides insight and caveats to ensure an optimal evaluation and diagnostic approach to this often-elusive condition. [Clin Pract Cases Emerg Med. 2018;2(4):283–285.]

CASE 1: Anonymous v. Anonymous

A 16-year-old male arrived at the emergency department (ED) complaining of right lower quadrant abdominal pain with some associated nausea and vomiting. The emergency physician (EP) completed an abdominal exam, obtained labs, an abdominal ultrasound, and a computed tomography (CT) of the abdomen and pelvis. These were all unremarkable. Nevertheless, a surgical consultation was obtained to further evaluate for appendicitis. The surgeon did not feel appendicitis was present, and the patient was discharged. A genital exam was never performed. The following day, the patient returned with right testicular pain. He was immediately taken to the operating room for scrotal exploration and required a right orchiectomy. A lawsuit was initiated for failure to perform a genital exam, and failure to consider testicular torsion (TT) in the diagnosis. Before trial a settlement of $300,000 was reached.[3] Isolated abdominal pain is a frequent chief complaint associated with TT, and one review found that failure to complete a testicular exam was associated with 19% of TT malpractice cases.[2] It is imperative to consider this diagnosis whenever lower abdominal pain is present and complete a scrotal exam.

CASE 2: Graham v. Noreldin

A 14-year-old male was taken to the ED after awakening with abdominal pain. Laboratory studies, an abdominal CT, and a scrotal ultrasound were done. The CT was read as suggestive of appendicitis and thus a surgical consultation was obtained. The surgeon did not feel that appendicitis was present. The radiologist reviewed the ultrasound and diagnosed epididymitis. Based on the studies the EP discharged the patient on antibiotics. Three days later the patient awoke with testicle pain and was taken to a different ED where he was diagnosed with TT and received an orchiectomy. A review of the original ultrasound revealed there was decreased blood flow to the testicle. The patient litigated claiming that the diagnosis should have been made on the first visit and the testicle could have been salvaged. The case was solely against the EP and not the radiologist. There was testimony from the EP that he had ordered the "gold standard" test and relied on the interpretation by radiology. After trial, the jury awarded a $500,000 verdict.[4] This case is typical of others. When a radiologist misreads the testicular ultrasound, often the radiologist pays out less than the EP, or the EP pays out alone. The thought process was that

the EP had the ability to make a "clinical correlation" that the radiologist could not make.

DISCUSSION

Dr. Bass

There is no standard presentation for TT. Testicular torsion presentation can present similarly to epididymitis. A significant number of proven TT cases present with gradual onset discomfort, whereas alternative causes of scrotal pain, such as epididymitis, can present with sudden discomfort in up to 51% of cases.[1] Finally, circumstances surrounding the presentation may not reveal the ultimate diagnosis. TT is attributed to direct trauma in 4-8% of reported cases, and more frequently occurs during sleep, as a result of spontaneous cremasteric contractions.[5] Since there is a wide variety and overlap of symptoms and circumstances surrounding TT, it is imperative to not rely on historical features alone to guide further evaluation.

EPs should be hesitant to decide the absence (or presence) of TT based solely on clinical exam. Presence or absence of cremasteric reflexes, scrotal edema/erythema, pain along the upper pole of the testicle or epididymis, enlarged epididymis, transverse lie, Prehn's sign (pain relief with examiner lifting testicle), and retraction of testicle all fail to give a definitive answer.[1] Even when experienced urologists combine all these exam findings their initial impressions are frequently in error.[1]

The presence of a cremasteric reflex has historically been touted to rule out TT. This unfortunately is not completely true. Several case series, although mostly small, have reported TT with intact cremasteric reflexes.[1] Specifically, patients who were later diagnosed with TT had intact cremasteric reflexes in 12%-40% of cases.[1] Cremasteric reflex cannot be relied on. Additionally, cremasteric reflexes are absent in 30% of males with normal testicles.[1] Isolated pain along the upper pole of the testicle or epididymis has been reported to occur in 18.7% of patients with TT and 40.8% of patients with torsion of the testicular appendage.[1] A transverse testicular lie has been reported in 17% to 83% of TT cases, while a vertical lie has been observed in up to 54% of cases of TT.[1] Lastly, testicular retraction (high-riding testicle) is only present in 33%-80% of TT cases.[1]

Dr. Couperus

A scrotal ultrasound, the "gold standard" test, can be very helpful, although it is not foolproof. Lawyers will argue that "one simple and available test" could have been ordered and made the diagnosis. However, upon review of cases involved in litigation, we found that obtaining an ultrasound did not correlate with a more successful defense.[2,6] This is because a scrotal ultrasound can be misread as normal by radiologists.[2,6] In general, high resolution ultrasonography has a sensitivity of 96% but is not perfect.[6] If a negative ultrasound is reported, in the situation that a high clinical suspicion remains, a urologist should be consulted. Involving a consultant has historically created a very defensible position.[6]

The time window for possible salvage and survival of a torsed testicle is commonly thought to be 6-8 hours.[7] Recently, a review of 30 articles, with over 2,116 patients included, looked at outcomes related to time of torsion. When reported in six-hour intervals (1,283 patients), survival at 0-6 hours was 97.2%; 7-12 hours, 79.3%; 13-18 hours, 61.3%; 19-24 hours, 42.5%; 25-48 hours, 24.4%; and greater than 48 hours, 7.4%. Cumulative testicular survival data based on reporting for all three groups of patients were as follows: testicular salvage in the first 12 hours is 90.4%; from 13-24 hours survival is 54.0%; and beyond 24 hours survival is 18.1%. Vigilant urgency is prudent irrespective of the time that symptoms have been present when TT is a consideration.[7]

MEDICOLEGAL ISSUES

Dr. Pfaff

A review of jury verdict reports in cases of TT was done to identify causes of litigation and factors contributing to verdicts or settlements.[1] This review examined 52 pertinent case outcomes in which 51% resulted in favor of the physician and 49% in indemnity payment. EPs were the most commonly sued medical providers (48% of defendants), followed by urologists at 23%, and were significantly more likely to make indemnity payments than urologists. The majority of malpractice claims were failure of diagnosis (96%). Misdiagnosis of epididymitis was noted in 27 cases (65%).[8] A retrospective review of TT malpractice cases from 1985 to 2015 reported similar findings in 53 relevant cases, 88% with testicular loss.[2] Again, EPs were the most common type of provider sued (35%) followed by family physicians (17%), and urologists (13%). However, specialty was not shown to be associated with successful defense. Most claims for malpractice included missed diagnosis and negligence (98%). Half of providers diagnosed patients with epididymitis on first presentation (52%). Atypical presentation (31% with abdominal pain only) and failure to complete a testicular exam was associated with 19% of TT malpractice cases.[8] False negative ultrasound findings were common among these cases. When a radiologist misreads the testicular ultrasound, often the radiologist pays out less than the EP, or the EP pays out alone. The thought process is that the EP had the ability to make a "clinical correlation" that the radiologist didn't.[2]

Dr. Moore

The sudden onset of severe, unrelenting testicular pain is typically held to be diagnostic of TT. This is not the case, however, for a small but significant number of patients with a torsed testicle. The little-recognized fact that TT patients can present with minimal or no pain has proven to be a medicolegal pitfall for EPs.[2] A subset of TT patients reports

resolution of their initial severe pain followed by variable periods of hours to days of reduced or absent pain. Other patients report only mild pain described as gradual in onset. These "pain honeymoons" may be partially responsible for poor clinical outcomes because of delayed initial presentations or less-than-timely returns for secondary evaluation. The pain relief experienced by some patients with TT has been likened to an extremity paresthesia that develops after prolonged nerve compression. The pain again begins to worsen, and secondary scrotal inflammation and pain occur as inflammatory factors increase with infarction of the testicle.

A recent article highlights seven cases of TT and raises a serious liability concern. In all of these patients, there was a period of freedom from pain, or much decreased pain after the initial onset of symptoms ("pain honeymoon"). The diagnosis would be very easy to miss in this clinical scenario. The mechanism is thought to be one of compression of the nerves as they travel in the spermatic the cord with resultant paresthesia and anesthesia.[7]

CONCLUSION

Missed TT is a frequent source of successful litigation against EPs. There are many traditional paradigms in the areas of history, physical exam, imaging studies, and clinical course that can lead to diagnostic failure. Given these clinical uncertainties and high risk for testicle loss, EPs should routinely document a scrotal exam for young males with lower abdominal pain, have a low threshold for ultrasound imaging with any reasonable suspicion, and use a liberal threshold for urological consultation, if available.

TAKE HOME POINTS

1. Successful litigation for testicular torsion often occurs due to failure to do a genital exam in patients with abdominal pain.
2. Successful litigation for TT often occurs by a failure of the radiologist to notice pathology on scrotal ultrasound. Nevertheless, the EP is held responsible. A urologist should be involved when there is high clinical suspicion for TT in the face of a "negative" ultrasound.
3. Testicular salvage after 24 hours of torsion is still 18%, and physicians should aggressively pursue the diagnosis even in a delayed presentation.
4. The overwhelming majority of malpractice claims were failure of diagnosis, and 2/3 of these cases were diagnosed as epididymitis.
5. Recent reports described cases of "honeymoon" absence of pain in TT. The improvement of testicular pain or its absence after initial onset should *not* reassure the provider that the diagnosis is not likely.

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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Fascia Iliaca Compartment Block Efficacy in Resource-poor Emergency Departments

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Introduction: Although the fascia iliaca compartment block (FICB) seems to be an ideal technique for femoral neck and shaft fractures occurring in resource-poor settings, it has been unclear how effective it is when used by emergency physicians (EP) with little training in the technique, using equipment, medications and methods that differ from those commonly available in developed countries. This series was designed to demonstrate that EPs in a resource-poor setting can provide effective analgesia for femur fractures with anatomic landmark-guided FICBs, clinician-compounded lidocaine-epinephrine (1:100,000), and a standard injection needle.

Methods: Over a three-month period, patients ≥12 years old presenting to the emergency department with hip or femur fractures and a Likert visual analogue scale >4 had an EP-administered FICB. EPs used a standard intramuscular needle and a lidocaine-epinephrine solution they compounded at the bedside and located the injection site using only anatomic landmarks. EPs evaluated the patient's pain level at 30 minutes and at two hours post-FICB. We also reviewed articles since 2016 that describe the FICB.

Results: We enrolled a non-consecutive sample of 10 patients in the case series. Five had femoral neck (hip) fractures and five had femoral shaft fractures. All patients had a reduction in their pain levels after the FICB. On average, the block took effect about three minutes after injection. At 30 minutes all patients reported clinically meaningful pain reduction. The analgesic effect of the compounded agent lasted approximately 200 minutes. No adverse effects were reported. No published journal articles about FICB since 2016 were from resource-poor settings, and only one was from a developing country.

Conclusion: This series suggests that the FICB is effective even when performed with the minimal materials that are usually available in resource-poor settings. Methods such as this, which use simplified clinical tests and techniques applicable in resource-poor settings, can assist global emergency medicine (EM). We can assist global EM by similarly finding methods to simplify useful clinical tests and techniques that can be used in resource-poor settings. [Clin Pract Cases Emerg Med. 2018;2(4):286–290.]

INTRODUCTION

Patients with femur and hip fractures commonly present to emergency departments (ED). Promptly providing analgesia reduces the significant physical and psychological effects on patients. It speeds their operative management, lessens the length of hospitalization,[1] and decreases opiate-induced complications, especially in the elderly.[2] Yet only 2% of elderly patients with fractures receive adequate analgesia.[1]

Building on Sharrock's accidental discovery of the fascia-iliaca compartment block (FICB) in 1980,[3] BJ Dalens[4]

popularized its use and Godoy-Monzon[5,17] introduced it into the emergency medicine (EM) literature in 2007. The FICB offers a safe, inexpensive, rapid and effective analgesic for patients with hip fractures.[5] A single injection of anesthetic solution into the fascia iliaca space spreads to block the femoral, lateral femoral cutaneous, and obturator nerves, providing anesthesia to the hip, femur, and, often, the knee.[6,7] The FICB's safety stems from the needle insertion site's location being distant from the femoral triangle (artery, vein, and nerve), which minimizes the risk of intravascular injection and nerve injury.[1, 6]

While numerous publications have demonstrated the block's efficacy in relieving pain for patients with acute femur fractures, the methods relied on equipment and medications not available in most facilities outside the developed world and few have originated from resource-poor facilities in developing countries. Resource-poor settings, which often have only parenteral opiates to provide analgesia after acute fractures, are where FICBs may prove most useful. This paper reviews recent publications that describe FICB settings and techniques and uses a case series from a resource-poor hospital ED in a developing country to demonstrate the block's efficacy using basic equipment, inexpensive and readily available medications, and novice practitioners.

CPC-EM Capsule

What do we already know about this procedure?
Fascia Iliaca Compartment Blocks (FICB) are ideal for providing analgesia to emergency department (ED) patients with femoral fractures but are rarely used in resource-poor settings.

What makes this case series reportable?
Emergency physicians in a resource-poor setting successfully did FICBs using only anatomical markers, clinician-compounded analgesic and standard injection needles.

What is the major learning point?
FICB blocks can be successfully performed (clinically meaningful pain reduction within 30 minutes) with the minimal equipment and materials present in any ED.

How might this improve emergency medicine practice?
This study (the first from a developing country since at least 2016) should encourage clinicians in similar settings to use the FICB despite lacking high-level resources.

METHODS

Setting and Population

The convenience study sample was patients ≥12 years old with moderately and severely painful (4/10 on the Likert visual analogue [VAS] pain scale) injuries to the hip and femur. Patients were excluded if they had a history of allergies to local anesthetics; a history of femoral bypass surgery; signs of an infection at the FICB injection site; a bleeding diathesis (known history or currently evident); or a history of second- or third-degree heart block. EM faculty and resident physicians performed all the blocks. The Medical Advisory Council (equivalent to an institutional review board [IRB]) approved this study. All participants or their surrogate decision-makers provided informed consent.

Protocol

Prior to the study, participating emergency physicians (EP) attended a short teaching session, received a printed protocol with anatomical illustrations, and were shown the procedure for compounding the lidocaine-epinephrine mixture. After identifying appropriate patients with a VAS pain scale ≥4, the EPs obtained informed consent. They then completed a neurovascular examination of the affected limb and prepared the lidocaine-epinephrine 1:100,000 solution (1 milligram [mg] =1 milliliter [mL] 1:1000 epinephrine/100 mL lidocaine). Compounding the solution at the bedside, they added 0.2 mL of epinephrine (1:1000) to 20 mL of lidocaine 1% (10 mg/mL). They aspirated the mixture into a sterile syringe attached to a 21-gauge, 2-inch intramuscular injection needle.[5,8]

With the patient supine, the inguinal ligament was identified, the femoral artery palpated, and the skin cleaned with iodopovidone. The needle was inserted perpendicular to the skin at a point one centimeter (cm) below the juncture of the lateral and medial 2/3 of a line from the pubic tubercle to the anterior superior iliac spine (Image). Using the loss-of-resistance method, the needle was inserted until a loss of resistance was first felt as it passed the fascia lata and again when the fascia iliaca was pierced (often described as two "pops"). After aspirating to exclude intravascular placement, the mixture was injected into the fascia iliaca compartment. All patients received a standard 20 mL dose (200 mg lidocaine) of this mixture. Each patient's pain level was re-evaluated 30 minutes and two hours after FICB administration.

Literature Review

We searched PubMed for publication dates between 2016 and mid-May 2018 using the following search strategy: "Best Matches" for "fascia iliaca block" OR "iliofascial block": ["fascia iliaca block"[All Fields] OR (iliofascial [All Fields] AND block [All Fields])]. The papers were then culled for non-review papers

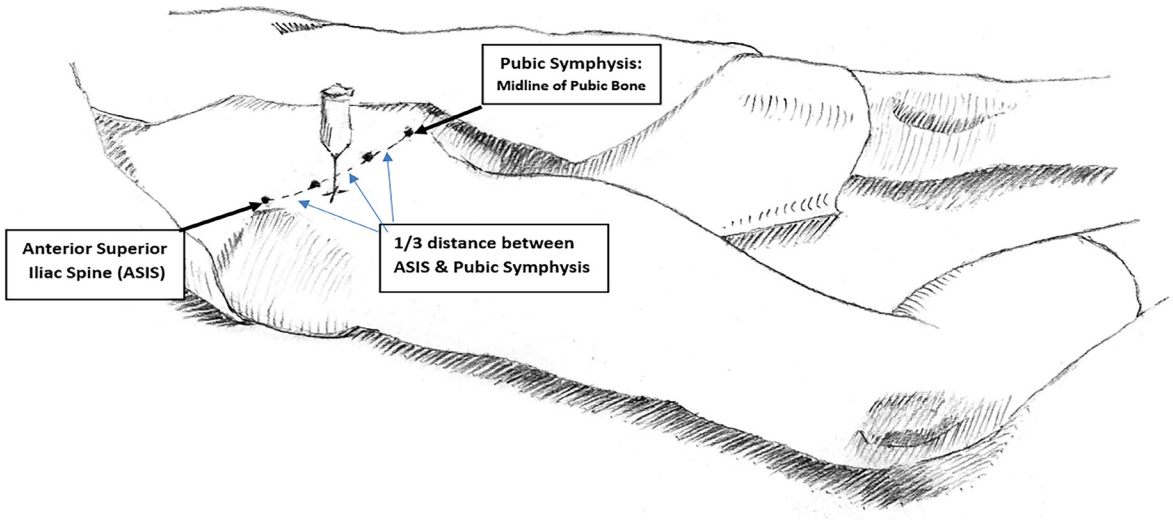

Image. Anatomic landmarks for fascia-iliaca compartment block needle placement: one centimeter inferior to the point on a line joining the anterior superior iliac spine (ASIS) and the pubic symphysis.

in English that had information describing their setting and preoperative-analgesia FICB protocol using a single (non-continuous catheter) injection for acute femur fractures.

RESULTS

Ten patients aged 12 to 93 years (median 59 years) had FICBs using bedside-compounded lidocaine-epinephrine. Five patients were males; five had femoral neck and five had femoral shaft fractures. Seven EPs performed the blocks: five residents and two faculty. All seven initially performed the block for the first time. Medication preparation and block performance generally took less than five minutes. Repeat pain levels were measured at 30 minutes and 120 minutes post-block. All patients experienced clinically relevant pain reduction post-block at both time points. VAS pain scores averaged 7.1 (range 5-9) on presentation, 2.7 (range 0-4) at 30 minutes post-block, and 0.6 (range 0-2) at 120 minutes. The average duration of analgesia was 200 minutes. No adverse events were reported from the blocks. Most study patients were admitted to the operating room (OR) or ward before the block lost effect. For the few who remained in the ED, an additional FICB was administered. One patient refused to be transferred to the ward before he received a second FICB.

We identified 10 articles using the PubMed search. One was in Chinese, one described a femoral nerve block, and one had insufficient information. The Table contrasts the remaining articles with the current case series.

DISCUSSION

Since 2016, all journal articles describing the use the FICB for analgesia after acute fractures have originated from resource-adequate facilities; all but one are from developed countries. In contrast, the current case series is from a resource-poor teaching hospital in a developing country, a situation in which the FICB is particularly useful. Resource-poor settings have less monitoring available for patients who have been given mind-altering analgesics, surgery may be significantly delayed due to a lack of staff or equipment, and inpatient admission delays may require EPs to repeatedly administer analgesia over several days. Yet clinicians in these facilities may hesitate to do FICBs without the equipment and medications most commonly described for the procedure. We conducted this case series to demonstrate that the FICB can be performed successfully with limited equipment, medication, and training.

Reviewed Cases

Of the seven PubMed articles that met the criteria, only one was from a developing country.[9] Based on website descriptions, none would be considered a resource-poor facility. In contrast, Guyana is a developing country and the ED routinely lacks basic medication and equipment, including those typically described to perform the FICB.

Ultrasound Use

The loss-of-resistance FICB using anatomic landmarks does not require ultrasound guidance, since its safety relies on the distance of the thigh's major neurovascular structures from the injection site. Neither the study that first described the FICB nor the initial studies demonstrating its utility for acute fractures in EDs used ultrasound guidance, although they had high success rates.[4,5,17] Despite that, many publications that describe the procedure now advocate the use of ultrasound (or neurostimulators in the OR), with three of the recent articles describing its use. While our ED has an ultrasound machine, it

Table. Comparison of current case series with recent (2016-2018) publications.

Reference number	Developing country?[9]	Resource-poor conditions?	Ultrasound?	Tuohy (epidural) needle?	Compounded anesthetic?	ED/EP?
10	Spain (NO)	NO	NO	Tuohy	Levobupivacaine 0.25% 0.3mL/Kg	YES/NO
11	USA (NO)	NO	YES	NS (Not specified)	YES. 0.5% ropivacaine + 2% lidocaine w epi	OR(Operating room)/NO
12	Bangalore, INDIA (YES)	NO	NO	Tuohy	0.375% ropivacaine; 30mL	OR/NO
13	Portugal (NO)	NO	YES	NO (Echoplex echogenic needle)	YES. 1.3% mepivacaine + 0.5% ropivacaine	OR/NO
14	UK (NO)	NO	NO	NO (Spinal needle)	Levobupivacaine 0.25%; 30 or 40 mL	YES/NO
15	UK (NO)	NO	NO	Tuohy	Levobupivacaine 0.25% + saline	YES/YES
16	Romania (NO)	NO	YES	NS (Not specified)	≤40 mL 0.5% Ropivacaine	YES/YES
	[This Series' Country] (YES)	YES	NO	NO	1% Lidocaine + Epinephrine	YES/YES

ED, emergency department; *EP*, emergency physician; *USA*, United States of America; *UK*, United Kingdom; *mL*, milliliter; *Kg*, kilogram.

does not have the linear transducer commonly recommended for use with the FICB. We therefore used the anatomic landmark, loss-of-resistance (or "two-pop") technique for our blocks.

Needle

A common recommendation for performing the FICB is to use a blunt 21G or a Tuohy (slightly curved epidural) needle to more easily recognize passage through the tissue planes. Three of five articles described using a Tuohy needle, while another used an echogenic needle with ultrasound guidance. In our setting, neither recommended needle was available. We successfully used an intramuscular injection needle.

Medication

As the reviewed articles demonstrated, several long-acting local anesthetics work well for single-injection (as opposed to catheter-infusion) FICBs. Cost and logistics prevent our ED from stocking anything but plain lidocaine. To produce long-acting anesthesia, we added epinephrine to lidocaine, often doubling its anesthetic duration.[18] For simplicity and safety, all patients received a standard 20mL dose (200mg lidocaine) of this mixture, which is less than the average maximum dose for a 12-year-old child (41kg=287mg), for an average adult (70kg=490mg), and for an elderly or renal-hepatic compromised patient (55kg=385mg/2=193mg).

Expertise, Specialty and Location

No inexperienced clinicians in the reviewed publications performed FICBs, and only two had EPs doing the block. As in our setting, clinicians in resource-poor EDs often must perform novel techniques based on written, oral, or video descriptions. The blocks performed by our ED staff and resident physicians were successful, although they had learned the procedure using written and oral descriptions.

LIMITATIONS

This study had two major limitations: only 10 patients met the study criteria; and the IRB did not permit sham injections for a control group. Nevertheless, the high success rate our EPs had with the FICB using inexpensive and readily available materials is consistent with the results in studies with the normally recommended equipment and medications.

CONCLUSION

EM faculty and residents in a resource-poor ED successfully reduced the pain from femoral fractures by performing FICBs using only anatomic landmarks, physician-compounded lidocaine-epinephrine, and a standard injection needle. This demonstrates that physicians in resource-poor settings can safely and quickly perform the FICB with little training, using the equipment and medications they typically have available. Using the FICB in resource-poor, often rural settings also reduces risks related to opioid use and provides an excellent method to ease the pain of patients transferring to higher levels of care.

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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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Threatened Respiratory Compromise in the Setting of Isolated Angioedema

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Isolated angioedema of the uvula, or Quincke's disease, is a rare condition that can cause respiratory compromise. Although typically self-limiting, episodes of angioedema may require prompt therapy to prevent obstruction of the proximal airway. In this case report we review the appropriate steps for initial evaluation of patients with suspected angioedema, primary etiologies, and appropriate initial therapy. [Clin Pract Cases Emerg Med. 2018;2(4):291–293.]

INTRODUCTION

We present a patient with isolated angioedema of the uvula via a likely mechanism of drug-induced histaminergic release vs. direct thermal insult. Although histaminergic angioedema rarely causes respiratory compromise, we report here how mechanical obstruction can create a life-threatening emergency even without associated signs of anaphylaxis. The patient responded well to antihistamine medications and steroids, and was discharged after resolution of symptoms.

CASE REPORT

A 55-year-old male presented to the emergency department (ED) complaining of one hour of difficulty breathing that woke him from sleep. Symptoms worsened when lying down on his left side. He endorsed a mild sore throat that was felt in the oropharynx; however, he spoke in a normal tone of voice and denied any difficulty swallowing, fever, nausea, vomiting, diarrhea, cough, or previous neck surgery or radiation. He denied history of food allergies, drug allergies, or reaction to toxic insults. He denied any recent changes to diet or travel. He was employed as a mechanic but denied prolonged exposure to exhaust or working without appropriate protective equipment. He admitted to frequent methamphetamine smoking, most recently the evening before presenting to the ED. He denied any current medications, previous exposure to angiotensin-converting-enzyme (ACE) inhibitors, or previous adverse reaction to nonsteroidal anti-inflammatory drugs (NSAIDs). He denied previous diagnosis of lymphoproliferative disorders or family history of angioedema.

Upon arrival to the ED, the patient had the following vital signs: blood pressure 141/93 millimeters of mercury, temperature 98.8º F, heart rate 86 beats per minute, respiratory rate 16 breaths per minute, and oxygen saturation 99% on room air. On physical exam his lungs were clear to auscultation bilaterally, without vesicular breath sounds and no evidence of stridor or wheezing. Oropharyngeal exam did not reveal any significant erythema; however, the patient's Mallampati score of 4 obstructed sufficient visualization of the posterior oropharynx. Using a tongue depressor, we observed an erythematous and edematous uvula (Image). No tonsillar hypertrophy or exudates were observed. Given the patient's ability to speak in full sentences with normal oxygen saturation on room air, he was not deemed an appropriate candidate for intubation despite continued complaint of shortness of breath.

Laboratory values of complete blood count and complete metabolic panel were unremarkable, reducing the likelihood of infectious etiology. Due to the patient's discomfort and the likelihood of inflammation vs. angioedema etiology of complaint, he was treated with 60 milligrams (mg) of methylprednisolone, 25 mg of diphenhydramine, and 20 mg of famotidine. The patient markedly improved within several hours and reported that he felt "100% better." He was discharged with a prescription of 20 mg prednisone once daily for a three-day course. Strict return precautions were given and he was instructed to follow up with his primary care physician.

DISCUSSION

Angioedema is a self-limiting condition that results from fluid extravasation into subcutaneous or submucosal tissues. Angioedema typically has a rapid onset with preferential

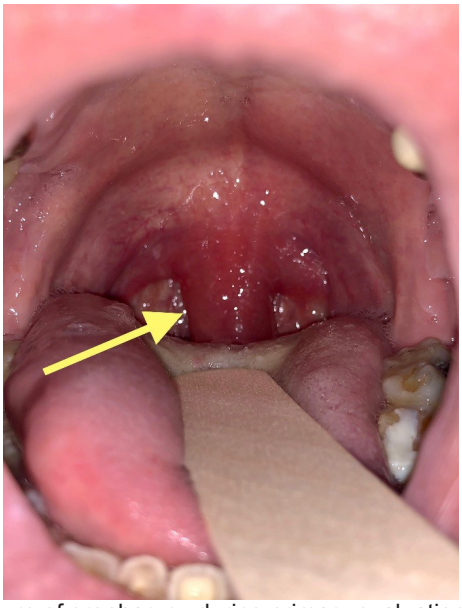

Image. Picture of oropharynx during primary evaluation of patient demonstrating isolated uvular angioedema (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Uvular angioedema (Quincke's disease) is a well-established phenomenon within the current literature.

What makes this presentation of disease reportable?
This report shows how Quincke's disease may present without a clear trigger or systemic signs. Additionally, it highlights the importance of the physical exam.

What is the major learning point?
This case delineates the signs and symptoms in patients presenting with angioedema, the appropriate first-line diagnostic and therapeutic approach, and a strategy to help identify the underlying etiology.

How might this improve emergency medicine practice?
This report seeks to reinforce the diagnostic and therapeutic approach to treating patients with Quincke's disease in the emergency department.

involvement of the face, lips, larynx, and bowels.[1] This case presentation of uvular angioedema, or Quincke's disease, is an example of an uncommon yet potentially life-threatening condition that may be seen in the ED. Specifically, this patient presented with symptoms of a histaminergic angioedema with threatened respiratory compromise. Frequent triggers of this complication are infection, calcium channel blockers, or eosinophilia. In some cases, drug use has been attributed as the inciting agent, which may have been the case in our patient.

For patients with suspected angioedema or isolated angioedema a detailed medication and allergy history is critical.[2] Reported family history of subcutaneous swelling or patients complaining of recurrent bouts of angioedema with abdominal pain should raise suspicion for hereditary angioedema or acquired C1-inhibitor deficiency. A basic array of laboratory tests is necessary for confident diagnosis, including complete blood count, chemistry panel with liver function tests, and inflammatory markers.[3]

The primary etiologies of angioedema are mast cell or bradykinin mediated.[3] Mast cell-mediated causes, such as allergic reaction or adverse reaction to aspirin or NSAIDS, typically present with acute onset and associated urticarial rash, generalized pruritus, bronchospasm, and hypotension. In certain cases, angioedema may be histaminergic in origin but not associated with mast-cell degranulation.[4] In these events there is usually no associated urticarial rash or respiratory compromise. If the airway is patent without concern for compromise then antihistamine and glucocorticoid administration is the first-line therapy. Airway compromise or signs of anaphylaxis should instigate immediate intramuscular epinephrine with the aforementioned therapies. In cases without a clear inciting agent causing angioedema, reports of high doses of antihistamines have been helpful (up to 200 mg of diphenhydramine).[5] For severe and refractory cases there are successful treatments with dapsone, icatibant, and rituximab found in the literature.[6,7,8]

Bradykinin-induced angioedema is typically not associated with urticarial rash, pruritus, or bronchospasm and develops over a period of days. ACE inhibitors and fibrinolytic therapies are common aggravating agents. Inherited and acquired angioedema causes also fall into this class. If family history is concerning, quantifying complement component 4 may be prudent. For hereditary angioedema with C1-inhibitor deficiency or acquired C1-inhibitor deficiency, consider on-demand treatment with icatibant, ecallantide, or recombinant C1 inhibitor. Fresh frozen plasma is a second-line therapy if none of the above is available.[9]

CONCLUSION

In the ED, respiratory distress is a frequently encountered chief complaint that warrants immediate evaluation for the

underlying cause. This case of isolated uvular angioedema – an uncommon yet potentially life-threatening condition – highlights the breadth of possibilities that need to be considered when approaching a non-specific patient complaint. Here we have outlined the appropriate history, physical exam, critical laboratory values, and therapies that emergency physicians should be familiar with to manage isolated angioedema.

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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Rib Osteomyelitis in a Pediatric Patient: Case Report and Review of the Literature

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We present a case report and review of the literature of rib osteomyelitis in a pediatric patient presenting to the emergency department (ED) with fever and increased work of breathing. The patient was seen on a return visit to the ED after discharge with presumed viral illness approximately 12 hours prior. On the second ED visit, there was concern for occult bacteremia, and work-up ultimately revealed a subperiosteal abscess with rib osteomyelitis, a rare etiology for fever in the pediatric patient. The patient was treated with antibiotics, had surgical debridement, and fully recovered. [Clin Pract Cases Emerg Med. 2018;2(4):294–296.]

INTRODUCTION

Fever is a common presenting complaint of pediatric patients in the emergency department (ED). Fever can represent non-specific viral syndromes or serious bacterial infections. Occult bacterial infections, such as osteomyelitis, are less common than viral infections but can frequently elude diagnosis. We present a case report and review of the literature of rib osteomyelitis in a pediatric patient presenting to the ED with fever and increased work of breathing.

CASE REPORT

A nine-month-old male presented to the ED with a four-day history of fever and increased work of breathing. He was first seen by his primary care physician with the onset of symptoms. His mother later took him to the ED where he had a fever but no respiratory distress. He was treated symptomatically and discharged with presumptive diagnosis of viral illness. He returned to the ED approximately 12 hours after discharge with abdominal pain and recurrence of his fever. His parents noted that the patient seemed to be in pain and had grunting with expiration. He also had decreased oral intake, but was still making wet diapers.

His parents also reported cyclical periods of crying during which he seemed uncomfortable. They noted that he seemed to be pale with decreased energy and activity from baseline. Mom also reported no bowel movements for the prior 24 hours, with the patient normally having 2-3 bowel movements daily. Parents denied any cough, congestion, wheezing, stridor, vomiting, or rash. He had no past medical history, and his vaccinations were up to date.

On exam the patient had a rectal temperature of 101.5 degrees Fahrenheit, heart rate of 187 beats per minute, respiratory rate of 36 breaths per minute, and oxygen saturation of 99% on room air. He appeared to be developmentally appropriate and in moderate distress with pale skin; he exhibited no cyanosis, rash, or lesions. He had an expiratory grunt with each breath. No cardiac murmur was appreciated and the lungs were clear without wheezes. The abdomen revealed no focal tenderness. Muscle tone was within normal limits. His neurological exam was without focal deficits and age appropriate.

Labs showed a white blood cell count of 17.0 K/microliter (mcL) (normal 6.0-17.5 K/mcL), C-reactive protein of 13.16 milligrams per deciliter (mg/dL) (normal 0-0.80 mg/dL), lactic acid of 1.8 millimoles/L. Urinalysis was unremarkable. The chest radiograph and ultrasound of the abdomen were unremarkable. An electrocardiogram showed sinus tachycardia. Blood cultures were ordered and the patient was started on empiric antibiotics (piperacillin/tazobactam) in discussion with the pediatric hospitalist service. He was admitted for additional evaluation of fever of unknown origin. Blood cultures were initially positive for methicillin-susceptible *Staphylococcus aureus* (MSSA). An echocardiogram ordered for suspicion of endocarditis was normal. Antibiotics were continued, and the

patient improved clinically; however, his parents noted during his hospitalization that he seemed to be in pain when he was picked up, particularly in his axillae.

Repeat blood cultures obtained after antibiotic therapy were negative, and the patient's fevers were less frequent. He was transitioned to oral cephalexin and observed. On hospital day six, the patient's mother noted a 2x4 centimeter tender, non-erythematous mass in the right axilla. Formal ultrasound showed a soft tissue mass adjacent to the rib, without a definitive fluid collection (Image 1). The patient was transferred to a specialized pediatric hospital where magnetic resonance imaging (MRI) revealed that the axillary mass was consistent with osteomyelitis and subperiosteal abscess of the lateral seventh rib (Image 2). The patient had operative debridement and thereafter made a full recovery with discharge home on hospital day 12.

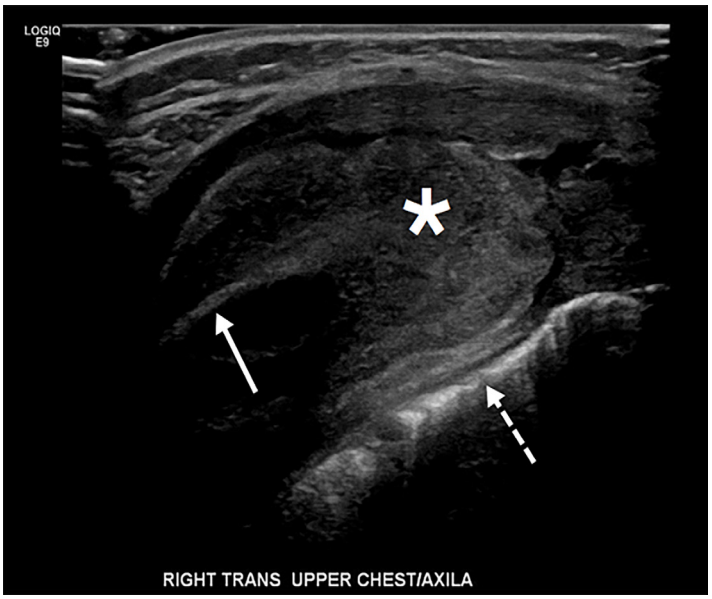

Image 1. Formal ultrasound of right upper chest/axilla showing rib (arrow), lung border (dashed arrow), and periosteal abscess (*).

CPC-EM Capsule

What do we already know about this clinical entity?
In pediatric patients presenting to the emergency department (ED) with fever, occult bacterial causes, such as osteomyelitis, are less common than viral causes but frequently elude diagnosis.

What makes this presentation of disease reportable?
Rib osteomyelitis in pediatric patients is rare, with less than 60 cases reported in the literature.

What is the major learning point?
The diagnosis of rib osteomyelitis requires a high index of suspicion by the emergency physician, especially because the presentation can be non-specific.

How might this improve emergency medicine practice?
Uncommon and rare causes of fever of unknown origin, such as osteomyelitis, should be considered in the evaluation of pediatric patients presenting to the ED.

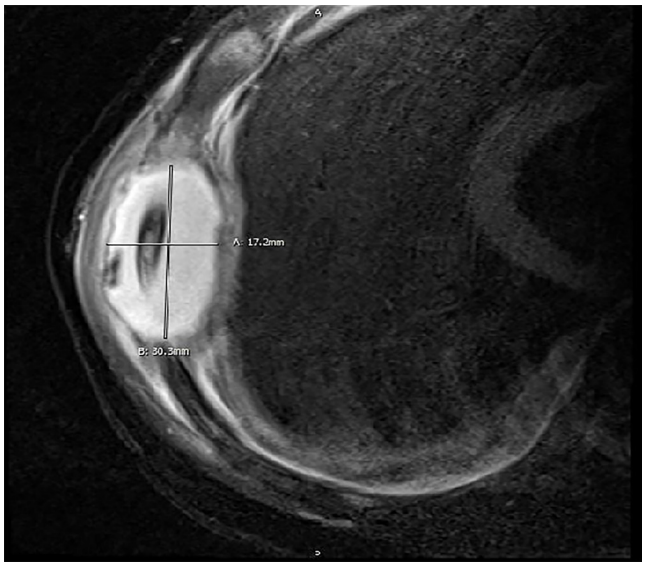

Image 2. Magnetic resonance imaging (axial view: T1, T2 fat saturation technique) showing 17.2 x 30.3 mm periosteal abscess of rib.

DISCUSSION

Rib osteomyelitis in pediatric patients is rare, with less than 60 cases reported in the literature.[1,3] It has been described in association with tuberculosis,[4] via hematogenous spread,[5] and as a sequela of child abuse.[6] Our case illustrates that uncommon and rare causes of fever of unknown origin, such as osteomyelitis, should be considered in the evaluation of pediatric patients presenting to the ED. The etiology of osteomyelitis in this case was not clear, and could have been the result of hematogenous spread, although no primary site of infection was evident. Most cases of rib osteomyelitis are *S. aureus*,[2,7,8] although other organisms

including *Streptococcus pneumonia* and Group A beta hemolytic *Streptococcus* have also been implicated.[9-11]

CONCLUSION

The diagnosis of rib osteomyelitis requires a high index of suspicion by the emergency physician, especially because the presentation can be non-specific[2] as in the case presented. Definitive diagnosis often requires advanced imaging such as computed tomography or MRI, or invasive testing such as biopsy or aspiration with culture, although ultrasound[12] and plain radiography[13] are potentially useful in the initial evaluation. As in the case presented, ultrasound has some role in identifying subperiosteal abscess.[14]

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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48-year-old Man with Fevers, Chest Pain, and a History of Substance Abuse

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A 48-year-old male with a history of intravenous (IV) drug use presented to the emergency department (ED) for an area of mild pain and erythema on his chest. He was then triaged to the urgent care, or fast track, area of the ED. He was well appearing with normal lab findings and vital signs, but his workup revealed mediastinitis with osteomyelitis of the manubrium and clavicles, a surgical emergency. His treatment course included IV antibiotics and operative intervention with thoracic surgery. The patient *looked too good to be sick*, yet he had a life-threatening infection. [Clin Pract Cases Emerg Med. 2018;2(4):297–299.]

INTRODUCTION

Intravenous (IV) drug abuse is a risk factor for several insidious and life-threatening infections seen in the emergency department (ED). This case describes our experience treating one such infection: mediastinitis. We also discuss the physical exam findings associated with this disease.

CASE REPORT

A 48-year-old male with a history of polysubstance abuse came to the urgent care area of our ED with a complaint of constant, aching pain over his sternum and right clavicle. The pain had gradually worsened and was accompanied by intermittent subjective fevers over the prior week. That day the area developed erythema, swelling, and fluctuance (Image 1). The patient was afebrile and had a pulse of 89 beats per minute, a blood pressure of 116/70 milligrams of mercury, a respiratory rate of 16 breaths per minute, and 100% pulse oximetry on room air. He was well appearing on exam, requesting food, and he frequently left to smoke cigarettes outside. The patient had a normal white blood cell count and venous lactate. An ultrasound of the area of pain and swelling (Image 2) and a computed tomography (CT) of the patient's chest (Image 3) were completed.

The preliminary ultrasound report revealed an abscess adjacent to the patient's right sternoclavicular joint (Image 2). A CT of the chest revealed bony destruction of the manubrium and clavicles with abscess extending into the anterior mediastinum (Image 3). The patient was admitted for IV antibiotics and underwent a bilateral sternoclavicular debridement and abscess drainage with thoracic surgery.

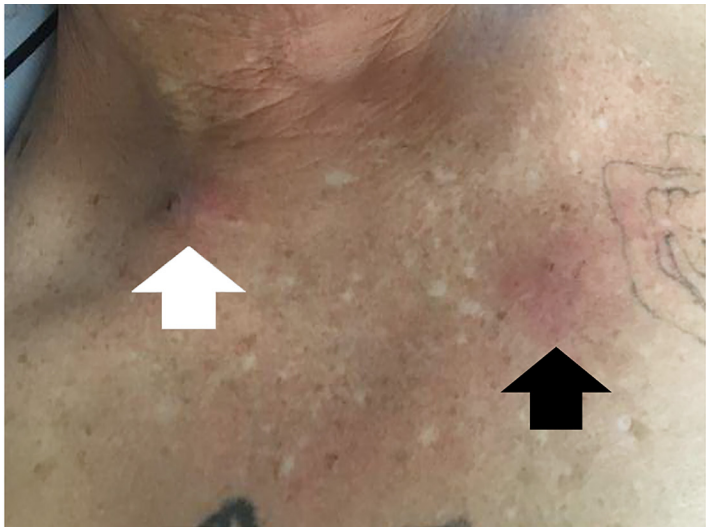

Image 1. Skin findings as seen while the patient is sitting upright (base of neck at top of image). Note the puncture mark within the supraclavicular space (white arrow) and the erythema across the sternum (black arrow).

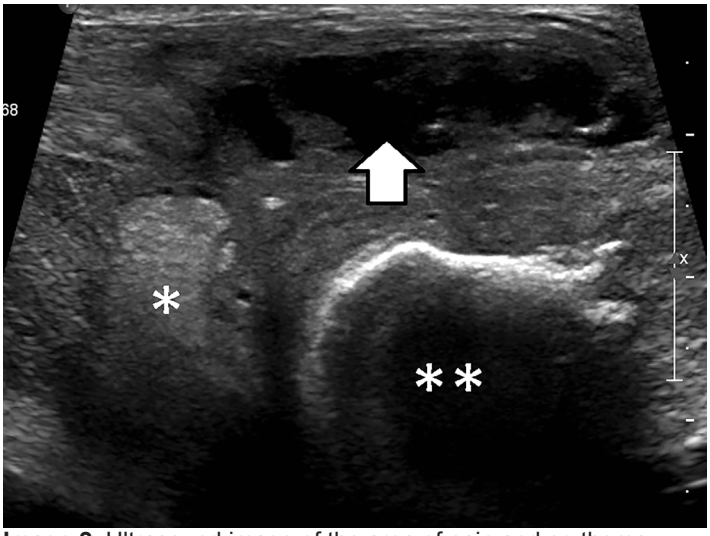

Image 2. Ultrasound image of the area of pain and erythema between the sternum (*) and the right clavicle (**), demonstrating a subcutaneous fluid collection (arrow).

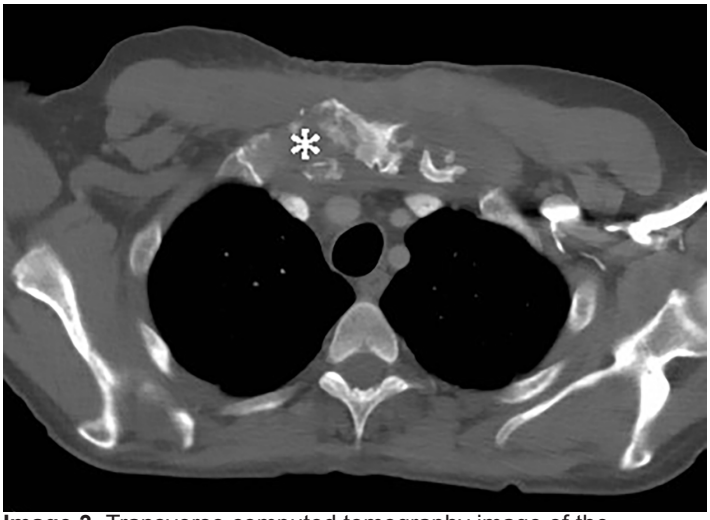

Image 3. Transverse computed tomography image of the chest, taken at the level of the manubrium, showing widespread destruction of the bony tissue (*).

CPC-EM Capsule

What do we already know about this clinical entity?
Intravenous drug use is a known risk factor for devastating and sometimes subtle infections like medistinitus.

What makes this presentation of disease reportable?
This case links the easily-overlooked physical features of a well-appearing patient with their ultrasound and computed tomography results showing an extensive case of mediastinitis.

What is the major learning point?
Patients who are functionally immune-suppressed, like those who abuse substances intravenously, are at high risk for cryptic and devastating infections.

How might this improve emergency medicine practice?
Readers will be able to better recognize and screen patients at high-risk for these infections.

DISCUSSION

Septic arthritis and osteomyelitis of the manubrium is most commonly associated with IV drug use (21%), often a result of patients using non-sterile technique to access their internal jugular or subclavian veins to inject illicit substances using a supraclavicular approach ("pocket shot").[1,2] Other risk factors include infections at a distant site (15%), diabetes (13%), trauma (12%), and infected central venous access (9%).[1,2] Local cellulitis progresses to an abscess and septic arthritis, leading to osteomyelitis and mediastinitis, as seen in this patient. Mediastinitis is rare in the current era of antibiotics, with complications from cardiac surgery, esophagogastroduodenoscopy, or suppurative infections of the head and neck being the most common causes.[3] Patients present after several weeks of chest pain, fevers, and dyspnea.[3] Inflammatory markers like C-reactive protein, erythrocyte sedimentation rate, and procalcitonin will be elevated in these patients and imaging should include CT of the chest with contrast.[4,5] Blood cultures should be drawn prior to the administration of broad-spectrum antibiotics, with the causative organism being Gram-positive (*Staphylococcus aureus*) more often than Gram-negative (*Pseudomonas aeruginosa*).[2] Thoracic surgery should be consulted on these patients as most require surgical abscess drainage.[1,5]

CONCLUSION

This case report is an excellent example of someone who *looked too good to be sick*, yet his workup revealed an impressive and extensive infectious process that required emergency surgery. His presentation was innocuous as he

did not have a toxic appearance; and his vital signs, white blood cell count, and serum lactate were within normal limits. The only abnormalities in his presentation were the area of mild erythema and fluctuance, although the area was only mildly tender and he appeared comfortable. Initially only basic labs and a limited ultrasound study were ordered because the initial presentation did not seem to warrant CT, blood cultures or other more extensive measure. But once the ultrasound revealed an abscess adjacent to the sternoclavicular joint, the complete workup was ordered.

As primary care offices, urgent care centers, and EDs across the nation become increasingly overwhelmed, it is often difficult to justify an extensive workup for a person who looks well and has no demonstrable signs of a worrisome infection. A clinician who is less experienced with invasive soft tissue infections could have reasonably diagnosed this man with cellulitis and a superficial abscess and elected to treat him with outpatient antibiotics, which would not have been effective. By convincing the patient to be forthcoming regarding injection drug use, we were able to add serious infectious process to the differential diagnosis even in the absence of classic symptoms, and that was the key to making the correct diagnosis in a timely manner.

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 Ultrasound Diagnosis of an Atypical Acute Aortic Dissection

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Aortic dissections have a vast array of clinical presentations that rarely follow traditional teachings. Dissections are rapidly fatal conditions requiring immediate diagnosis and treatment to reduce morbidity and mortality. We present a case of an acute aortic dissection presenting as abrupt onset, atraumatic leg pain with absent distal extremity pulses. The prompt use of point-of-care ultrasound detected an intimal flap within the abdominal aorta allowing immediate surgical consultation and intervention. [Clin Pract Cases Emerg Med. 2018;2(4):300–303.]

INTRODUCTION

The first documented case of an aortic dissection occurred in 1760 during the autopsy of King George II: "the pericardium was found distended with a quantity of coagulated blood… and in the trunk of the aorta we found a transverse fissure on its inner side."[1] Over 200 years later, our current understanding of acute aortic dissections is still evolving. The International Registry of Acute Aortic Dissection (IRAD), a large multicenter research consortium, has amassed a vast database providing physicians with extensive information regarding risk factors, examination findings, and historical clues that elicit further testing. Despite our present knowledge and technology, the overall in-hospital mortality is 27.4%.[2] Diagnosing acute aortic dissections remains exceedingly difficult due to its varied presentations, unreliable physical exam findings, and relatively common risk factors associated with numerous other pathologies. We describe a case of an atypical presentation of an acute aortic dissection and the utility of point-of-care ultrasound (POCUS) in expediting the diagnosis and treatment.

CASE PRESENTATION

A 67-year-old man presented to the emergency department (ED) complaining of sudden onset atraumatic right lower extremity pain shortly after using crack cocaine. He described the pain as sharp and burning throughout his entire leg. The patient denied chest pain, shortness of breath, abdominal pain, and focal neurologic symptoms. His past medical history was otherwise unremarkable.

On examination, the patient was alert and oriented but in obvious distress. Vital signs were within normal limits excluding a blood pressure of 180/100 millimeters of mercury (mmHg). The cardiopulmonary and abdominal examinations were benign with notably equal radial pulses. The patient's right lower extremity was cool to touch without palpable pulses distal to and including the common femoral artery. The left lower extremity was warm with bounding pulses. On neurologic exam, the patient had normal and symmetric strength in his bilateral upper and lower extremities without apparent sensory deficits.

Based upon the patient's acute presentation a POCUS was performed immediately in the ED. Our emergency ultrasound division has developed a protocol combining transthoracic echocardiography (TTE) and abdominal aorta ultrasound to evaluate for aortic pathology.[3] POCUS demonstrated a large, undulating intimal flap within the abdominal aorta (Image 1). TTE did not reveal evidence of a Stanford Type A dissection. Immediate aggressive blood pressure control was initiated, and the patient was taken emergently for computed tomography (CT), which confirmed a Stanford Type B dissection (Image 2). Ultimately, the patient underwent thoracic endovascular aortic repair without complications.

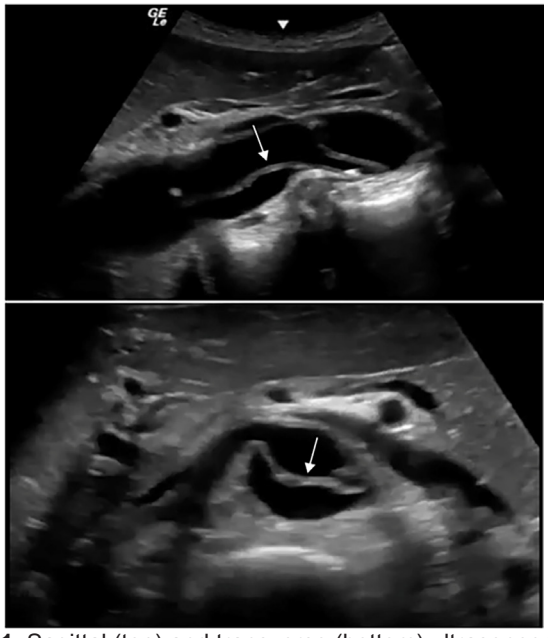

Image 1. Sagittal (top) and transverse (bottom) ultrasonography view of abdominal aorta. Two views of the abdominal aorta. An arrow points to a large, undulating intimal flap noted within the abdominal aorta.

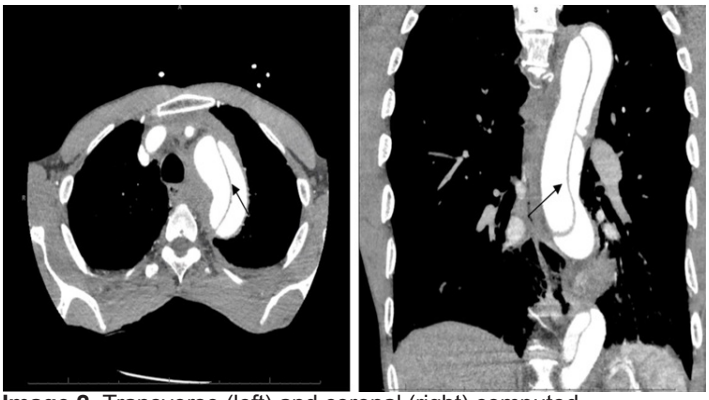

Image 2. Transverse (left) and coronal (right) computed tomography imaging of the thoracic aorta. Images show a large aortic dissection extending distal from the left subclavian artery. An arrow points to the intimal flap that is visible within the aorta.

CPC-EM Capsule

What do we already know about this clinical entity?
Aortic Dissection is a rare disease with a high mortality rate. Diagnosis remains difficult to due to variances in how patients present to the emergency department.

What makes this presentation of disease reportable?
The rapid use of ultrasound to identify a highly specific finding for acute aortic dissection.

What is the major learning point?
Ultrasound can be used to aid in the rapid diagnosis of acute aortic dissection but is not sufficient to rule out the disease.

How might this improve emergency medicine practice?
It serves to remind us that use of point-of-care ultrasound is a rapid diagnostic tool and can be used to help expedite further work up to improve patient care.

DISCUSSION

Characterized by an intimal tear in the aortic wall, an aortic dissection is a relatively rare entity with an annual incidence of nearly three per 100,000 patients.[4] Even more challenging are the diverse clinical signs and symptoms encountered with dissections. The classic presentation of tearing, ripping chest pain is seen in <50% of patients.[2] Furthermore, 10-15% of patients deny pain.[5] The presentation of aortic dissection is so varied and difficult that Sullivan et al. found that emergency physicians evaluating confirmed cases only suspected aortic dissection in 43% of instances.[6] In the absence of chest or abdominal pain, aortic dissection was not suspected.[6]

IRAD has amassed a database to enhance our understanding of acute aortic dissections. Their findings highlight how unreliable history and physical are at diagnosing aortic dissections. Typical risk factors include male gender, age >50, connective tissue disorders, a family history of dissection, congenital aortic pathologies, trauma, and cocaine abuse.[2] Chronic hypertension is the most common risk factor, yet systolic pressures above 150 mmHg are present in only 35.7% of Type A dissections and 70.1% of Type B dissections.[2]

Due to the wide variety of presentations for acute aortic dissections, most attempts at creating a clinical decision rule have failed. Rogers et al. attempted to create a simple clinical decision rule based on initial clinical suspicion as well as a quick, three-step bedside risk assessment. Patients at risk also had chest radiography and electrocardiograms. When the study was retrospectively applied to the IRAD it failed to identify nearly 5% of patients.[7] The American College of Emergency Physicians (ACEP) evaluated this study, and other clinical decision rules, stating that the decision to work

a patient up for acute aortic dissection should be based on physician discretion and that no evidence supported the routine use of clinical decision rules.[8]

One of the most significant impediments in diagnosing aortic dissections is limiting unnecessary advanced imaging without missing any presentations. Currently, ACEP recommends CT angiography as one of three gold-standard diagnostic imaging modalities for aortic dissection with a sensitivity above 98%.[8] Magnetic resonance imaging and trans-esophageal echocardiography (TEE) are also considered gold standard, but limited availability precludes their routine utilization. More recent studies have analyzed laboratory values to enhance diagnostic ability. In particular, D-dimer values have been studied as a potential rule-out test but have not been validated.[8]

The use of POCUS in the ED for aortic pathology is increasing; however, there are limited data on its use for dissection. The majority of data regarding measurements and criteria for detection of aortic dissection is based on studies using TTE with which most emergency physicians have limited experience. The recent ACEP clinical policy reviewed six studies which evaluated the role of TTE in the diagnosis of aortic dissection. Each of the studies involved sonographer-performed or cardiologist-performed TTE, and their sensitivities and specificities varied significantly from 52%-80% and 0-100%, respectively.[7] Most studies looked specifically for aortic root dilation, pericardial effusions, and recognition of an intimal flap; however, no set criteria were used for cutoff measurements. Although the presence of an intimal flap within the abdominal aorta has not been studied in isolation, Roudat et al. noted this finding to be 100% specific and 67% sensitive for aortic dissection. [9]

Recent studies highlight the capability of emergency physician-performed POCUS to diagnose acute aortic dissections. An early case series published by Fojtik et al. initially underscored the ability of emergency providers to identify intra-aortic intimal flaps for the diagnosis of aortic dissections.[10] A more recent prospective study published by Nazerian et al. demonstrated that emergency physicians using POCUS were 88% sensitive for Type A Stanford acute aortic dissections.[11] When combined with a positive finding on their Aortic Dissection Detection risk score, their sensitivities improved to 96%.[11] One of the most significant advantages of POCUS is its immediate availability. Pare et al. accentuated this fact, demonstrating a significant reduction in time to diagnosis of Sanford Type A dissections (>145 minutes) when employing emergency physician-performed sonography.[12]

Gibbons et al. developed an aortic dissection POCUS protocol combining TTE with abdominal aorta ultrasound.[3] The protocol assessed for the presence of one of the following sonographic findings: pericardial effusion, intimal flap, or aortic outflow tract diameter measured at end-diastole >3.5cm.[3] Their protocol identified 96.4% of patients with aortic dissections, confirmed on CT.[3] Furthermore, their protocol was 100% sensitive for Sanford Type A dissection.[3]

Despite advances in diagnostic and treatment technology, the mortality of aortic dissection remains exceedingly high. Every hour delay in diagnosis results in a 1-2% increase in mortality. POCUS is becoming ubiquitous across emergency medicine, and it is a rapid, accurate means to screen for aortic dissections.

CONCLUSION

Diagnosing an acute aortic dissection presents a unique challenge for all emergency providers. Its signs and symptoms lack sensitivity and specificity, and its exceedingly high mortality rate mandates prompt diagnosis and treatment. The role of point-of-care ultrasound continues to expand within the field of emergency medicine, and the aforementioned case and studies validate its efficacy to expedite the diagnosis and treatment of acute aortic pathology.

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 Hepatitis B with Pancreatitis and Cholecystitis Leading to Acute Liver Failure and Death

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Acute liver failure is defined as severe acute liver injury, concurrent with encephalopathy and loss of hepatic synthetic function, in a patient without known pre-existing liver disease. Evaluation of acute liver failure in the emergency department should focus on identification of treatable causes. Acute liver failure from acute hepatitis B infection is a rare but potentially lethal occurrence. Multi-organ dysfunction from acute liver failure may be exacerbated by metabolic and inflammatory reactions associated with acute pancreatitis, which accompanies approximately 5% of cases of acute viral hepatitis. Transplant-free survival rate with liver failure from acute hepatitis B is unfortunately less than 20%. [Clin Pract Cases Emerg Med. 2018;2(4):304–308.]

INTRODUCTION

The critically ill patient presenting with fever, abdominal pain, and jaundice represents a diagnostic and therapeutic challenge in the emergency department (ED). Acute hepatitis B causing acute liver failure is a rare occurrence; multi-organ complications of acute liver failure are associated with high case fatality, despite advances in etiology-specific therapies and availability of urgent liver transplantation.[1] Pancreatitis may complicate up to 35% of acute liver failure cases. Acute viral hepatitis is associated with pancreatitis and cholecystitis in about 5% of cases, due to direct tissue penetration by the virus itself as opposed to ductal obstruction. [2] Although pancreatitis associated with viral hepatitis generally follows a benign course, multisystem failure with hepatic encephalopathy in the setting of acute liver failure may be exacerbated by impaired metabolism and inflammatory cytokine reactions that accompany acute pancreatitis.[2-4]

CASE REPORT

A 39-year-old man with no known health issues prior to ED presentation,was brought to the ED by family members with concerns for fever, generalized weakness, and abdominal pain of one week's duration. He had been evaluated at an urgent care facility and referred to the ED after treatment with acetaminophen for temperature of 39.2 ºC and ondansetron for nausea; rapid antigen testing for influenza and streptococcus were negative. He reported one week of fevers, night sweats, and anorexia with non-radiating, right upper quadrant pain, as well as multiple episodes of vomiting and the development of loose, gray-colored stools. He denied back pain or urinary symptoms. He had taken a small number of acetaminophen tablets of unknown dosage over the preceding week for fever, but denied regular or excessive use. He denied recent travel, unusual foods, herb or mushroom ingestion, ethanol use, or intravenous (IV) drug abuse. Socially, he admitted to daily marijuana and tobacco use.

Physical examination revealed an acutely ill man with scleral icterus, who was diaphoretic and moaning, complaining of pain. Vital signs on presentation included an oral temperature of 37.3 ºC, heart rate of 78 beats per minute, blood pressure of 132/70 millimeters of mercury, and respiratory rate of 20 breaths per minute. Mucous membranes were dry, lungs were clear, and the heart sounds were regular without murmurs or gallops. The abdomen was soft with moderate tenderness in the right upper quadrant without guarding. The liver edge was palpable two centimeters (cm) inferior to the costal margin in the midclavicular line. He was slow to answer but oriented to person, place and time.

Laboratory findings were notable for significantly elevated white blood cell count of 22,300 / millimeters cubed (mm^3), hemoglobin of 18.7 grams per deciliter (g/dL), platelets of 171,000 /$mm.^3$ Liver function studies were remarkable for total bilirubin of 16.4 milligrams per deciliter (mg/dL), direct bilirubin of 10.6 mg/dL, aspartate aminotransferase of 2,682 international units per liter (IU/L), alanine aminotransferase of 7,521 IU/L, and alkaline phosphatase of 288 IU/L. Lipase was markedly elevated at 16,879 IU/L. Serum bicarbonate was 24 milliequivalents per liter (mEq/L), blood urea nitrogen was 26 mg/dL, and creatinine 1.53 mg/dL. Glucose was 61 mg/dL, and the remainder of electrolyte panel was normal. Coagulation studies revealed prothrombin time of 41.1 seconds and international normalized ratio (INR) of 4.3. Venous blood gas was notable for pH of 7.38 with lactate significantly elevated at 12.7 mEq/L. Toxicology workup was negative: ethanol less than 3 mg/dL and acetaminophen less than 2 micrograms per liter (μg/mL). Urine screen for drugs of abuse was positive for cannabinoids but negative for benzodiazepines, phencyclidine, opiates, and amphetamines.

Initial imaging included a right upper quadrant ultrasound (US) that revealed gallbladder distension with sludge, pericholecystic fluid and anterior wall thickening, but no gallstones. The common bile duct was measured at five millimeters (mm). Computerized tomography (CT) of the abdomen and pelvis obtained without contrast confirmed abnormal gallbladder findings from US and also demonstrated an edematous pancreas with peri-pancreatic fat stranding, without obstructing mass, as well as trace ascites. Initial management included a bolus of two L of IV crystalloids as well as broad-spectrum antibiotic coverage for possible biliary sepsis (1.5 grams [g] vancomycin and 3.375g piperacillin-tazobactam). A repeat bedside glucose of 53 mg/dL was treated with IV dextrose.

The patient was admitted to the medical intensive care unit with consults from the surgical and gastroenterology services. Primary admission diagnoses included acute hepatitis, acalculous cholecystitis and acute pancreatitis of unclear etiology. Lipid panel, including triglycerides, returned normal and the pattern of liver function abnormality did not point to an obstructive picture. Hepatitis serologies subsequently returned positive and suggestive of acute infection: hepatitis B core immunoglobulin M (IgM) reactive, hepatitis B surface antigen reactive, hepatitis B e-antigen and antibody reactive, and hepatitis B deoxyribonucleic acid level significantly elevated at 51,416 IU/mL, indicating highly active viral replication. Testing for cytomegalovirus, hepatitis C virus, hepatitis A virus, hepatitis D virus, hepatitis E virus, Epstein-Barr virus, and human immunodeficiency virus 1 and 2 were all negative.

The patient was questioned regarding risk factors for viral hepatitis; he stated he was sexually active with male and female partners and admitted to unprotected oral intercourse three weeks prior to his acute illness and barrier-protected anal intercourse six months prior to presentation. There was no history of tattoos or blood transfusions and no known prior history of hepatitis infection.

Antiviral treatment was initiated with IV entecavir; IV *N*-acetylcysteine therapy was given for hepatoprotective effects. Antimicrobial therapy was continued with piperacillin-tazobactam, although cultures of blood and urine showed no growth on hospital day 2, and remained negative. Consultation with transplant surgery placed the patient on emergent status for priority liver transplantation.

The patient developed acute mental status changes with drowsiness alternating with agitation; he could no longer speak in full sentences, and was not oriented to time or situation. Serum glucose was 153 mg/dL and CT of the brain did not reveal structural abnormalities. Progressive decline in level of consciousness with asterixis was associated with ammonia level of 228 micromoles per liter (μmol/L); hepatic encephalopathy was treated with lactulose and rifaximin, given orally. IV vitamin K1 and fresh frozen plasma were given for treatment of worsening coagulopathy, with INR increased to 9.0. He was

CPC-EM Capsule

What do we already know about this clinical entity?
Acute liver failure from acute hepatitis B virus is rare, but potentially lethal; case fatalities are associated with encephalopathy and cerebral edema.

What makes this presentation of disease reportable?
A rapidly progressive case of acute hepatitis B infection, associated with pancreatitis and cholecystitis, was refractory to aggressive therapeutic measures.

What is the major learning point?
Acute hepatitis B must be considered when determining the etiology of acute liver failure; early directed therapies in a transplantation center may improve survival.

How might this improve emergency medicine practice?
Although rare, acute hepatitis B may present with acute liver failure, with a high case fatality, necessitating specific therapeutic measures to improve survival.

Table. Etiologies of acute liver failure.

Etiologies of acute liver failure	Commonly reported	Uncommonly reported
Toxic/drug induced	Acetaminophen Ethanol Isoniazid Phenytoin Carbamazepine Valproate	Rifampicin Methotrexate Halogenated hydrocarbons *Amanita* mushroom
Viral	Hepatitis A,B,D,E	Hepatitis C Cytomegalovirus Epstein-Barr virus Herpes viruses Varicella zoster Parvo-virus B19 Adenovirus Hemorrhagic fevers Yellow fever
Herbal products		Black cohosh Chaparral Kava Camphor Castor oil
Vascular	Veno-occlusive disease Budd-Chiari Sepsis/"shock liver"	
Metabolic		Wilson disease Alpha-1-antitrypsin deficiency Acute fatty liver of pregnancy Reye syndrome
Miscellaneous		Non-alcoholic steatohepatitis Autoimmune hepatitis Heatstroke Malignant infiltration: lymphoma, leukemia

intubated for airway protection after a vomiting episode with possible aspiration.

The patient's mental status continued to worsen, and repeat unenhanced head CT demonstrated interval development of cerebral edema with early signs of brainstem herniation. After neurosurgery consultation, hyperosmolar therapy was initiated with IV mannitol and hypertonic saline infusions. Decline in renal function with a fall in serum sodium to 131 mmol/L and minimal urine output was treated with continuous veno-venous hemofiltration. The patient's neurologic status continued to decline with loss of brainstem reflexes. Given the patient's likelihood of severe irreversible neurological disability, the possibility for successful liver transplantation was deemed unlikely and the patient's family elected to withdraw care. After compassionate extubation, the patient expired on hospital day 3, less than 60 hours after his initial ED presentation.

DISCUSSION

Acute liver failure is defined as severe acute liver injury, concurrent with encephalopathy and evidence for loss of hepatic synthesis, manifested by elevated prothrombin time or INR, in a patient without cirrhosis or pre-existing liver disease.[1] Acute liver failure can be further subcategorized by the time course of illness, whereby hyperacute disease is defined as illness duration less than seven days, acute disease 7-21 days, and subacute disease greater than 21 days. Cerebral edema is more common in hyperacute and acute disease.[5]

Evaluation of acute liver failure in the ED should focus on identification of treatable causes. Etiologies include viruses, hepatotoxic drugs (most commonly acetaminophen), amanita mushroom poisoning, idiosyncratic drug reactions, autoimmune hepatitis, environmental toxins, complications of pregnancy, sepsis, malignancy, and vascular conditions including acute Budd-Chiari syndrome.[6,7] The most common etiologies of acute liver failure in the U.S. are acetaminophen toxicity and idiosyncratic drug reactions; the low rate of viral hepatitis in developed countries is reflective of immunization programs, improved screening of blood products, and public health measures.[8,9] The following table summarizes commonly reported causes of acute liver failure.[6-10]

Acute hepatitis B is usually subclinical with only 30% of cases resulting in clinically apparent icteric disease.[10] Acute liver failure is a rare occurrence seen in only 0.1-0.5% of patients with acute hepatitis B.[11] Possible risk factors for severe course of illness include co-ingestion of acetaminophen, alcohol, or amphetamine use during illness.[11] Prodromal fever and temperature greater than 38 ºC are independent risk factors for development of acute liver failure.[12]

The diagnosis of acute hepatitis B may not be apparent in the ED setting, as viral serologies generally do not have rapid turnaround time. Inferences may be drawn by patterns of abnormalities of liver enzymes, which may be interpreted as predominantly hepatocyte injury vs. biliary tract obstruction. Hepatocyte injury is suggested by a pattern of elevation in aspartate aminotransferase and alanine aminotransferase. Predominant elevations in alkaline phosphatase and ɣ-glutamyl-transpeptidase are suggestive of a cholestatic injury pattern.[13]

Antiviral therapies are mainstays of therapy for severe acute hepatitis B.[3] The aims of antiviral therapy are to reverse or delay complications of cirrhosis and to decrease the risk of viral reinfection if patients ultimately receive a liver transplant. Lamivudine may be associated with higher rates of progression to chronic hepatitis B, not seen with entecavir.[14] One study suggests that early treatment (within one week of illness onset) with lamivudine may lead to decreased mortality compared to both control cases and delayed treatment (greater than one week after onset of illness).[15]

N-acetylcysteine is well established in the treatment of acetaminophen toxicity. Its utility in cases of acute liver failure from other causes is under debate.[3,16] *N*-acetylcysteine improves microcirculatory blood flow and oxygen delivery to vital organs by acting as an anti-inflammatory agent, antioxidant, and inotrope, and has improved transplant–free survival in one study of patients with non-acetaminophen-induced acute liver failure.[17-19]

Consideration should be given to treatment of coagulopathy related to acute liver failure. Current recommendations suggest a one-time prophylactic dose of 5-10 mg of Vitamin K1.[20] Treatment with fresh frozen plasma in the absence of active bleeding is not indicated and carries a risk of exacerbating volume overload and causing transfusion-related acute lung injury.[1]

CONCLUSION

Acute liver failure from acute hepatitis B is a rare but potentially lethal complication.[6] The transplant-free survival rate in patients with acute hepatitis B is only 19%, and case fatalities are associated with hepatic encephalopathy and cerebral edema.[21] Although viral causes of acute liver failure are uncommon, this case illustrates the importance of keeping viral hepatitis in the differential diagnosis for patients with acute liver failure. Early identification, directed therapy, and treatment at a transplantation center are associated with increased survival.[6]

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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Double Sequence Defibrillation for Out-of-hospital Cardiac Arrest: Unlikely Survival

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Survival from out-of-hospital cardiac arrest (OHCA) is highest with early defibrillation and immediate, high-quality cardiopulmonary resuscitation. Return of spontaneous circulation (ROSC) is rare in OHCA. The purpose of this discussion and case report is to highlight the use of double sequence defibrillation (DSD) for refractory ventricular fibrillation (RVF). We present a 58-year-old male with RVF who successfully achieved ROSC after 38 minutes using DSD and had a good neurological outcome. DSD has shown promise in many case reports and case series as a means of increasing ROSC and survival rates in OHCA. [Clin Pract Cases Emerg Med. 2018;2(4):309–311.]

INTRODUCTION

The global incidence of out-of-hospital cardiac arrest (OHCA) is 55 per 100,000 adults per year, and the average survival (to hospital discharge) is 7% in adults.[1] Ventricular fibrillation (VF) is the most common rhythm after OHCA occurring in approximately 70% of cases.[2] Return of spontaneous circulation (ROSC) is rare in OHCA; however, recently improved prehospital ROSC rates have been demonstrated.[3] These moderately improved rates are likely attributed to high-quality bystander cardiopulmonary resuscitation (CPR) with increased emphasis on uninterrupted CPR with good technique.

We report on a case where double sequence defibrillation (DSD) was used to treat refractory ventricular fibrillation (RVF). RVF is typically defined as persistent VF following three to five unsuccessful shocks. DSD has been used safely in electrophysiology labs for quite some time.[4] When using DSD, two sets of pads are placed in the anteroapical and anteroposterior positions and deliver a shock nearly simultaneously. (Image)

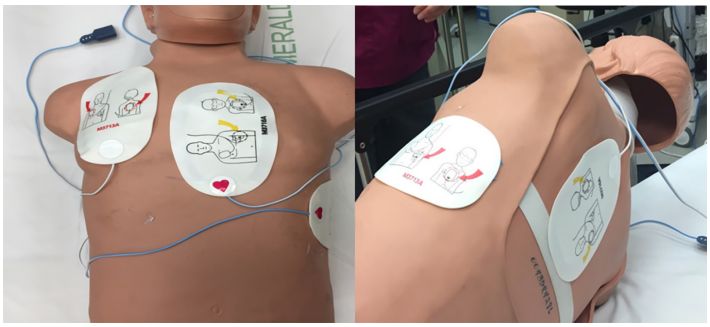

Image. Pad placement for double sequence defibrillation, two sets of pads are placed in the anteroapical (red arrows) and anteroposterior (yellow arrows) positions as demonstrated on mannequin.

CASE REPORT

A 58-year-old male with a history of hypertension and cardiac stents two years prior had just arrived at a Saturday morning prayer breakfast at church when he suddenly went unresponsive and was found to be pulseless and apneic by bystanders. CPR was immediately initiated. Emergency medical services (EMS) arrived with a six-minute response time. The patient was found to be in VF and defibrillated at 200 joules (J) unsuccessfully. Intraosseous access was established and epinephrine was administered. A supraglottic airway device was placed for ventilations and amiodarone 300mg was given. Over the course of 26 minutes five subsequent standard defibrillations were administered unsuccessfully.

At this point EMS contacted online medical control, and DSD was ordered. The patient had a second set of pads placed in the anteroapical position. Both the engine and medic defibrillators were used to deliver 360J, each nearly simultaneously. The

post-shock rhythm revealed pulseless electrical activity (PEA) on the monitor, and CPR was resumed along with the seventh dose of epinephrine. The patient then was noted to be back in ventricular fibrillation, and dual sequential defibrillation was again performed. The post-shock rhythm analysis revealed a brief period of systole into sinus bradycardia with the ROSC. ROSC was achieved 38 minutes from the time of 911 dispatch.

En route to the hospital, the patient again lost pulses and was found to be in PEA. On arrival to the emergency department, he was confirmed pulseless, and CPR was continued. Point-of-care ultrasound showed cardiac activity shortly after ROSC was achieved again. The patient was intubated and central venous access established with norepinephrine infusion to stabilize blood pressure. His electrocardiogram revealed ST-segment elevation in augmented vector left (aVL) and the patient was urgently taken to the cardiac catheterization lab where he was found to have 100% occlusion of his circumflex artery and had a single stent placed. The patient was transferred to the intensive care unit where he was extubated less than 24 hours later. The patient was then discharged to home after only six days in the hospital. His inpatient echo showed an ejection fraction of 60%. At 30 days post-arrest he had some minor, short-term memory issues but had returned to work. The only complication was a lower extremity deep vein thrombosis.

CPC-EM Capsule

What do we already know about this clinical entity?
Return of spontaneous circulation (ROSC) is rare in out-of-hospital cardiac arrest (OHCA). Double sequence defibrillation (DSD) has shown promise as a means of increasing ROSC and survival rates in OHCA.

What makes this presentation of disease reportable?
Our patient achieved ROSC after 38 minutes of DSD, and survived neurologically intact. To our knowledge, this is the longest length of refractory ventricular fibrillation (RVF) time that was corrected using DSD.

What is the major learning point?
While effective and immediate cardiopulmonary resuscitation is most important for OHCA survival, other modalities should be considered for RVF including DSD.

How might this improve emergency medicine practice?
This case study helps justify further emergency medical services' research investigating new standards of care for OHCA.

DISCUSSION

What makes this case remarkable is that this patient made a full neurological recovery despite the unlikelihood based on a recent publication out of Japan. Goto et al. showed that survival after 30 minutes of cardiac arrest was only 0.8% in a study group of over 17,000 cardiac arrest patients. Neurologically intact survival was only 0.4% in the same group after 30 minutes of cardiac arrest.[6] This particular patient survived after 38 minutes with a cerebral performance category (CPC) score of one. The CPC score is a five-category scale for measuring neurological status after cardiac arrest (Table). A CPC score of one correlates to conscious and alert with good cerebral performance. A CPC score of five correlates to brain dead, circulation preserved.[7] Many other factors most likely contributed to the good outcome of this patient's case. First and foremost, this patient received immediate CPR after beginning cardiac arrest.

DSD is a tool that more physicians and EMS providers should become aware of. Several case reports and case series have been published in recent years supporting the potential use of DSD in OHCA. One case series by Cortez et al. showed a survival to discharge in three of 12 patients who presumably would have died if DSD had not been used after RVF. Two of the three patients (2/12, 17%) had a CPC score of one in the study.[8] Merlin et al. showed even better results in another case series. three patients out of seven (3/7, 43%) in whom DSD was used after RVF survived to hospital discharge; once again these were patients who otherwise would presumably not have survived without the use of DSD. All three patients were discharged with CPC scores of one.[9] Lastly, a less-successful case series by Cabanas et al. had 10 patients in whom DSD was used, but none survived to discharge. Three patients (3/10, 30%) achieved ROSC in the field with the utilization of DSD.[10] An important discussion point is the presumed survival due to DSD; this caveat should not be ignored. Without further research we do not know if the next standard defibrillation attempt would have converted the patient from RVF.

Current guidelines have established that survival from OHCA is highest with early defibrillation and immediate, high-quality CPR.[5] We would like to suggest that early use of DSD defibrillation may contribute to survival from OHCA. In the above trials mentioned, the average time from the determination of arrest to first DSD was 32.3 minutes. In those patients who achieved ROSC, it was 31.3 minutes. In the patients who survived, average time to first DSD was 24.2 minutes. While these data suggest that earlier DSD could potentially improve outcomes, further research is needed. The patient we

Table. Cerebral performance category (CPC) score: measuring neurological status after cardiac arrest.

CPC score	Definition
1	Conscious and alert with normal function or only slight disability
2	Conscious and alert with moderate disability
3	Conscious with severe disability
4	Comatose or persistent vegetative state
5	Brain dead or death from other causes

present achieved ROSC after 38 minutes of VF and survived neurologically intact.

CONCLUSION

Further research is required to establish DSD as an effective means for treating RVF. DSD has shown promise in these case reports and case series as a possible tool of increasing ROSC and survival rates in OHCA. We present a case that adds to this literature and shows that positive outcomes can be achieved beyond the time window generally accepted if coupled with effective and immediate CPR.

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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Spontaneous Thrombosis of the Aortic Arch after Outpatient Urologic Procedure

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A healthy, 42-year-old woman presented to a local community hospital with abdominal pain and left arm pain after laser stone ablation and ureteral stenting performed earlier that day. She was diagnosed with a spontaneous aortic thrombus and embolization of the radial, ulnar and splenic arteries and transferred to a tertiary care facility for cardiothoracic surgery evaluation. This case report discusses her emergency department course, disposition, and one-year outcome. [Clin Pract Cases Emerg Med. 2018;2(4):312–315.]

INTRODUCTION

Spontaneous arterial occlusion is an uncommon but critical clinical entity with significant potential for morbidity and mortality. Stroke, infarct of solid organs such as kidney or spleen, mesenteric ischemia, ischemic colitis, renal failure, myocardial infarction, and loss of limb are several sequelae that require prompt diagnosis and intervention in the emergency department (ED).[1] Clinical presentations range from straightforward, such as a painful, pulseless extremity, to more complex and nuanced cases such as abdominal pain due to organ ischemia that can mimic renal colic, colitis, or mechanical back pain.[2] Diagnostic modalities used to confirm arterial occlusion include nonspecific laboratory analysis such as serum lactate acid level, sonographic imaging, computed tomography (CT), and arteriography and digital subtraction arteriography. The mainstay of treatment is anticoagulation with heparin.[1-3] Other possible treatment includes surgical embolectomy/thrombectomy, clot dissolution with tissue plasminogen activator via systemic administration, or catheter-directed thrombolysis.[3-5] Secondary treatment goals include pain management and searching for the underlying cause and sequelae of thrombosis.[1-3]

CASE REPORT

A 42-year-old female presented to the ED of a community hospital six hours after lithotripsy and laser stone ablation for left ureterolithiasis with complaints of sudden onset of non-radiating, left-sided, aching abdominal pain for one hour. It was associated with mild nausea and left arm pain that she described as an aching, throbbing sensation. She rated her discomfort as severe in intensity. The pain was unchanged after taking a hydrocodone/acetaminophen 5/325 milligrams (mg) tablet at onset of symptoms. She denied any fever, chills, shortness of breath, or chest pain. She appeared quite uncomfortable.

Past medical history was significant only for kidney stones. She denied any family history of blood-clotting disorders. She had a past surgical history of cesarean section, and recent stone ablation and ureteral stenting described above. The only medication she took was hydrocodone 5/325mg tablets, prescribed post-operatively. She denied any smoking or illicit drug use. The patient admitted to an occasional alcoholic beverage.

Physical exam revealed an overweight female in obvious discomfort with an oral temperature of 97.6°F, heart rate of 92 beats per minute, respirations of 22 breaths per minute, and a blood pressure of 93/60 millimeters of mercury. Significant physical findings included pallor of the left distal forearm with no palpable radial or ulnar pulses, and slightly delayed capillary refill to the fingers of the left hand. Right radial and bilateral pedal pulses were 2+. The abdomen was soft and mildly tender in the left middle and lower quadrants

with diminished bowel sounds. There was some mild, left costovertebral angle tenderness.

A working differential of arterial occlusion of the left arm, sepsis secondary to urinary tract infection or pyelonephritis, local peritonitis from ureteral rupture, mesenteric ischemia, and abdominal organ injury from lithotripsy was used to formulate the initial work-up. Vascular surgery was consulted and agreed to see the patient emergently in the ED. Diagnostic studies included a complete blood count (CBC), comprehensive metabolic panel, lactic acid, blood cultures, computed tomography angiography (CTA) of the left upper extremity and CT of abdomen and pelvis, pain and nausea control with doses of fentanyl and ondansetron.

CBC showed a white blood count of 16.2 and 96% neutrophils, a hemoglobin level of 11.3, and platelets of 218. Chemistries were within normal limits, except for a mild hypokalemia at 3.3 (repleted intravenously) and a lactic acid elevated at 4.0. CTA of the left upper extremity revealed a non-occlusive thrombus in the aortic arch extending into the origin of the left subclavian artery measuring 12 millimeters (mm) by 12 mm (Image 1), as well as thromboembolic occlusion of the distal left brachial artery at the elbow with reconstitution at the level of the proximal radial and ulnar arteries (Image 2). CT of the abdomen and pelvis showed majority of the spleen was non-enhancing, suggesting a large infarct (Image 3). Given initial concern for sepsis, broad-spectrum coverage with vancomycin and piperacillin/ tazobactam was initiated. Vascular surgery recommended transfer to a tertiary facility for possible urgent vs. emergent aortic thrombectomy as our facility did not have cardiovascular surgical capabilities. The patient was

CPC-EM Capsule

What do we already know about this clinical entity?
Aortic thrombus is rare with only a handful of cases noted in the literature. Almost all occurrences are diagnosed after presentation as secondary downstream embolization.

What makes this presentation of disease reportable?
Rare occurrence coupled with life-and-limb threat make this a no-miss diagnosis in the emergency department.

What is the major learning point?
Prompt diagnosis and cardiovascular surgery referral is imperative. Pain from organ ischemia will require aggressive care with possible intubation and sedation.

How might this improve emergency medicine practice?
Keeping the differential of aortic thrombus in mind with any arterial occlusion can expedite appropriate referral and coordination of care.

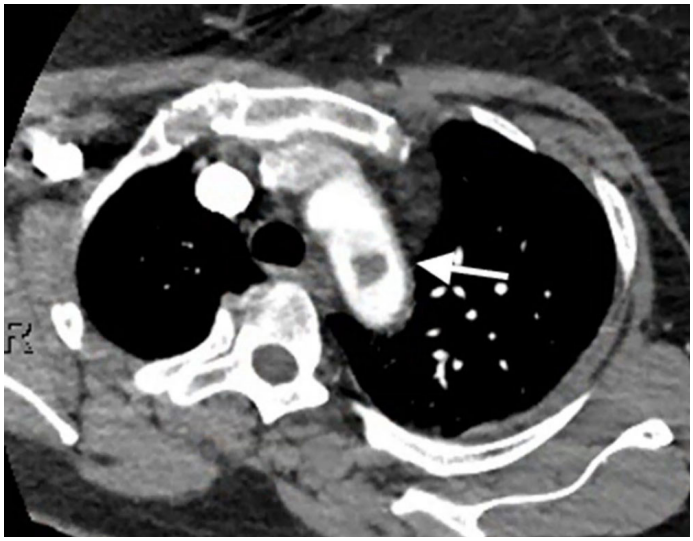

Image 1. Computed tomography showing thrombus in the aortic arch (arrow).

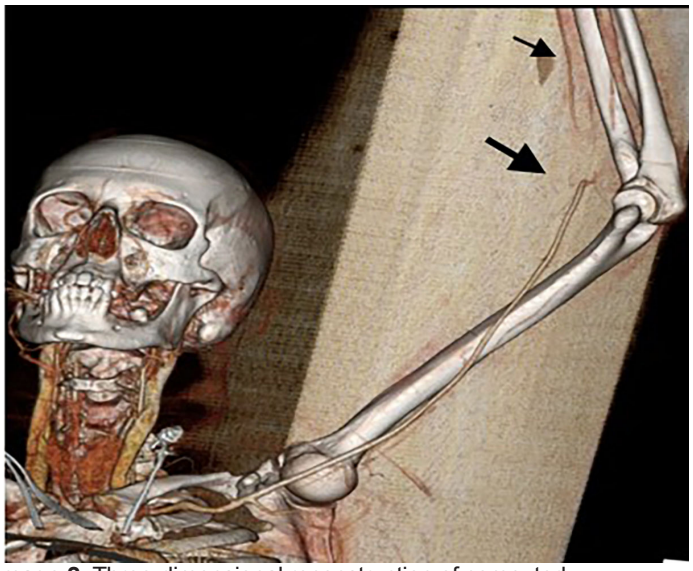

Image 2. Three-dimensional reconstruction of computed arteriography of forearm showing occlusion of the brachial artery (wide arrow) with reconstitution distally (thin arrow).

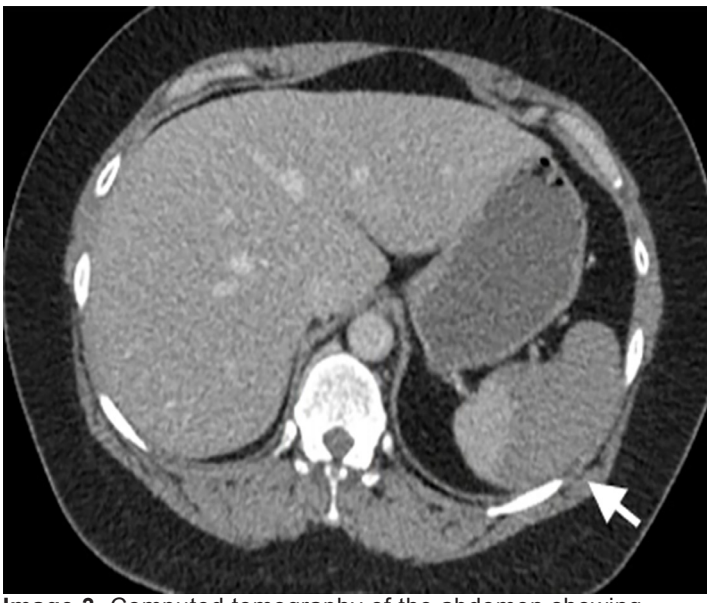

Image 3. Computed tomography of the abdomen showing decreased contrast uptake in the spleen consistent with large area of infarct (arrow).

heparinized in the interim. Initial coagulation studies, obtained after initial heparin bolus, showed international normalized ratio range (INR) of 1.25 and partial thromboplastin time of 74. Consultation with the cardiovascular surgeon was obtained at the nearest tertiary facility. They agreed with the possible need for urgent intervention, and the patient was accepted for transfer with no further recommendations regarding her care at that time. The patient's pain was initially treated with intermittent doses of opioids. While awaiting transport, she became increasingly restless and unable to remain still in bed causing dislodgement of monitoring equipment and intravenous access. Due to the intractable pain, compliance with care, and concern for patient safety during transport, the decision was made to intubate her. Routine rapid sequence intubation was performed. Sedation and analgesia with propofol and fentanyl was initiated. The patient was adequately sedated at this point and stable. Transport to the tertiary facility proceeded without further issue.

At the tertiary facility, the patient remained stable. She experienced return of circulation to the upper extremity without intervention. A repeat CTA of the aorta several hours after arrival showed resolution of the aortic thrombus. The remainder of the patient's hospital stay was uneventful. No underlying pathology was identified as the cause of her acute thrombus formation. She was transitioned to warfarin and discharged home to follow up with her primary care physician for management of her anticoagulation. Several weeks after this event, the patient had outpatient workup for left chest discomfort. INR at that time was therapeutic, and a CTA of the chest showed no evidence of pulmonary embolism. There was a reactive left lower lobe process with a small pleural effusion, believed to be related to a perisplenic fluid collection. The spleen was noted to be slightly larger than on prior study and continued to show changes consistent with an infarct. Her symptoms resolved with supportive care, and follow-up studies showed resolution of the inflammatory process and subsequent splenic atrophy. She currently remains on warfarin and has experienced no post-embolic sequelae other than splenic atrophy.

DISCUSSION

Thrombus in the aortic arch is an infrequent clinical event with few case reports in the recent literature. In a case series looking at thoracic aortic thrombosis, nine cases were found over a five-year period with only two involving the aortic arch.[6] Generally, a thrombus is formed secondary to an atherosclerotic plaque with other potential causes including coagulation disorders, paraneoplastic process, cardiac structural abnormalities, or hypercoagulable states.[1,3] In all cases reported in our literature review, the diagnosis was made after an acute presentation for an ischemic limb.

Treatment includes both medical and surgical options. The most common approach is systemic anticoagulation with heparin and transition to oral warfarin or thrombolysis and anticoagulation. While this is effective in most cases, re-embolization is a concern.[1-3] Until recent technological advancements, surgical treatment required cardiopulmonary bypass and carried a high morbidity with up to 14% of patients suffering cerebral insult.[7] More recently, endovascular therapy has been used in a few cases with good results and avoids postoperative morbidity.[8-10] No studies were found that compared endovascular vs. open surgical embolectomy and repair. In cases and investigations reviewed, all patients were discharged home on some form of anticoagulation, with warfarin the most common agent. No consensus regarding standard treatment was described in the literature. ED treatment remains systemic anticoagulation on heparin and supportive care.[2]

CONCLUSION

Aortic thrombus and subsequent downstream embolization presents a time-sensitive and life-threatening challenge to the emergency physician. Rapid identification of aortic involvement and subsequent cardiothoracic surgical evaluation is critical. Disposition at a tertiary center with the availability of both endovascular and open cardiopulmonary bypass capabilities is necessary. The physician should be prepared to administer thrombolytic therapy to the hemodynamically unstable patient, as well as deeply sedate the patient if pain cannot otherwise be controlled.

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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Pacemaker-associated Phlegmasia Cerulea Dolens Treated with Catheter-directed Thrombolysis

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Phlegmasia cerulea dolens (PCD) is a rare and severe form of deep venous thrombosis that is classically associated with the lower extremities. We report a case of upper extremity PCD developing abruptly in a 37-year-old female with an indwelling cardiac pacemaker who presented to the emergency department complaining of pain and paresthesias in her left arm, adjoining left chest wall, and inferior neck. Her condition was promptly diagnosed and successfully treated with intravenous unfractionated heparin and balloon venoplasty with catheter-directed thrombolysis without any known residual signs or symptoms at hospital discharge. [Clin Pract Cases Emerg Med. 2018;2(4):316–319.

INTRODUCTION

Phlegmasia cerulea dolens (PCD) is the most severe form of deep venous thrombosis (DVT) wherein the venous outflow of an extremity becomes completely obstructed.[1] It is considered an emergency condition that can lead to venous gangrene, limb loss, and even death due to massive pulmonary embolism. This condition has an extremely high morbidity and mortality, with 20-50% of cases requiring limb amputation and 35-40% of affected patients progressing to death.[1] PCD presents with the rapid development of a classic triad of symptoms consisting of worsening pain, swelling, and cyanosis of the affected limb.[2,3] These clinical manifestations are the result of massive venous congestion that develops when the most proximal deep vein of an extremity is occluded. This leads to fluid extravasation and increased interstitial pressure, which impairs capillary blood flow.[4] While overall uncommon, PCD of the lower extremity is well studied and characterized in the literature. PCD of the upper extremity is even more rare, with the current medical literature on the topic existing mostly in the form of individual case reports.

Unlike lower extremity DVT, which can occur spontaneously, upper extremity deep venous thrombosis (UEDVT) is typically a provoked phenomenon. UEDVT is most often associated with an indwelling foreign body such as a central venous catheter (CVC) or cardiac pacemaker/defibrillator.[5,6] Herein we present a case of PCD of the left upper extremity (LUE) developing in a young female with a permanent cardiac pacemaker who had recently undergone re-implantation of a dislodged atrial lead. We discuss the factors involved in distinguishing PCD from the much more common and less-morbid symptomatic DVT and the potential benefits of educating both providers and patients about this rare yet serious complication.

CASE REPORT

A 37-year-old female with a history of attention deficit disorder and postural orthostatic tachycardia syndrome (POTS) with neurocardiogenic syncope presented to our hospital's emergency department (ED) complaining of an abrupt onset of pain and paresthesias in her left arm, adjoining left chest wall, and inferior neck that began one hour prior to arrival. Her surgical history was significant for transvenous cardiac pacemaker placement one year earlier, pacemaker pocket revision six weeks prior, and re-implantation of a dislodged atrial pacer lead four weeks prior. She denied history of tobacco, alcohol, or illicit drug use. She also denied any history of prior stroke, prior blood clot, or recent trauma of any kind. On review of systems, she denied any recent fevers, headache, vision change, shortness

of breath, abdominal pain, nausea, or vomiting. Upon arrival, the patient's vital signs were unremarkable. Her physical exam was significant for mildly decreased sensation to light touch over the LUE and adjoining left superior anterolateral chest wall. She had 4/5 strength in all muscle groups of the LUE with otherwise normal strength and range of motion throughout her other extremities. She had no facial asymmetry, dysarthria, mental status changes, or pronator drift. Visual inspection of her LUE revealed mild skin erythema compared to the right side. She had 2+ peripheral pulses throughout and no obvious venous engorgement.

Because of the patient's acute onset of objective neurologic signs and concern about her unilateral dysesthesia and weakness, she received an expedited stroke workup that included laboratory studies as well as a non- contrast computed tomography (CT) of the head, perfusion CT of the head, and CT angiogram of the head and neck. Upon repeat examination when the patient was brought from the CT scanner to her room in the ED, she was noted to have developed significant edema and purple discoloration of her LUE from the shoulder to the fingers. Her LUE was cold to touch but her distal pulses were still palpable. She reported worsening pain in her entire LUE. Venous color-flow Doppler imaging had been ordered but was expedited after the findings of her repeat examination. It showed occlusive thrombus throughout the veins of the LUE including the subclavian, brachial, axillary, and basilic veins. Arterial color-flow Doppler imaging of the LUE showed normal arterial flow.

The remaining laboratory and imaging studies of her workup were negative. She was immediately started on intravenous unfractionated heparin, and vascular surgery was consulted emergently. Although the on-call vascular surgeon felt that prompt intervention was crucial, the patient's indwelling cardiac pacemaker presented a dilemma that required input from the on-call electrophysiologist regarding the safety of removing the patient's pacemaker. The electrophysiologist recommended against removal of the patient's pacemaker due to the significant risks associated with her severe and debilitating POTS and neurocardiogenic syncope.

The patient was admitted to the intensive care unit, and the following morning she was taken to the operating room by vascular surgery where a venogram revealed an occlusive thrombus of the left subclavian and brachiocephalic veins extending proximally to the junction with the superior vena cava. She underwent balloon venoplasty and ultrasound-guided, catheter-directed thrombolysis using tissue plasminogen activator, after which her symptoms completely resolved. Repeat venogram after 24 hours showed near-complete resolution of the clot with some mild stenosis noted in the proximal left subclavian vein (Image). During her hospitalization she underwent a full workup for thrombophilia, which was negative. The patient was discharged on hospital day four without any residual signs or symptoms. Prior to

CPC-EM Capsule

What do we already know about this clinical entity?
Phlegmasia cerulea dolens (PCD) is the most severe form of deep venous thrombosis wherein the venous outflow of an extremity becomes completely obstructed.

What makes this presentation of disease reportable?
This young patient presented with PCD of the upper extremity associated with an indwelling cardiac pacemaker.

What is the major learning point?
Patients with an indwelling central venous catheter or cardiac pacemaker are at risk of deep venous thrombosis that can be severe and even limb- or life-threatening.

How might this improve emergency medicine practice?
Awareness of this clinical entity may allow emergency physicians to make the diagnosis and initiate therapy promptly, increasing the chance of a successful outcome.

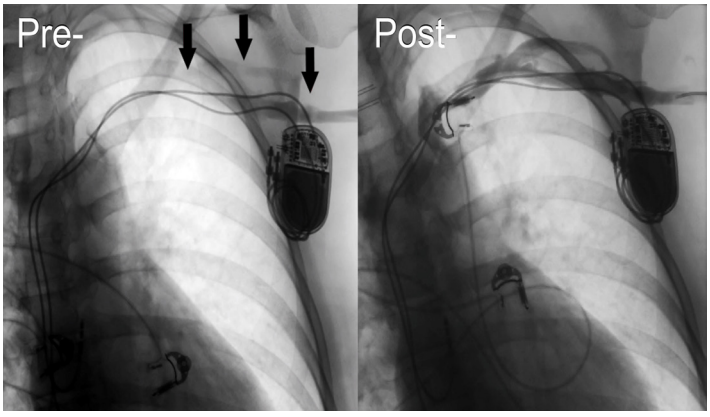

Image. Left subclavian venogram reveals total occlusion of venous outflow from the left upper extremity on pre-venoplasty and thrombolysis imaging. Arrows indicate the site of filling defects consistent with thrombosis. Imaging obtained 24 hours post-venoplasty and thrombolysis shows marked improvement with near total resolution of the clot.

discharge, she was started on oral rivaroxaban, which she was to continue for at least six months.

DISCUSSION

While UEDVT is known to be associated with indwelling devices such as CVCs and permanent cardiac pacemakers, we believe this is the first case report to detail acute venous outflow obstruction and development of PCD in a patient with an indwelling, permanent cardiac pacemaker. Nonetheless, we believe that this is an under-recognized and under-reported phenomenon that requires further attention and increased awareness. Because PCD is so strongly associated with its lower extremity variant—iliofemoral DVT—physicians are much less likely to consider it as a possibility in the upper extremity. Even in situations where a diagnosis of symptomatic UEDVT is made, physicians may not be aware that the condition is a form of PCD, with symptoms indicative of underlying microvascular (and eventually macrovascular) ischemia due to massive venous congestion.

The presenting signs and symptoms are largely dependent upon the degree of ischemia at the time. Very early in the clinical course, patients may present with more nonspecific signs and symptoms including minor arm discomfort, paresthesias, and weakness than can sometimes involve the adjacent chest wall and inferior neck.[7] In contrast to symptomatic DVT, which tends to develop over one or several days, PCD symptoms will progress rapidly over the course of hours to include the "classic triad" of signs and symptoms including worsening pain, swelling, and cyanosis of the affected limb.[8]

Several factors likely contributed to the successful outcome of this case. First, the patient presented to the ED immediately after her initial symptoms began. This allowed emergency providers to directly observe the rapid development of the classic triad of PCD in her LUE. Additionally, the patient was very open about disclosing her medical and surgical history, including her recent procedure to revise and re-implant her cardiac pacemaker. This disclosure directed her emergency providers to consider UEDVT as a possible etiology of her symptoms.

Subclavian vein stenosis is the most common complication of permanent cardiac pacemaker implantation, occurring in some 30-50% of patients.[6,9] However, this complication usually develops over a prolonged period of time, allowing for the development of collateral venous circulation. Therefore, venous thrombosis does not usually result in complete venous outflow obstruction in these individuals. There is no doubt that PCD in any setting is a rare condition. Nonetheless, in the immediate post-operative period before adequate collateral circulation develops, these patients are most prone to venous thrombosis and are at the highest risk of complete venous occlusion and PCD.[6,10] From 1993-2009, the rate of pacemaker placement in the United States increased by >55%.[11] With the increasing size of the elderly patient population in the U.S., this trend is likely to increase even more. For this reason alone, emergency physicians should become familiar with potential emergent complications of pacemaker placement, even those that are rare.

There is no firm consensus in the literature regarding the best approach to treatment of PCD. However, prompt initiation of therapeutic anticoagulation as the first step is considered standard of care. In cases of severe venous outflow obstruction with risk for progression to venous gangrene, systemic thrombolysis, surgical thrombectomy, and catheter-directed thrombolysis are available treatment options. Studies comparing the outcomes of each modality have found that catheter-directed thrombolysis is associated with the greatest reduction in risk of post-thrombotic syndrome and best chance of preventing limb loss.[3] When successful, catheter-directed thrombolysis has the additional benefit of allowing an indwelling cardiac pacemaker to remain in place. Our case was successfully managed using this method without any known complications or adverse effects after the intervention.

CONCLUSION

PCD is a rapidly progressive condition associated with exceptionally high morbidity and mortality. Prompt recognition of the diagnosis and initiation of therapeutic anticoagulation in the ED is paramount. Therefore, in patients presenting with upper extremity symptoms and a history of CVC, pacemaker, or defibrillator placement, emergency physicians need to consider the diagnosis of PCD. While PCD is a serious and potentially life-threatening condition, this case demonstrates that when recognized early and managed appropriately, positive outcomes are possible.

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 Ultrasonography of a Rare Cause of Hemoperitoneum

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A young woman presented to the emergency department with lethargy, hemodynamic instability, and diffuse abdominal tenderness. On point-of-care ultrasound (PoCUS), she was found to have intraperitoneal free fluid and a large pelvic mass, which were discovered intraoperatively to be hemoperitoneum due to ruptured vessels of a uterine leiomyoma. Although rare, a life-threatening, ruptured leiomyoma may be treated surgically if recognized in an expedient fashion. A PoCUS can aid the emergency clinician in prompt diagnosis. [Clin Pract Cases Emerg Med. 2018;2(4):320–322.]

INTRODUCTION

While uterine leiomyomas are common among reproductive-aged women, they rarely result in acute life-threatening events. However, the vessels overlying a leiomyoma may rupture leading to hemoperitoneum.[1,3-6,8-10] Patients with ruptured leiomyoma are often considered too unstable for confirmatory imaging and taken to the operating room without delay.[1-8] Although uncommon, this high-acuity condition is worth considering in the hemodynamically unstable, young female patient due to its amenability to surgical intervention. This report describes a case of ruptured leiomyoma that was promptly diagnosed with point-of-care ultrasonography (PoCUS) in the emergency department (ED) and discusses pathophysiology, diagnosis, and management from the emergency medicine perspective.

CASE REPORT

A 43-year-old, previously known to be healthy woman was brought to the ED by emergency medical services (EMS) after her husband found her to be confused. EMS provided oxygen by facemask and obtained peripheral venous access. Upon arrival to the ED, the patient demonstrated depressed mental status but when aroused, she complained of generalized weakness. Her heart rate was 80 beats per minute, blood pressure was 78/52 millimeters of mercury, respiratory rate was 14 respirations per minute, and temperature was 37.1° Celsius (98.8° Fahrenheit). The bedside glucose level was 170 grams per deciliter (g/dL). A cardiorespiratory monitor was attached to the patient, and one liter (L) crystalloid was infused with a pressure bag. The patient was somnolent but arousable, diaphoretic, and pale. Upon auscultation, the chest was clear and cardiac rhythm regular. Radial pulses were diminished in both upper extremities. There was mild, diffuse abdominal tenderness and a firm mass appreciated in the pelvis. Rectal examination revealed brown stool.

The emergency physician performed point-of-care ultrasonography (PoCUS), specifically a Rapid Ultrasound for Shock and Hypotension (RUSH) examination. The PoCUS demonstrated a moderate amount of free fluid (Image 1) and a well-circumscribed mass adjacent to the uterus (Image 2). Laboratory analysis was significant for a venous lactate level 2.5 millimoles per liter (mmol/L), white blood cell count 11.46 cubic milliliter (K/uL), hemoglobin 9.0 g/dL, and negative urine human chorionic gonadotropin. The patient's unstable condition precluded confirmatory computed tomography, and gynecology and general surgery were notified immediately for operative management of suspected hemoperitoneum. A repeat RUSH demonstrated an increased amount of free intraperitoneal fluid. While blood products were prepared, two additional L of crystalloid were infused.

Two hours after arrival, the urine output totaled 40 milliliters (mL), and the repeat hemoglobin fell to 5.0 g/dL. The gynecology consultant performed a bedside transvaginal sonogram and suspected the mass to be a uterine leiomyoma. The ED staff transfused the patient with three units of packed red blood cells, and collaborative gynecology and general surgery services brought the patient to the operating room (OR) for explorative

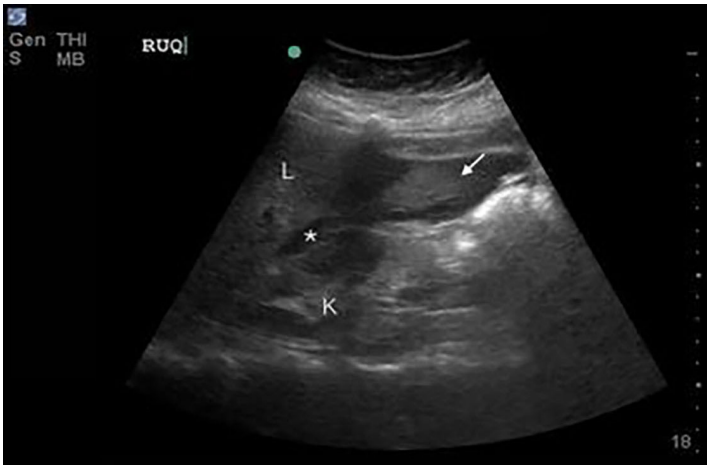

Image 1. A right upper-quadrant image in a coronal plane with free fluid (*) noted in Morison's pouch between the liver (L) and kidney (K). Hyperechoic (arrow) - material appreciated at liver tip is consistent with likely coagulated blood.

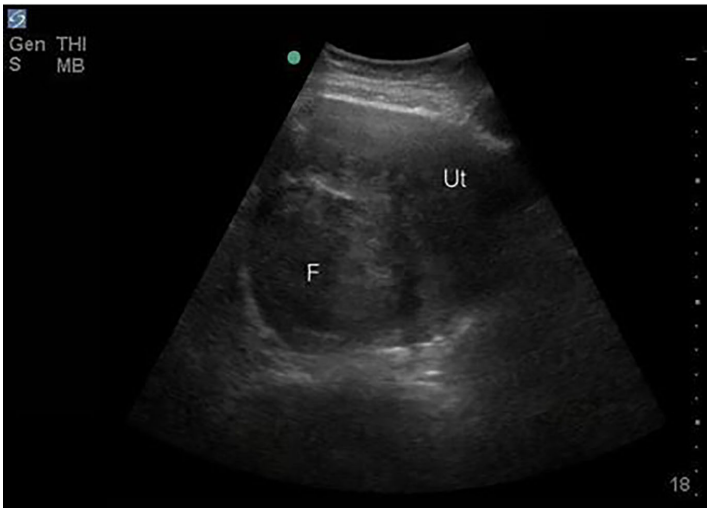

Image 2. A trans-abdominal pelvic image in a transverse plane with a large fibroid (F) noted within the uterus (Ut).

laparotomy. The gynecology team noted a leiomyomatous uterus and 2.5 L of blood in the peritoneum. The source of hemorrhage was localized to multiple bleeding vessels overlying a leiomyoma, and the gynecology team performed a myomectomy. The OR staff transfused an additional five units of packed red blood cells and three units of fresh frozen plasma. The patient had an uneventful postoperative course and hospital stay. She was discharged from the hospital several days later and appeared healthy at postoperative checkups.

DISCUSSION

Although uterine leiomyomas are common among reproductive-aged women, spontaneous life-threatening bleeding is exceedingly rare. Most reported cases seem to occur without insult,[3-7,10] although one occurred after a large bowel movement,[1] and another involved a laceration to the leiomyoma after a "vigorous coital experience."[2] There has also been report of bleeding that may have been exacerbated by pregnancy or delivery.[8,9] These cases are consistent with the theory that increased intra-abdominal pressure may increase the risk of rupture of overlying vessels.[3,4] The clinical presentation typically involves sudden hemodynamic collapse with abdominal pain, tenderness, or distention. Patients are uniformly found to have free intraperitoneal fluid on abdominal[4-8] or pelvic ultrasonography,[1,3] frequently with visualization of the leiomyoma.[1,3,5-7]

The most frequent cause of hemoperitoneum related to uterine leiomyoma is the rupture of an overlying, superficial artery or vein.[1,3-10] However, there has been report of spontaneous bleeding from leiomyoma itself.[7] From the emergency medicine perspective, the management approach should be as for any unstable patient with signs of peritonitis and intraperitoneal free fluid on RUSH – resuscitation with blood products and consultation with appropriate surgical consulting services. The definitive treatment may include ligation of the bleeding vessel,[5,8] myomectomy,[1-4,6] or hysterectomy,[7,9,10] depending on origin of bleeding and age and reproductive status of the woman.

CPC-EM Capsule

What do we already know about this clinical entity?
Uterine leiomyoma are common among reproductive-age women and typically presents with pelvic pain or non-life threatening vaginal bleeding

What makes this presentation of disease reportable?
Rarely, uterine leiomyoma may rupture and present with hemoperitoneum, a potentially life-threatening condition

What is the major learning point?
A female patient who presents with hemodynamic instability and abdominal pain, tenderness, or distention may be promptly diagnosed with ruptured uterine leiomyoma using point-of-care ultrasonography (PoCUS)

How might this improve emergency medicine practice?
Prompt PoCUS diagnosis of ruptured uterine leiomyoma may expedite urgent resuscitation with blood products and transfer to the operating room for definitive intervention

CONCLUSION

Despite uterine leiomyoma being a common tumor in young women, the rupture of associated blood vessels resulting in hemoperitoneum appears to be exceptionally rare. However, this condition may be fatal, and swift action is required for diagnosis and surgical intervention. This case report is unique in that it presents sonographic images to demonstrate the utility of PoCUS for the diagnosis of ruptured uterine leiomyoma. This diagnosis should be considered in any female patient who presents with hemodynamic instability and abdominal pain, tenderness, or distention. A quick diagnosis with PoCUS may expedite urgent resuscitation with blood products and transfer to the OR for definitive intervention.

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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FascIOtomy: Ultrasound Evaluation of an Intraosseous Needle Causing Compartment Syndrome

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Intraosseous (IO) needles are used in critically ill patients when it is not possible to quickly obtain venous access. While they allow for immediate access, IO infusions are associated with complications including fractures, infections, and compartment syndrome. We present a case where point-of-care ultrasound was used to quickly identify a malfunctioning IO needle that resulted in compartment syndrome of the lower extremity. [Clin Pract Cases Emerg Med. 2018;2(4):323–325.]

INTRODUCTION

Intraosseous (IO) needles are often used in high-acuity patients for whom having vascular access is essential. Malfunctioning IO needles may be associated with patient harm as there is decreased medication delivery and complications secondary to the IO placement, such as compartment syndrome. Further, identifying compartment syndrome may be difficult in critically ill patients who are unable to communicate and for whom serial compartment checks may not be possible or a priority due to competing medical priorities. For this reason, rapid identification of IO needle functionality is needed and can be achieved using point-of-care ultrasound (POCUS). POCUS use has been described as a tool to facilitate evaluation of an IO needle by looking for Doppler signals in the area surrounding the IO during infusions. POCUS can add to patient care and safety by identifying malfunctioning IO needles and potentially preventing complications. We present a case report in which POCUS aided in rapid recognition of IO needle malfunction and in the identification of compartment syndrome.

CASE REPORT

A 63-year-old male was brought into the emergency department (ED) after being found unresponsive. Paramedics in the field noted that the patient was obtunded, with a Glasgow Coma Score of 3 and a blood sugar of 33 milligrams per deciliter (mg/dL). After multiple unsuccessful attempts were made to gain intravenous (IV) access, paramedics used the EZ-IO® to place a 45 mm IO needle into the left proximal tibia(Image 1A) and administered dextrose at a concentration of 50% (D50). Upon arrival to the ED, the patient remained hypoglycemic and unresponsive. He was intubated, and two 50 mL doses of D50 were administered through the IO needle. Nurses noted resistance upon subsequent administration of medications.

POCUS was performed to evaluate the functionality of the IO needle. A high-frequency linear probe (5-10 MHz; SonoSite® M-turbo) with color Doppler was used to evaluate the area proximal and distal to the IO access in transverse (short) plane of the tibial bone. Color Doppler showed absence of flow in the IO space during injection of a small amount of normal saline, concerning for inappropriate IO needle placement (Image 2). Subsequently, the tibial IO needle was removed and a second, 45 mm IO needle was placed into the right humerus (Image 1B). The patient was resuscitated and stabilized, receiving medications without complication through the humeral IO infusion.

Fifteen minutes after arrival to the ED, the patient's left lower extremity was noted to be cool and mottled. Examination of the extremity showed firm compartments and decreased peripheral pulses concerning for compartment syndrome. The deep posterior compartment pressure was 85 mmHg. A radiograph showed that the IO needle had punctured both the anterior and posterior cortex of the tibia, extending 2 mm beyond the posterior cortex (Image 3). The patient was

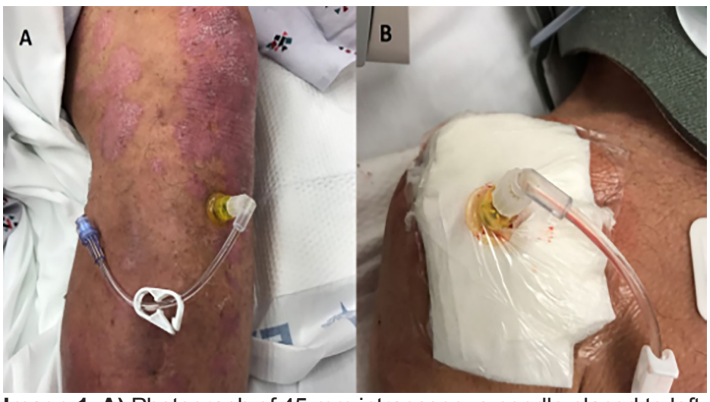

Image 1. A) Photograph of 45 mm intraosseous needle placed to left proximal tibia. **B)** Photograph of 45 mm intraosseous needle inserted to right proximal humerus.

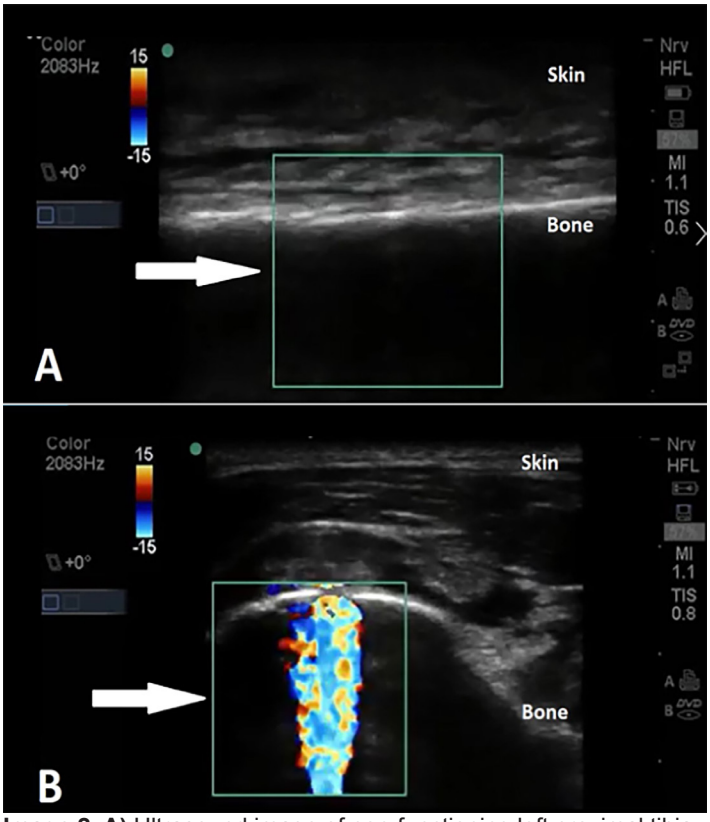

Image 2. A) Ultrasound image of non-functioning left proximal tibia intraosseous needle demonstrating no flow (arrow). **B)** Ultrasound image of properly functioning right humerus intraosseous needle demonstrating flow (arrow).

emergently taken to the operating room (OR) by orthopedic surgery for a lower extremity, four-compartment fasciotomy. The procedure demonstrated bulging muscle in all compartments without necrosis. In the OR, all compartments successfully underwent decompression with subsequent return of 2+ palpable distal pulses.

CPC-EM Capsule

What do we already know about this clinical entity?
Intraosseous (IO) needle functionality can be assessed using point-of-care color Doppler ultrasound to evaluate for flow in the IO area during infusion through the IO needle.

What makes this presentation of disease reportable?
This is the first reported case in which ultrasound has helped to identify a malfunctioning IO needle and aided in the rapid recognition of a compartment syndrome.

What is the major learning point?
Just as radiographs are used to confirm proper placement of central venous catheters, ultrasound can be used to evaluate placement and functionality of IO needles.

How might this improve emergency medicine practice?
Point-of-care ultrasound can be used to improve patient safety by rapidly identifying malfunctioning IO needles and preventing complications such as compartment syndrome.

DISCUSSION

IO needles are indicated in critically ill patients for whom peripheral venous access is not possible. Correct placement of the IO needle with the appropriate size needle are both essential to ensure proper function. IO needles may be placed in any large bone with palpable landmarks – usually the distal tibia, proximal tibia, distal femur, or proximal humerus. A 15-mm long needle (pink) is recommended for children (3 kg-39 kg), whereas a 25-mm long needle (blue) is recommended in adults. A 45-mm long needle (yellow) is considered the ideal length for humeral placement in patients with excessive soft tissue or musculature overlying the insertion site.

IO access is considered a fast and effective alternative to peripheral and central lines in critically ill patients. This case, however, showcases a potential risk and complication of its use as well as the value of POCUS in mitigating these risks. The most common complications of IO needle insertion include insertion site infection, hematoma formation, extravasation of medications, and compartment syndrome.[1,2] The incidence of extravasation of IV medications administered through the IO needle is 3.7%.[3] Compartment syndrome is rare, occurring

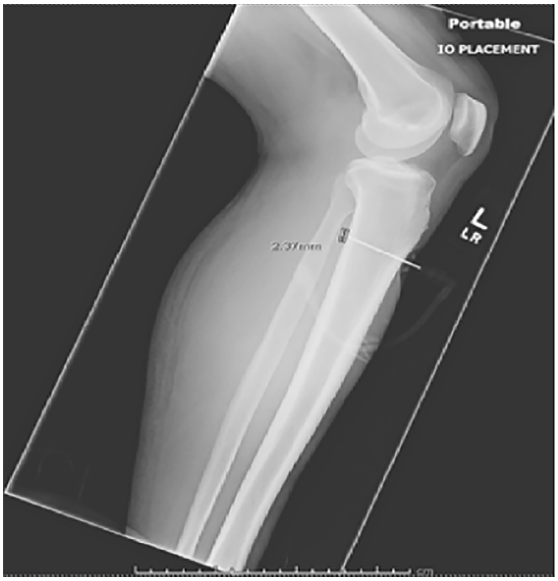

Image 3. Radiographic demonstrating intraosseous needle extending 2 mm past posterior cortex of tibia.

in 0.6% of cases.[3-5] Nonetheless, compartment syndrome is considered a surgical emergency; thus, prompt recognition of an infiltrated or mispositioned IO needle can avoid this complication and prevent the morbidity associated with neurovascular compromise of the affected limb.[6,7]

POCUS is an adjunct that can be used to evaluate functionality of IO needles and ensure safety and efficacy of any medications that may be administered through this access site.[8] To assess the functionality of the IO needle, a high-frequency linear transducer is placed near the IO site, usually in a transverse plane, and the sample box is manipulated to include the IO space and ideally include the IO needle and surrounding tissue posteriorly. A normal saline flush is then pushed quickly through the IO needle while obtaining real-time imaging focusing on the IO space. Color Doppler signal in the IO space signifies a functioning IO needle. In contrast, absence of signal within the IO space, or signal in the extraosseous compartments including the subcutaneous tissue or muscle signifies extravasation.

CONCLUSION

Intraosseous needles are often placed in critically ill patients when lifesaving medications need to be administered emergently. A non-functioning or mispositioned IO needle can lead to inadequate medication administration as well as complications, as in our case. It is important to frequently check for signs of infiltration of medications from the IO needle as well as to re-evaluate the neurovascular status of the patient's limb. In patients with altered mental status in whom a thorough neurovascular exam is difficult, or in an obese patient whose landmarks and compartments may be hard to evaluate, the clinical signs of compartment syndrome can be difficult to assess. By using POCUS to evaluate functionality of the IO needle immediately after placement, clinicians can rapidly confirm properly placed and functioning IO devices, ensuring adequate medication delivery in critically ill patients and potentially avoiding serious 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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Fungal Rhino-orbital Cerebritis in a Patient with Steroid-induced Ketoacidosis

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Fungal rhino-orbital cerebritis is a devastating opportunistic invasive disease. Survival requires urgent diagnosis. Thus, all patients at risk who present with rhinosinusitis-type symptoms and have co-morbid conditions that decrease their immunocompetence should trigger the clinician's consideration of this disease. Treatment includes antifungals and emergent surgical debridement. [Clin Pract Cases Emerg Med. 2018;2(4):326–329.]

INTRODUCTION

Fungal rhino-orbital cerebritis is an uncommon but devastating opportunistic, invasive fungal infection with a grim prognosis. Survival requires early diagnosis and aggressive treatment with antifungals and surgical debridement. The following case demonstrates the importance of considering this disease as part of the differential diagnosis especially in patients who are immunocompromised. This patient was initially diagnosed with conjunctivitis and then was thought to have developed possible anaphylaxis. Her rapidly progressing physical exam findings helped lead to making the unfortunate diagnosis.

CASE REPORT

A 52-year-old woman with multiple comorbidities, including obesity and chronic use of prednisone presumably for pulmonary fibrosis, originally presented to an urgent care center two days prior to presenting to our academic hospital and was prescribed polymyxin for presumed conjunctivitis. The patient then presented to our community campus emergency department (ED) because she felt that her "head is swollen and feels like her throat [is] starting to swell." She believed she was having an allergic reaction; after using her EpiPen® without resolution, she came to the ED to be treated. On initial exam her vital signs were blood pressure 160/90 millimeters of mercury, pulse 120 beats per minute, temperature of 36.8°C, respiratory rate of 22, and oxygen saturation of 100% on room air. She was noted to have posterior oropharyngeal edema; she was treated for possible anaphylaxis but did not improve. During her work-up it was discovered that she was developing acute vision loss in the right eye. She was transferred to our downtown campus ED for ophthalmological specialty evaluation.

Over the course of a few hours, exam findings progressed to include severe bilateral periorbital swelling and severe chemosis. Repeated extraocular motor tests revealed an initial unilateral ophthalmoplegia that later progressed to bilateral cranial nerves III, IV, and VI palsies. Pupillary light- response exam revealed relative afferent pupillary defects suggesting retinal and/or optic nerve involvement. Her labs were consistent with steroid-induced diabetic ketoacidosis (DKA), white blood cell count 18,400 cells per microliter with 96% neutrophils, and acute kidney injury. Computed tomography (CT) only revealed right proptosis, right periorbital soft tissue swelling, and mucosal thickening within all the paranasal sinuses (Image); CT was unable to characterize the cavernous sinus without venous contrast phase.

Ophthalmology was consulted and reported a funduscopic exam that revealed retinal pattern consistent with right eye central retinal artery occlusion. Otolaryngology was consulted and performed a nasal endoscopy in the ED, finding soft black crusting on the septum and turbinates bilaterally with positive

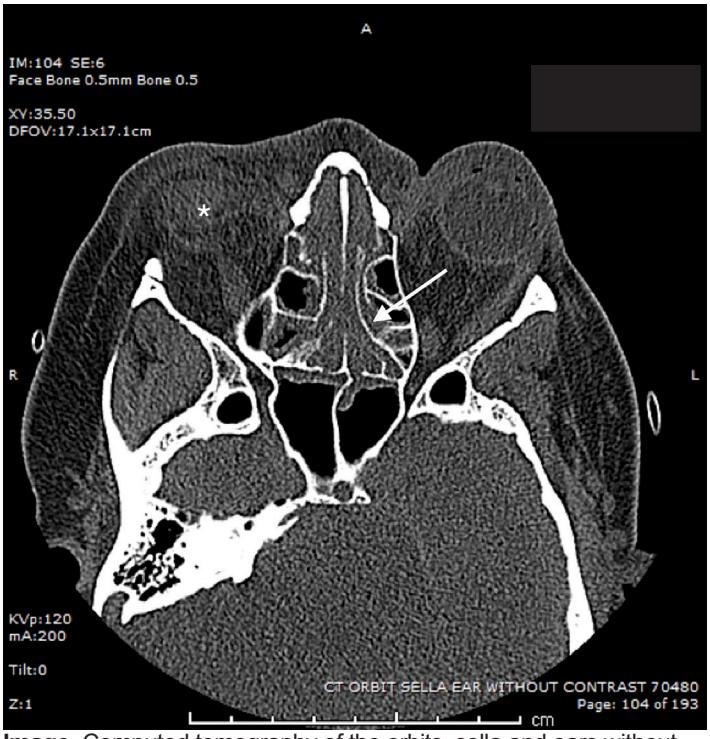

Image. Computed tomography of the orbits, sella and ears without contrast. Right periorbital soft tissue swelling (*), and mucosal thickening within all the paranasal sinuses (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Fungal rhino-orbital cerebritis is a rare and deadly infection.

What makes this presentation of disease reportable?
The initial misdiagnosis and rapid progression of findings within the course of this patient's emergency department visit illustrates the difficulty of recognizing this rare infection.

What is the major learning point?
Identifying at-risk patients and performing a thorough ear, nose, throat, and cranial nerve exam may help facilitate an early and accurate diagnosis.

How might this improve emergency medicine practice?
Fungal rhino-orbital cerebritis should be in the differential diagnosis when immunosuppressed patients present with sinusitis-type symptoms.

potassium hydroxide (KOH) preparation. In consideration of the patient's clinical presentation, this was presumed to be most consistent with necrosis from invasive mucormycosis. The patient was admitted to the medical intensive care unit and was treated with broad-spectrum antimicrobials, including amphotericin B liposome. Surgical debridement was discussed with the patient and family who were informed of a likely chance of mortality regardless of intervention, considering the likely cavernous sinus involvement. The patient declined surgical intervention, choosing comfort care, and died eight days after admission.

DISCUSSION

The constellation of cranial neuropathies, periorbital inflammation, necrotic nasal mucosa, positive KOH preparation, steroid-induced immunosuppression, and DKA eventually led to the clinical diagnosis of fungal rhino-orbital cerebritis, most consistent with the classic presentation of rhinocerebral mucormycosis.[1] Most estimates of incidence of this disease are expected to be underestimated because of the difficulty with diagnosis; the majority of estimates are based on case series.[2, 3] For example, a review of four cases found that it took on average seven days from time of presentation to diagnosis.[4] No large epidemiologic study in the United States specific to rhino-orbital cerebritis was found on our literature review, but one study in France found only 530 mucormycosis cases between 2001 and 2010, less than half of them involving the rhino-orbito cerebral system.[5]

Our patient's symptoms initially began with periorbital swelling and conjunctival infection, which was treated as an allergy initially. Indeed, the initial symptoms of rhinocerebral mucormycosis are usually mistaken for bacterial or viral sinusitis or orbital cellulitis. The absence of fevers in up to 50% of cases further contributes to the delay of the correct diagnosis.[1] Additional considerations in the differential diagnosis would be based on the timing of presentation. Early on, the patient may have little in the way of neurologic findings; thus, infections such as sinusitis, and facial or orbital cellulitis, as well as upper respiratory and maxillary odontogenic infections, would be included in the differential. Non-infectious considerations would include allergic reactions.

If neurologic findings are present, then one must consider intracranial infections, mass lesions and cavernous sinus thrombosis. The diagnosis of invasive fungal rhino-orbito cerebral mucormycosis became only suspected with development of proptosis, chemosis, vision loss and bilateral

ophthalmoplegia. Further supportive of the diagnosis was the recognition of her steroid-induced hyperglycemia and acidosis, an environment usually required to allow this ubiquitous fungus to spread from the sinuses to the orbit, invade the orbital musculature, optic nerve or arterial supply, and to cause ophthalmoplegia, central retinal artery occlusion, and afferent pupillary defects, respectively.[1,6,7] If bilateral ophthalmoplegia ("bilateral frozen globes") later develops, or if cranial nerves are involved, as was seen with our patient, it is an ominous sign that the fungus has invaded the cavernous sinus.[1,6]

Definitive diagnosis of mucormycosis must be through surgical exploration and biopsy. Typical laboratory studies such as complete blood count, basic metabolic panel, lactic acid and blood gas analysis may help to suggest sepsis or identify co-morbid conditions such as DKA that increase the risk for this disease but would not be diagnostic. Cultures rarely are useful because Mucorales species are ubiquitous in nature and a common contaminate causing frequent false positives; false negatives are also common because the hyphae are fragile and easily crushed during the culturing process. There are no serological studies that can help with rapid diagnosis, which is why up to half of cases aren't diagnosed until autopsy.[1]

Unfortunately, radiographic studies are often negative or only depict subtle findings such as thickening of the sinus mucosa as was seen in this case (Image).[6,8] When considering this diagnosis, obtaining a CT venography study of the brain may allow for the recognition of cavernous sinus involvement, although in the absence of fever or other infectious clues it would not be definitive proof of septic etiology. Magnetic resonance imaging is an alternative to CT but would take longer to obtain.

To improve chances of successful treatment, the diagnosis must be made as soon as possible, the underlying risk factors such as hyperglycemia or acidemia reversed, and antifungal therapy and surgical debridement must begin immediately.[8] There are no formal surgical guidelines; however, in many of the reported cases complete orbital exenteration was required, a course that our patient did not pursue.[6] The antifungal agent of historical choice, presuming mucormycosis, is amphotericin B.[9-11] Case reports suggest that adjunctive hyperbaric oxygen therapy may improve outcomes, possibly by increasing neutrophilic activity.[1,12] In one large epidemiologic review of 196 patients, rhinocerebral mucormycosis had a 62% mortality. Those who received no treatment had only a 3% survival rate.[13]

One limitation to this case report was our inability to obtain additional information about the patient's initial presentation to an urgent care center two days prior to her presenting to the ED. Based on the history provided to us by the patient and her family, as well as a review of her medications, we were able to determine her diagnosis and treatment prior to our encounter; but it would have been informative to have an objective medical record that included her initial physical exam findings.

CONCLUSION

In summary, we present a case of fungal rhino-orbital cerebritis, presumably from mucormycosis. Patients with risk factors (e.g., immunosuppression or DKA) who present with symptoms consistent with sinusitis should have a complete head, ears, eyes, nose and throat evaluation, and a thorough cranial nerve exam.[14] To maximize chance of survival, urgent diagnosis, reversal of underlying predisposing risk factors, prompt antifungal chemotherapy, and surgical debridement are all critical.[4,14]

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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Bulbar Muscle Weakness in the Setting of Therapeutic Botulinum Injections

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We present a case of new-onset bulbar muscle weakness in the setting of therapeutic botulinum injections for spasticity in a teenaged patient with cerebral palsy. Through a careful history, a systemic effect of the local injections was suspected, and the patient's symptoms improved with a decrease in the dosing of the botulinum injections. [Clin Pract Cases Emerg Med. 2018;2(4):330–333.]

INTRODUCTION

Though uncommon, systemic botulinum toxicity from local injections has been reported. With expanding use of therapeutic botulinum, it is important for emergency physicians (EP) to be aware of possible complications, the usual presentation, and management. As with foodborne botulism, recognition is key to preventing complications.

CASE PRESENTATION

A 16-year-old girl with cerebral palsy, secondary to prenatal hypoxic brain injury, was brought into the emergency department (ED) by her parents because of progressive, nighttime gagging. The patient's parents related that over the prior 10 months she had been having episodic and worsening episodes of choking on her own saliva at night while falling asleep and during sleep. These episodes would progressively worsen over five to six weeks and then improve. A few weeks later, the cycle would repeat.

During the day, the patient had no difficulty clearing secretions. The patient did not have fevers, chills, cough, or sputum production. She was taking her baseline diet. The patient's speech was unchanged. The parents did endorse waxing and waning generalized weakness in the patient over the prior several months, the course of which tracked with the gagging. The patient's only medication was famotidine, which she took by mouth. She had no allergies and had no other medical or surgical history. The patient was enrolled in high school, where she was doing well and had many friends. The parents strongly doubted any drug or alcohol use.

The patient's parents related that her care was managed primarily by her neurologist with bi-monthly appointments. The choking episodes had, without fail, resolved by the time of each visit. There had been no changes in the patient's health and no new illnesses or diagnoses for the preceding five years. The parents mentioned that at each visit for the prior two years the patient had been receiving local botulinum toxin injections in her arms and legs to relieve her muscle spasticity. The parents relayed that the neurologist had been increasing the dosing of botulinum over the prior year in an attempt to achieve better spasm control.

Physical exam revealed a friendly girl in no acute distress. No gagging or coughing was noted. Her vitals signs showed an oral temperature of 36.4 degrees Celsius, a heart rate of 71 beats per minute, a blood pressure of 115/78 millimeters of mercury, a respiratory rate of 14 breaths per minute, and an oxygen saturation of 100% on room air. The oropharynx was clear. Oral mucosa was moist without any obvious lesions. There was no drooling or difficulty clearing secretion, and she had an intact gag reflex. Extraocular movements were intact with no ptosis, and pupils were reactive to light. The patient's tongue projected midline, and speech was at baseline per parents. There was no lymphadenopathy in the head or neck. The patient's lungs were clear. The remainder of the exam was notable only for diffuse symmetrical muscle spasticity in the upper and lower extremities. A chest radiograph showed no consolidation or evidence of foreign body.

After careful clarification of the time course of the patient's gagging episodes, it was suggested to the patient and her parents that she might have been having bulbar muscle weakness from botulinum toxin injections, which became noticeable when she was falling asleep, or asleep, and her muscle tone was already relaxed.

DISCUSSION

The indications for and therapeutic uses of botulinum toxin continue to expand.[1,2] Beyond its cosmetic effects, botulinum toxin has shown varying degrees of success in the treatment of symptoms in a variety of diseases, including Hirschsprung's disease, cervical dystonia, spasticity, trigeminal neuralgia, amyotrophic lateral sclerosis, and hyperhidrosis.[3,4] The near-ubiquity of therapeutic botulinum toxin injections belies the fact that it remains, based on dosage, the most lethal poison known.[5] The estimated human median lethal dose (LD_{50}) is 1.3-2.1 nanogram/kilogram (ng/kg) intravenously (IV) or intramuscularly (IM), 10-13 ng/kg inhaled, and 1 ng/kg oral.[6,7] Given this, the probability that the EP will encounter adverse effects from its use continues to rise.

Botulinum toxin is produced by the gram-positive anaerobe *Clostridium botulinum*, a spore-forming bacterium that is ubiquitous in soil and water. The spores are usually dormant and quite hardy. The toxin, by contrast, is heat labile and is quickly destroyed by a few minutes of boiling water. The toxin is composed of light and heavy protein chains, and it exerts its effect in presynaptic nerve terminals by irreversibly preventing the releases of acetylcholine, resulting in flaccid muscle paralysis and anticholinergic autonomic effects.[6]

If a patient has been exposed to enough toxin, he will then develop bulbar palsies that can include dysphonia, dysarthria, dysphagia. Diplopia and ophthalmoplegia are also prominent findings, as well as dilated, non-reactive pupils. The patient may also endorse a sore throat and a dry mouth. Tendon reflexes and mental status are preserved, and the patient remains afebrile. Orthostatic hypotension or autonomic instability may be present. Flaccid extremity paralysis follows. Respiratory muscle weakness leading to respiratory failure is a potential life-threatening event in any patient with botulism.[6-8]

While the general practice is to give therapeutic botulinum toxin in the outpatient setting, adverse effects can occur. The therapeutic effect of botulinum injections occurs at the neuromuscular junction, which is the same mechanism by which the disease functions; therefore systemic toxicities suggestive of botulism are exaggerations of the therapeutic effect.[1] One study of adverse events in therapeutic botulinum injections found that in such events, bulbar muscle weakness was present in 15% of patients, respiratory issues in 38%, ocular problems in 23%, bowel or bladder problems in 15%, and other muscle weakness in 15% of patients. Patient were also found to have infections from the injections 15% of the time (Table).[2]

Iatrogenic botulism is one of the seven types of botulism described by the World Health Organization (WHO).[8] The other forms of botulism recognized by the WHO include the four classic distinct disease forms: foodborne botulism; infant botulism (for which nearly half of reported American cases have been in the state of California); wound botulism; and botulism of undetermined origin (in a patient over one year of age without a clear food or wound source). The WHO also recognizes two other potential forms of the disease, inhalational botulism (were it to be aerosolized as a weapon) and waterborne botulism (which is theoretical as water treatment inactivates the toxin).[6,8]

Foodborne botulism often presents first with nondescript gastrointestinal symptoms, including nausea, vomiting, and

CPC-EM Capsule

What do we already know about this clinical entity?
Iatrogenic botulism is one of the seven types of botulism recognized by the World Health Organization and is caused by therapeutic botulinum toxin injections.

What makes this presentation of disease reportable?
The world's deadliest toxin, botulinum toxin, is used for an expanding number of therapeutic purposes, and systemic effects, similar to other botulism entities, can be seen.

What is the major learning point?
Though uncommon, iatrogenic botulism should be kept on the differential for patients who present with muscle weakness and a history of botulinum toxin injections.

How might this improve emergency medicine practice?
After reviewing this case study and overview of botulism, emergency physicians may be more likely to consider therapeutic botulinum injections as the etiology of a patient's muscle weakness.

Table. Types of symptoms among patients with adverse effects from botulinum toxin injections for spasticity.[2]

Symptom category	Percentage
Muscle weakness	15
Oropharyngeal	15
Respiratory	38
Ocular	23
Bowel/bladder-related	15
Infection	15

abdominal pain and distension. These may occur at any point from a few hours to eight days after ingestion of the culprit item. Outbreaks of foodborne botulism have been caused by homemade foods, mishandled foods, and even industrially produced foods.[9] Botulism has also been caused by consumption of prison-made wine (known as "pruno" or "hooch," among other terms).[10]

Infant botulism is the most common form of botulism in the United States. It presents as constipation followed by flaccid neuromuscular paralysis. Cranial nerves are usually first affected, followed by peripheral and respiratory function. Honey, corn syrup, vacuum bag dust, and soil have been identified as sources of *C. botulinum* spores; it is believed that the infant gastrointestinal (GI) tract lack the bile acids and gut flora that normally inhibit *Clostridium* growth.[6]

Wound botulism presents identically to foodborne botulism, without GI symptoms. The culprit wounds are often in avascular areas or due to a crush injury, though most commonly they are due to intravenous drug use. A particularly high incidence has been noted among "skin popping" users of black tar heroin.[7]

Iatrogenic botulism, like wound botulism, can present with symptoms ranging from the very mild to impending respiratory compromise without GI symptoms.[6] It is due to therapeutic injection of one of the three FDA-approved formulations of the toxin. A clinician should be suspicious of botulism based on a history that includes possible exposures via any of the previously described routes. Findings of cranial nerve palsies and extremity weakness support the diagnosis. Routine laboratory values and imaging, including brain computed tomography and magnetic resonance imaging, are nondiagnostic in botulism. Cerebral spinal fluid studies will also be nondiagnostic (though can have a slightly elevated protein count).[6] Stool, serum and, as applicable, wound samples should be sent for anaerobic cultures and botulinum toxin detection. Consultation with a neurologist is also necessary to obtain electromyography, as botulism has a typical pattern of small units with subtle signs of denervation, and increased jitter with blocking.[1] Patients with respiratory compromise may need to be intubated and mechanically ventilated.[9]

A clinician who suspects botulism should contact the state health department, and they will be referred to the Botulism Clinical Consultation Service at the Centers for Disease Control and Prevention (CDC).[9] The CDC will send botulinum antitoxin from their stockpiles, and will also help coordinate epidemiologic research to quarantine possible outbreaks. Antitoxin does not reverse symptoms but rather prevents further progression. It is imperative that antitoxin be given as soon as possible if botulism is suspected, prior to confirmatory testing. Subsequent doses may be given at two-to-four hour intervals based on clinical disease progression.[6,9]

There is a human botulism immune globulin available for infant botulism, and it is available only from the California Department of Health Services Infant Botulism Treatment and Prevention Program. In wound botulism, surgical debridement is the primary therapy. Penicillin G should also be administered.[6] Iatrogenic botulism can present along a wide spectrum of symptoms, and the management should be in relation to the severity of the symptoms.

CONCLUSION

The patient was clinically stable and was monitored in the ED for several hours. The patient and her parents were offered admission, but they declined because they felt that the patient was on an "upswing" ahead of an appointment with her neurologist in two weeks. The patient was seen by her neurologist the next week, and he reduced the dose of botulinum toxin being used for her spasticity. The patient's gagging did not return.

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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Temporary Memory Steal: Transient Global Amnesia Secondary to Nephrolithiasis

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Transient global amnesia (TGA) is typified by an abrupt and transient anterograde amnesia, "with repetitive questioning and often variable retrograde amnesia persisting up to 24 hours." A 54-year-old male presented to our emergency department with paroxysms of left-sided flank pain, suggestive of renal colic. Computed tomography (CT) of the abdomen/pelvis revealed a three-millimeter left ureterovesicular-junction calculus. Pain control proved difficult, necessitating multiple doses of opioid and non-opioid analgesia. Subsequently, the patient developed repetitive questioning and perseveration with anterograde amnesia with a negative CT brain and unremarkable further workup. He experienced a complete resolution of symptoms within a 24-hour period, with a discharge diagnosis of TGA secondary to nephrolithiasis. This is the third case of TGA attributed to nephrolithiasis in the medical literature. [Clin Pract Cases Emerg Med. 2018;2(4):334-337.]

INTRODUCTION

Transient global amnesia (TGA) is an acute-onset clinical entity typified by an abrupt and transient anterograde amnesia, "with repetitive questioning and often variable retrograde amnesia persisting up to 24 hours."[1,2] Inherent to the diagnosis of TGA is the preservation of neurologic functioning including procedural memory (ability to remember and apply a series of steps to a task).[2] Diagnostic criteria for TGA demand that there be "no clouding of consciousness, other impairments of cognition, or a history of epilepsy or head trauma."[3] Several studies have postulated that the mechanisms of TGA are comparable to processes underlying "cerebral ischemia, epilepsy, and migraines, or may arise from disturbance of venous hemodynamics."[4] Yet more than a century after it was first described, there is no definitive evidence supporting any of these mechanisms.

Recently, a growing body of evidence supports the role of emotional and psychological factors as precipitating events in up to 90% of reported TGA cases.[5,6] Recent magnetic resonance imaging (MRI) data on individuals following an episode of TGA show development of small hippocampal lesions as a result of increased vulnerability of selective neurons from metabolic stress, further supporting the postulation that TGA is triggered by transitory "stress-induced inhibition of memory formation in the hippocampus."[4] TGA occurs in men and women equally with a mean age of 50-70 years. The alarming symptoms often culminate in an emergency department (ED) visit with the incidence ranging from 3-10 per 100,000 patients per year.[7] No laboratory investigations or imaging modality can confirm the diagnosis of TGA. Thus, the diagnosis "relies on a detailed clinical history, cognitive evaluation, and physical examination."[1] The diagnosis is also dependent on eliminating other life-threatening etiologies including toxidromes, metabolic derangements, cerebrovascular accident (CVA), seizure activity, and central nervous system (CNS) infections.[8] Although, there is no specific treatment for TGA, when alternative diagnoses are suspected, focused investigation, treatment, and secondary prevention should be pursued to address those clinical entities. TGA episodes are self-limited, and improvement is noted within 24 hours without any intervention with favorable short- and long-term prognosis.[1]

CASE REPORT

A 54-year-old male with a past medical history of nephrolithiasis and hypertension arrived to our ED at 9:55 a.m.

with complaints of left flank pain with nausea and vomiting. The patient noted that he had experienced similar paroxysms of pain with his previous episode of nephrolithiasis. He described his current symptoms as starting suddenly four hours prior to arrival to the ED. The pain was localized to his left flank with no alleviating or exacerbating factors, and he described it as a sharp sensation with radiation to the left inguinal region. The patient rated this pain numerically as a 10/10 in severity with associated nausea and episodes of non-bloody, non-bilious vomiting.

Upon arrival, his vitals revealed a blood pressure of 179/87 millimeters of mercury, pulse 63 beats per minute, respiratory rate 16 breaths per minute, 100% oxygen saturation on room air and temperature of 97.0˚F. His blood glucose was 132 milligrams per deciliter (mg/dL). Lab work obtained included a complete blood count, complete metabolic panel, and urinalysis, which were found to be unexceptional aside from microscopic hematuria. The patient had a similarly unremarkable physical examination. A computed tomography (CT) of his abdomen and pelvis was obtained, and attempt at parenteral analgesia with intravenous (IV) ketorolac 15mg followed by hydromorphone 1mg IV were done. The CT revealed a three-millimeter obstructing calculus at the left ureterovesicle junction (UVJ) with left-sided perinephric stranding and mild hydronephrosis.

The patient continued to have significant pain despite continued opioid dosing. Shortly after his CT, the patient's wife alerted ED staff that he was repeatedly asking her, "How did I get here?" and "Why am I here?" The patient was evaluated, and his physical examination was repeated to reveal no neurologic deficits. His presentation and physical examination were not consistent with opioid intoxication or medication side effect as he maintained his respiratory drive at baseline as well as his procedural memory, and was not opioid naïve, having received similar medications during his previous episode of renal colic. It was revealed at this point that the patient did not have any recall about his IV placement, lab draws, or his CT. The decision was made to obtain a non-contrast CT of his brain, which revealed no acute intracranial abnormalities. Because of the patient's presentation and clinical course, the decision was made to admit him to the observation unit and obtain a neurology consult for possible TGA secondary to stress and pain induced by the patient's nephrolithiasis. The neurologist consulted obtained a thorough history and examined the patient. He underwent MRI and magnetic resonance angiography (MRA) of the brain, electroencephalography (EEG), carotid ultrasound, and echocardiogram, as well as further lab testing that included a thyroid stimulating hormone, vitamin B-12 levels, folic acid level, and rapid plasma reagin testing. All additional testing was normal. The patient's symptoms resolved within the same day of admission (within 24 hours). He was discharged with a diagnosis of TGA.

CPC-EM Capsule

What do we already know about this clinical entity?
Transient Global Amnesia (TGA) is characterized by transient anterograde amnesia with repetitive questioning, no focal deficits, and preservation of procedural memory, cognition, and identity.

What makes this presentation of disease reportable?
This is the third case-report of nephrolithiasis-related amnesia in the literature, where a painful experience induced sympathetic activation and precipitated TGA.

What is the major learning point?
TGA is commonly misdiagnosed, with diagnosis being primarily clinical and can be made if diagnostic criteria as described by Hodges and Warlow are fulfilled.

How might this improve emergency medicine practice?
An understanding of the precipitating factors, and diagnostic criteria can focus the investigation and differential diagnosis to more accurately diagnose TGA.

DISCUSSION

TGA, with its abrupt development of prolific symptoms of dense anterograde amnesia, "remains one of the most enigmatic syndromes in neurology."[1] Although most literature on TGA comes from the neurology realm, it is a syndrome that is encountered by the full range of medical specialties, particularly emergency medicine. Although uncommon, it is paramount to be able to distinguish TGA from other life-threatening clinical entities because while the prognosis of TGA is generally benign, other similar disease states carry potential for life-threatening sequelae.[9] The diagnosis of TGA is primarily clinical and can be made if diagnostic criteria, as described by Hodges and Warlow and adapted from Caplan, are fulfilled (Figure). These include the presence of an anterograde amnesia that is "witnessed by an observer, no clouding of consciousness or loss of personal identity, cognitive impairment limited to amnesia, no focal neurological or epileptic signs, no

recent history of head trauma or seizures, and resolution of symptoms within 24 hours."[8] The hallmark of TGA is anterograde amnesia with freshly acquired memories at greatest risk, while long-term memories, self-awareness, and procedural memory, "as well as an awareness of what one should know, are typically preserved."[10] Repetitive questioning is frequently reported during an episode of TGA, likely due to an inability to acquire new information coupled with retrograde memory loss, with one case series showing 90% of patients experiencing repetitive questioning.[11] Patients suffering from TGA will return to baseline within a few hours, except for a "dense, residual amnestic gap for events that occurred during the TGA attack."[12] Helpful in making the diagnosis of TGA is the occurrence of a stressful precipitating event. "Up to 90% of TGA episodes have an identifiable physical or psychological precipitating factor."[13]

The differential diagnosis of TGA includes transient ischemic attack (TIA), subarachnoid hemorrhage, complex partial seizures, transient epileptic amnesia, psychogenic amnesia, drug-related states, metabolic derangements, CNS infections, and toxic ingestion. Misdiagnoses often occur in patients with TGA, as patients are often labeled without adherence to the criteria with diagnoses such as CVA, migraine headaches, and epileptic discharges. In the case presented, the episode was witnessed with noted anterograde amnesia and a clear distinct precipitating event. Additionally, the patient maintained his consciousness during the episode, with a normal neurological examination, and had resolution of symptoms within 24 hours. The patient underwent further testing with MRI, MRA, and EEG, but it should be noted that these studies are often normal in TGA and cannot be used to reliably diagnose the condition.

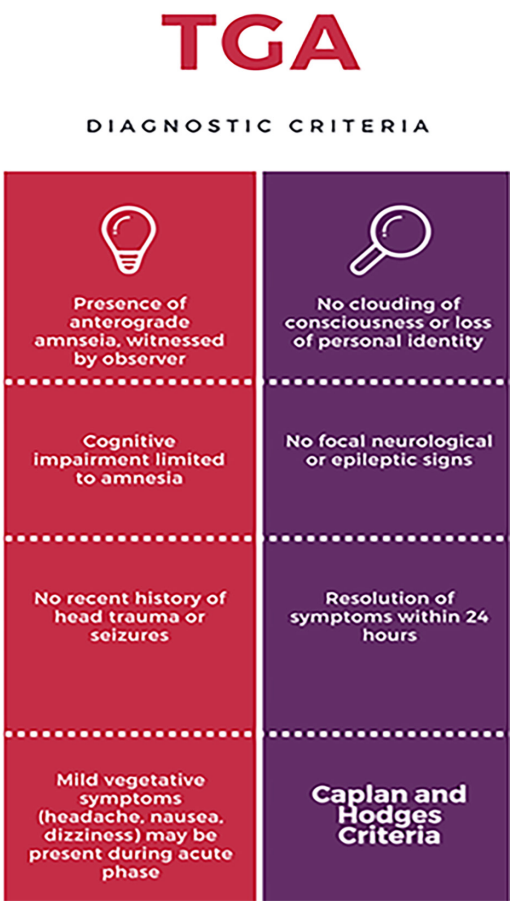

Figure. Diagnostic criteria for transient global amnesia.[3]

Studies that have examined the different precipitating factors contributing to the development of TGA have classically grouped these events (in order of prevalence) as physical effort, emotional stress, temperature changes, other factors, post-coital, and lastly acute pain. Nephrolithiasis as an inciting cause of TGA, especially in an individual with prior episodes of renal colic, has only been described twice previously and should reinforce the varied presentation by which TGA may manifest.[13] A search of MEDLINE, PubMed, and Scopus revealed only two cases of TGA attributed to nephrolithiasis.[14,15] Our case is the third such presentation of TGA attributed to nephrolithiasis, where a severe, painful experience induced sympathetic activation and precipitated TGA.[14] As the prognosis of TGA is extremely favorable, an important aspect of management after diagnosis is meeting the psychological needs of the patient and his or her family.

CONCLUSION

In summary, the patient's episode of TGA was precipitated by the acute stress induced by his nephrolithiasis. This article is the third case report of nephrolithiasis-related amnesia in the medical literature. This case should serve to reinforce the unique presentation of TGA and the different factors that may precipitate an event, as well as the differential diagnosis that the emergency physician must consider to arrive at the diagnosis. TGA is commonly misdiagnosed, without adherence to diagnostic criteria, as TIA, complex migraine, and seizures. These misdiagnoses can result in unnecessary in-hospital workup, lifelong stigmata and changes in individual's daily functioning, along with unwarranted, subsequent follow-ups and costly medical care, as there is no increased risk of TIA or increased mortality for patients with TGA.

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 Epiglottitis with Concurrent Pneumonia and Septic Shock in an Alcoholic Adult Patient

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Historically epiglottitis has been considered a childhood disease. However, the introduction of the *Haemophilus influenzae* type B vaccine has decreased the incidence of epiglottitis in children. It is important to recognize modern epiglottitis as a disease of adults. This report describes a case of acute bacterial epiglottitis in an adult patient secondary to infection caused by *Streptococcus pyogenes*, a group A streptococcal infection. This case demonstrates the importance of early recognition of epiglottitis in adults, as they can experience rapid clinical decline. The progression of this disease can lead to abrupt airway obstruction necessitating emergent airway management. [Clin Pract Cases Emerg Med. 2018;2(4):338–340.]

INTRODUCTION

Epiglottitis is a rare and potentially life-threatening disease that leads to edema and inflammation of the supraglottic tissues, which may include the epiglottis, aryepiglottic folds, arytenoids, and adjacent tissue.[1] After the introduction of the *Haemophilus influenzae* type B vaccine in 1988, the incidence of epiglottitis in children decreased.[1,3] In 2006, there were 1.6 cases of epiglottitis per 100,000 adult patients (≥ 20 years old) and 0.5 cases per 100,000 pediatric patients (< 20 years old).[1] Epiglottitis is traditionally thought to be caused by an infectious etiology, but it is important to consider other potential etiologies such as thermal insult, caustic insult, and trauma from foreign body ingestion.[1]

The emergency physician (EP) must have a high clinical index of suspicion for epiglottitis, as securing the airway early in the course of disease can prevent devastating complications.[1] The surface epithelium of the epiglottis is highly vascularized and contains many lymphatic vessels making the area prone to rapid spread of infection and resultant inflammation.[1] We present here a novel case of acute bacterial epiglottitis caused by a less-frequent organism and complicated by concurrent pneumonia in an alcoholic adult patient. The case also outlines the patient's rapid clinical decline requiring establishment of an emergent airway in the emergency department (ED).

CASE REPORT

A 58-year-old female smoker with a history of alcohol abuse presented to the ED at a community hospital with a chief complaint of shortness of breath, sore throat, and fever. She reported progressively worsening sore throat and odynophagia over the preceding two days as well as voice change and shortness of breath on the day of presentation to the ED. The patient admitted to fever, chills, diaphoresis, cough productive of phlegm, pain in the sides of her neck, and nausea. She denied sick contacts or recent travel.

On arrival to the ED, the patient's vital signs were temperature 38.3° Celsius; pulse rate 130 beats per minute; blood pressure 118/72 millimeters of mercury; respiratory rate 20 breaths per minute; and pulse oximetry 99% on room air. The patient was acutely ill appearing with diaphoresis. She was able to phonate in complete sentences. She had full, active range of motion of her neck. On examination of the posterior oropharynx, there was no appreciable edema or peritonsillar abscess noted. She did have a small amount of frothy yellow phlegm in her posterior oropharynx and appeared unable to swallow these secretions. Auscultation of her lungs demonstrated diffuse rhonchi and slight expiratory wheeze.

Sepsis was suspected on arrival, and the ED sepsis protocol was initiated with a suspected source of community-acquired pneumonia. The sepsis protocol included

administration of intravenous (IV) fluids at 30 milliliters (mL) per kilogram of body weight and broad-spectrum antibiotics. The patient was given two grams of IV ceftriaxone and 500 milligrams (mg) of IV azithromycin as well as 10 mg of IV dexamethasone. We ordered an albuterol and ipratropium nebulizer treatment as the patient was suspected to have undiagnosed, underlying chronic obstructive pulmonary disease given her smoking status and lung examination.

One hour after initial ED evaluation, the patient was receiving the nebulized breathing treatment when she was found to be sitting up on the side of her stretcher in acute respiratory distress with an oxygen saturation in the seventies. She was anxious appearing and unable to phonate. The patient was immediately transferred to a nearby resuscitation bay. She was placed on 15 L of oxygen by nasal cannula as well as on a non-rebreather mask. Materials for an emergent surgical airway were readied at bedside.

Staff included two nurses, a respiratory therapist, an emergency medicine (EM) attending physician, and two EM resident physicians. Rapid sequence intubation was initially attempted by the EM resident using video laryngoscopy. Upon visualization, we noted significant edema and purulence involving the epiglottis, arytenoids, and adjacent soft tissues causing severe deformity of normal anatomy, which obscured visualization of the glottis. There was pooling of secretions in the supraglottic region as well. The supraglottic tissues were bulbous and friable with diffuse exudates and active bleeding. While initial intubation attempts using the video laryngoscope and a bougie were unsuccessful, the attending physician was able to successfully secure the airway with an endotracheal tube placed with over-the-bougie technique. Fortunately, we were able to achieve adequate ventilation via bag-valve-mask between direct laryngoscopy attempts preventing need for a surgical airway.

Upon review of the laboratory results, the patient was noted to have a white blood cell count of 18,300 cells per microliter and an initial point of care lactic acid of 5.51 millimoles per liter (of note, value was obtained prior to treatment with albuterol). Group A streptococcal testing of the oropharynx was positive. Her troponin I level was elevated at 0.068 nanograms per mL and felt to be secondary to sepsis. Computed tomography (CT) of the chest with IV contrast demonstrated bilateral patchy consolidative changes in the lower lobes consistent with pneumonia, a thickened distal esophagus with retained fluid, multiple wedge-shaped hypodensities in the kidneys consistent with renal infarcts, and hepatic steatosis. A CT of the neck with IV contrast demonstrated enlargement of the tonsils with adjacent edema and narrowing of the airway without definite fluid collection. Following intubation the patient became hypotensive, necessitating placement of a central venous catheter and the use of norepinephrine for continued management of septic shock. The patient was admitted to the intensive care unit.

CPC-EM Capsule

What do we already know about this clinical entity?
Acute epiglottitis is a rare but serious condition that can result in life-threatening airway obstruction.

What makes this presentation of disease reportable?
This is the first presentation reported in the literature of acute epiglottitis in an adult with concurrent pneumonia caused by Streptococcus pyogenes.

What is the major learning point?
We highlight the importance of early assessment and planning in patients with potential threats to airway patency.

How might this improve emergency medicine practice?
This case demonstrates the importance of having an increased awareness of this disease, especially among adult patients, as the consequences of unrecognized acute epiglottitis can be fatal.

The patient was extubated the following day, and she was transferred to another facility two days following admission for otolaryngology evaluation. Upon arrival to the second hospital, the patient was evaluated by an otolaryngologist who performed flexible laryngoscopy, which demonstrated epiglottitis with a mottled epiglottis and fibrinous exudate that appeared to be consistent with early resolution of epiglottitis. The patient was continued on IV steroids as well as IV broad-spectrum antibiotics. She continued to require oxygen supplementation to maintain an oxygen saturation above 88%. Respiratory cultures and blood cultures were positive for *Streptococcus pyogenes*. The patient was discharged from the hospital 15 days following her initial presentation to the ED, the completion of the course of IV antibiotics and the resolution of hypoxia.

DISCUSSION

Clinical suspicion and early recognition of acute epiglottitis is critical in the ED as this disease can progress rapidly and lead to sudden airway obstruction.[1] Because direct visualization of the epiglottis is not always possible on initial presentation, it is important to recognize clinical features

of epiglottitis, which may include sore throat, fever, stridor, drooling, dysphagia, odynophagia, pooling of secretions in the oropharynx, "tripoding," anxiety, respiratory distress, and muffled voice.[1] In addition to recognizing the clinical features of epiglottitis, EPs should be aware of certain risk factors that increase an adult patient's risk of developing epiglottitis. Notable risk factors for epiglottitis in adult patients include hypertension, diabetes mellitus, substance abuse, and immune deficiency.[1] Some factors associated with an increase in severity of epiglottitis include body mass index greater than 25, diabetes mellitus, concurrent pneumonia, and the presence of an epiglottic cyst at the time of admission.[4]

While the focus of this case report was to highlight the importance of early recognition of epiglottitis, it is important to note that certain studies and interventions should be initiated promptly. One may consider ordering a lateral neck radiograph if the patient appears stable.[1] Width of the epiglottis greater than eight millimeters and aryepiglottic fold width greater than seven millimeters measured on lateral neck radiograph appears be diagnostic of acute epiglottitis in an adult patient.[5] However, diagnosis is typically confirmed by direct visualization of the epiglottis.[1] Broad-spectrum antibiotics and IV hydration are crucial measures in the treatment of this disease.[6] The first-line recommended antibiotic regimen for acute epiglottitis is cefotaxime 50 mg per kilogram body weight intravenously every eight hours plus vancomycin 15 mg per kilogram body weight intravenously every 12 hours.[6] The use of steroids may be considered to help decrease inflammation and edema of the airway.[6]

Upon review of this case, the patient's alcohol abuse was her most significant risk factor for epiglottitis, and it is important to be cognizant of the immunosuppression caused by this manner of substance abuse.[7] Chronic alcohol use can accelerate inflammatory responses and increase the susceptibility of patients to viral and bacterial infections as well as to sterile inflammation.[7] This patient also had multiple factors that increased her likelihood of a severe episode of epiglottitis including a body mass index of 26.1 and concurrent pneumonia.

CONCLUSION

This case demonstrates the importance of early identification of clinical signs and symptoms as well as risk factors for epiglottitis. Adult patients with epiglottitis can rapidly develop airway compromise. By early identification of epiglottitis, the EP may be able to adequately prepare, manage, and prevent progression to surgical airway. Additionally, this case underscores the importance of evaluating high-risk patients for other sources of infection including pneumonia as these may be contributory and require additional treatment measures. The patient presented here ultimately achieved a good outcome and no surgical airway intervention was required. However, this case could have easily had a negative or adverse outcome, which is why we emphasize the importance of maintaining a high clinical index of suspicion for epiglottitis based on appropriate clinical presentation. Frequent reassessments, avoidance of anchoring bias, and early recognition of airway compromise are crucial.

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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Under-recognized Etiology of Altered Mental Status in a Patient with Alcoholism

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Wernicke's encephalopathy is an important condition for the emergency physician (EP) to consider in patients at risk for malnutrition. A 60-year-old man with history of alcoholism presented with word-finding difficulties, dysmetria, ataxia, and personality changes. After treatment with high-dose thiamine, his neurological status returned to his baseline. Although EPs routinely prescribe thiamine for patients with alcoholism, the common initial dose of 100 mg per day is likely subtherapeutic, and the population of patients at risk for malnutrition is much broader than only those with alcoholism, and includes those with cancer, anorexia nervosa, hyperemesis gravidarum, and others. EPs must be aware of this low-cost, readily available prophylaxis to prevent long-term neurological morbidity. [Clin Pract Cases Emerg Med. 2018;2(4):341-343.]

INTRODUCTION

We report a case of Wernicke's encephalopathy that presented with ataxia and confusion but without ocular involvement. This departure from the "classic" presentation of ataxia, confusion, and opthalmoplegia is common and likely under-recognized.[2] We describe this patient's seemingly unusual presentation and his subsequent improvement with high-dose thiamine. We also discuss evidence that this disorder is common not only in those with alcoholism but in other populations at risk for malnutrition.[2] Prescription of high-dose thiamine to such patients represents an opportunity for emergency physicians (EP) to pursue a low-cost, highly effective public health intervention that prevents long-term neurological disability.

CASE REPORT

A 60-year-old man with known history of alcoholism and mild vascular dementia presented to the emergency department (ED) due to a change in mental status. The patient's wife reported that for three weeks the patient had subtle personality changes, word-finding difficulties, gait worsening from prior independent ambulation to requiring a walker. Furthermore, the patient had difficulty standing up from the toilet earlier that same day, falling back onto the toilet seat. He did not hit his head or lose consciousness. The patient denied any focal weakness or numbness. He'd had no fevers or chills, headache, change in vision, respiratory symptoms, or urinary symptoms. The patient did endorse drinking daily; he was unable to quantify his alcohol intake, but estimated he had between 5-10 drinks daily. He did drink the day of presentation to the ED.

On physical exam, vitals were stable. The patient was a talkative, obese man with word-finding difficulties. His cranial nerves were normal; in particular, there was no abnormality in ocular movements and no nystagmus. There was no focal weakness or numbness. Finger-nose-finger testing revealed symmetric bilateral dysmetria. The patient declined evaluation of his postural stability and gait. Laboratory evaluation was largely unremarkable, and head computed tomography demonstrated chronic ischemic changes without mass lesion or bleed (Image).

In the ED, the patient received 500 mg of intravenous (IV) thiamine. He was admitted for altered mental status concerning for Wernicke's encephalopathy. He received 100 mg of IV thiamine daily, and by day three his mentation improved and his dysmetria resolved. He continued to require a walker to ambulate. The patient resolved to quit drinking. He was discharged from the hospital on a regimen of 100 mg of oral thiamine daily.

The patient followed up with a neurologist approximately one month after hospital discharge. At that visit, the neurologist

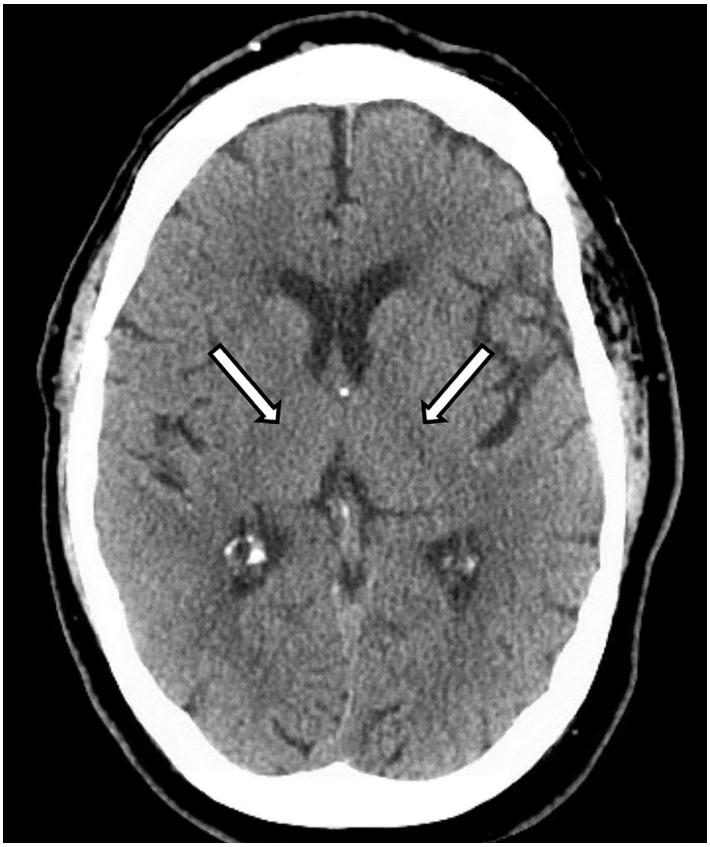

Image. Computed tomography (CT) brain of patient with Wernicke encephalopathy (WE). The utility of CT in WE is primary to rule out alternative diagnoses. Here, there is periventricular hypoattenuation of the white matter (arrows) without evidence of any other alternative diagnoses. This is nonspecific but consistent with chronic, small-vessel ischemic disease, which is consistent with his baseline mild vascular dementia but is insufficient as an explanation for his more acute neurological deficits. Magnetic resonance imaging would demonstrate findings specific for WE. Notably, this altered patient had difficulty remaining still for the examination, giving rise to significant motion artifact.

CPC-EM Capsule

What do we already know about this clinical entity?
Wernicke's encephalopathy, classically characterized by confusion, ataxia, and ophthalmoplegia, is a complication of malnutrition and is treated with thiamine.

What makes this presentation of disease reportable?
Here is reported a case of Wernicke's presenting only with confusion and ataxia, without ocular involvement.

What is the major learning point?
Wernicke's must be suspected in any patient at risk of malnutrition, even without the classic triad, and must be treated with high-dose thiamine.

How might this improve emergency medicine practice?
Emergency physicians must recognize patients at risk for malnutrition and administer higher-than-usual doses of thiamine to prevent permanent neurologic disability.

noted that the patient's memory and cognitive function had improved. He had mild gait instability and occasional falls, but this had improved as well. Neurologic exam was otherwise normal, including normal finger-nose-finger, heel-to-shin, and rapid repetitive and rapid alternating movements. The neurologist agreed with the clinical diagnosis of Wernicke's encephalopathy, but the patient refused to undergo magnetic resonance imaging (MRI) due to his claustrophobia.

DISCUSSION

Wernicke's encephalopathy is a neurologic disorder caused by lack of thiamine (vitamin B1) and most often affects patients with chronic alcoholism, although it also disproportionately affects patients with acquired immunodeficiency syndrome, hyperemesis gravidarum, malignancies, post-bone marrow transplants, post-bariatric surgery, as well as patients with eating disorders and others.[1,2] If untreated, the disease may progress to Korsakoff's syndrome, which is characterized by chronic, largely irreversible, primarily anterograde amnesia.[3] Treatment with thiamine can prevent progression to Korsakoff's syndrome. Although some patients may demonstrate radiographic evidence of the disease, most notably contrast enhancement of the mammillary bodies and thalami on MRI, these features are poorly sensitive.[2,4,5]

Although red blood cell transketolase levels can be low in thiamine deficiency and serum thiamine levels can be measured, laboratory studies are seldom helpful in practice. Therefore, diagnosis is made clinically. The classic triad includes opthalmoplegia, ataxia, and confusion. However, few patients clearly demonstrate all three signs (fewer than 16% by one report) and this schema does not depict the true breadth of symptomatology possible in patients with Wernicke's, which more broadly includes any eye movement abnormality,

any change in gait, or any change in mental status.[2] Wernicke's encephalopathy is likely under-recognized and undertreated: the prevalence of Wernicke-Korsakoff syndrome (WKS) in alcoholics is as high as 12.5%, and autopsies have demonstrated that over 80% of true cases are not diagnosed during life.[2] Furthermore, the appropriate dosing strategy for thiamine is not well-established. The frequently encountered dose of 100 mg IV daily is common for historical reasons and is not evidence-based. A 2013 Cochrane Review concluded that "evidence […] is insufficient to guide clinicians in determining dose, frequency, route or duration of thiamine treatment for prophylaxis against or treatment of WKS."[6] Thiamine is largely benign and well tolerated, and inadequate treatment is associated with significant, permanent neurocognitive disability. Based on this, one study published in *Annals of Emergency Medicine* recommends, "For patients for whom there is low suspicion for disease or for those simply requiring prophylaxis, a minimum of 100 mg should be administered intravenously. For those with confirmed or highly suspected disease and those who have 'failed' the 100-mg regimen, we recommend a dosage upwards of 500 mg intravenously."[4] In summary, the differential diagnosis of altered mental status in a patient with alcoholism must always include Wernicke's encephalopathy, even without ocular findings or objective malnutrition, and one should have a low threshold to treat with high-dose thiamine to prevent progression to permanent neurocognitive disability.

CONCLUSION

The differential diagnosis of altered mental status in a patient with alcoholism is broad and varied, but patients with Wernicke's encephalopathy secondary to thiamine deficiency are likely under-recognized and undertreated, which leads to significant, permanent, avoidable neurocognitive disability. EPs should maintain a high index of suspicion for Wernicke's in any patient with alcoholism or any other disorder predisposing to malnutrition who presents with altered mental status, even without the classic signs, and have a low threshold to treat with higher-than-usual doses of thiamine.

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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Convulsion and Atrial Fibrillation after Transforaminal Cervical Epidural Lidocaine Injection

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Lidocaine has been widely used as a local anesthetic as well as an antiarrhythmic. Its use in epidural anesthesia is increasing, which has introduced new risk and potential for harm not associated with older indications. We present a case of convulsion and atrial fibrillation seen after transforaminal cervical epidural injection with two milliliters of 2% lidocaine (40 milligrams) that resolved with no long-term sequelae. Patient had a negative serum lidocaine level. With cervical epidural injections being a common treatment for radicular pain, it is important for medical providers to be aware of the various complications associated with this procedure. [Clin Pract Cases Emerg Med. 2018;2(4):344–347.]

INTRODUCTION

Lidocaine quickly grew in popularity among local anesthetics when it came to market in 1948 due to its quick onset of action and safer side-effect profile.[1] Over time, new uses for the drug have come to light. Lidocaine is well known for treatment of ventricular dysrhythmias and has become a popular agent for use in epidural anesthesia. New indications and uses introduce new risk and potential for harm. As a Class 1B antiarrhythmic, lidocaine blocks voltage-gated sodium channels, which inhibits both initiation and conduction of nerve impulses by decreasing neuronal membrane permeability to sodium ions.[1] Adverse effects typically result with systemic toxicity in a dose-related response and primarily affect the central nervous system (CNS) and the heart, ranging from mild neurologic symptoms to seizures and, in severe cases, cardiac arrest.[2-4]

Serum lidocaine levels can be measured to monitor for toxicity in patients undergoing continuous infusion or in cases where toxicity is suspected. However, these laboratory tests are not readily available and are not widely used or particularly clinically helpful. Over the past several decades, cervical epidural injections with both local anesthetics and corticosteroids have become a mainstay of treatment for radicular neck and back pain.[5,6] Complications of these procedures are not common but can be devastating when they occur.[5-7] We present a case of CNS and cardiotoxicity effects seen during cervical epidural injection. With cervical epidural injections being a common treatment modality for radicular neck pain, it is important for medical providers to be aware of the potential complications associated with this procedure and their presentations.

CASE REPORT

A 51-year-old male weighing 131 kilograms (kg) presented to the emergency department (ED) via ambulance with altered mental status and slurred speech after undergoing cervical epidural injection with two milliliters (mL) of 2% lidocaine (40 milligrams [mg]) under fluoroscopic guidance in an ambulatory setting. He became unresponsive during the injection with subsequent brief convulsive activity for which he was given 2 mg of midazolam. Emergency medical services was called and found him obtunded with shallow breathing and low oxygen saturations requiring ventilation assistance. On arrival to the ED his breathing was spontaneous and erratic with low oxygen saturations. He remained somnolent with slurred speech, unable to answer questions appropriately or follow commands. Preparations were made for intubation given altered mental status and low oxygen saturations; however, oxygen saturations and

mental status improved within the first 10 minutes of arrival and ultimately intubation was not required.

On cardiopulmonary monitor he was noted to have an irregularly irregular heart rhythm. Electrocardiogram showed atrial fibrillation with a rate of 82 beats per minute. Hemodynamically he was stable. He converted to normal sinus rhythm 20 minutes later. He reported no history of atrial fibrillation. Within 60 minutes of ED arrival the patient's mental status was back to baseline without recollection of the events that had occurred after the start of the procedure. He only had chest wall pain, possibly from sternal rub or from any bystander chest compressions that may have been performed when he became unresponsive. Imaging studies obtained included the following: chest radiograph, computed tomography (CT) of the head, CT angiogram of the head and neck, and CT of the chest with intravenous contrast. No pertinent imaging abnormalities were identified. Serum/plasma levels of lidocaine and its primary active metabolite, monoethylglycinexylidide (MEGX) were obtained 15 minutes after patient arrival. Both levels returned undetectable. The patient was observed overnight in the hospital and remained asymptomatic and without any further dysrhythmia. He was discharged home the following day on aspirin 325 mg daily and with a referral to outpatient cardiology.

CPC-EM Capsule

What do we already know about this clinical entity?
Lidocaine toxicity can occur with excessive dosage or inadvertent intravascular injection. Common signs and symptoms involve the central nervous system and cardiovascular system.

What makes this presentation of disease reportable?
Our patient received a small dose of lidocaine, not expected to cause toxicity, and exhibited classic symptoms of toxicity and atrial fibrillation, which has not previously been described.

What is the major learning point?
Even small doses of lidocaine, which are considered therapeutic, may cause toxicity if inadvertently injected into an intravascular space.

How might this improve emergency medicine practice?
Lidocaine toxicity is a rare complication of a common procedure, epidural injection. It is important to be aware of this complication and the presentation of lidocaine toxicity.

DISCUSSION

Symptoms of lidocaine toxicity typically occur after excessive dosage or inadvertent intravascular injection, with the latter being more common in the case of local anesthetic toxicity.[4] Adverse effects of lidocaine toxicity include perioral numbness, visual and auditory disturbances, drowsiness, slurred speech, twitching, loss of consciousness, tonic-clonic seizures, respiratory depression and cardiac arrest.[2-4] The severity of CNS symptoms generally correlate with serum lidocaine level, with the risk of seizure and respiratory depression increasing as dosage and serum lidocaine level increase.[4,8] A serum lidocaine level of greater than five micrograms (mcg)/mL is considered to be representative of toxicity resulting in CNS manifestations.[3] Despite this relationship, toxicity has also been demonstrated in animal models at smaller dosages, attributed to retrograde passage of local anesthetic under pressure (reverse intracarotid flow), transarterial diffusion during stellate ganglion block, and inadvertent injection into arterial vasculature during dental procedures.[4,9,10] This has not been demonstrated in human subjects; however, we propose a similar mechanism as an explanation in this case by which toxicity occurred at a smaller-than-expected dosage in our patient via inadvertent intravascular injection in close proximity to the CNS.

Our patient exhibited typical CNS symptoms of lidocaine toxicity during and directly after attempted cervical epidural injection with 2 mL of 2% lidocaine including tonic-clonic seizure activity, respiratory depression, drowsiness and slurred speech. His lidocaine and MEGX levels returned undetectable, as we would expect after administration of 40 mg of lidocaine in a 131-kg male. There are no reports in the literature regarding the minimum dosage of lidocaine required to obtain a detectable serum level. However, the patient's serum lidocaine concentration can be estimated using the dosage he received (40 mg) and the reported volume of distribution (Vd) for lidocaine in the literature. The Vd for lidocaine varies between individuals but is reported between 0.6-4.5 L/kg.[11] Using the equation, dose divided by Vd equals serum concentration (D/V = C), we were able to estimate our patient's serum lidocaine concentration at the time of injection. Assuming complete and immediate absorption, given we suspected intravascular injection, and using the most conservative Vd available (0.6 L/kg), our patient's serum lidocaine concentration would have been no more than 0.51 mcg/mL at the time of injection. The reporting limit for detection of both lidocaine and MEGX on the tests performed was 0.5 mcg/mL. Thus, it is possible that even if the levels had been obtained immediately after injection they may have returned undetectable.

The elimination half-life of both lidocaine and MEGX is approximately 30-90 minutes.[12] Our patient's lidocaine and MEGX levels were not obtained until approximately 30-

40 minutes after the time of injection, further decreasing the likelihood of obtaining detectable levels. Typically, this dosage of lidocaine would not be expected to cause toxicity. We believe his CNS levels at the time of injection were likely elevated above the threshold to cause symptoms of toxicity secondary to inadvertent intravascular injection, and his symptoms support this hypothesis. Sharma et al. described a similar case of CNS symptoms after inadvertent intravascular injection with only 1 mL of 2% lidocaine (20 mg) into an aberrant carotid artery overlying the trachea during a percutaneous dilation tracheostomy. No lidocaine or MEGX levels were obtained; however, inadvertent intravascular injection was confirmed with aspiration of blood after symptom onset during injection. Arterial pulsation on magnetic resonance angiography revealed aberrant carotid artery overlying the area of injection.[13]

In addition, our patient exhibited atrial fibrillation, which to our knowledge is a dysrhythmia that has not been previously described with lidocaine cardiotoxicity. Direct cardiotoxicity has been shown to require higher serum lidocaine levels than required to produce CNS toxicity.[4,8] Lidocaine has commonly been used to treat ventricular dysrhythmias; however, in toxicity, myocardial depression, bradycardia, dysrhythmias, atrioventricular blocks as well as acceleration of ventricular response in atrial tachydysrhythmias have been reported.[3,4,8] Lidocaine depresses automaticity; thus, atrial fibrillation seems an unlikely dysrhythmia to occur. Given the ability of lidocaine to depress the sinus node, it is possible that atrial fibrillation occurred in response to suppression of the sinus node's automaticity in the setting of acute lidocaine toxicity. It has also been proposed that the CNS may play a role in cardiotoxicity; specifically, the exposure of the medulla to local anesthetics seems to result in cardiovascular changes.[4] This appears to be most profound with bupivacaine and has not been previously associated with atrial fibrillation. Other factors that may contribute to cardiovascular changes in the setting of lidocaine toxicity include hypoxia, hypercarbia and acidosis[4,7] and our patient may have suffered mild hypoxia for a period of time. Our patient's episode of atrial fibrillation was self-limited and rate-controlled without pharmacologic intervention, correcting to normal sinus rhythm within 30 minutes.

There are two common approaches for delivery of cervical epidural injections for treatment of radicular pain: transforaminal and interlaminar.[7] Our patient underwent transforaminal cervical epidural injection via anterolateral approach.[14] All epidural injections carry risks including increased radicular pain, vasovagal reaction, intravascular injection, vascular injury, dural puncture, spinal cord injury, and spinal cord infarction.[5,7,15] While transforaminal injections are considered superior in that they deliver medication to a more-specific anatomical area, this technique harbors increased risk of inadvertent intravascular injection and injury.[5,7] Additionally, multiple studies have identified cervical epidural injections as having higher risk for inadvertent intravascular injection when compared to thoracic, lumbar, and sacral epidural injections.[7,10] We surmise that our patient's symptoms were the results of acute lidocaine toxicity from inadvertent intravascular injection while undergoing transforaminal cervical epidural injection.

CONCLUSION

Epidural injections are commonly used to treat radicular pain, and while complications are rare they can be devastating. Lidocaine is often used as a treatment medication or as a test injection prior to corticosteroid injection. Lidocaine toxicity can range from subtle to severe CNS and cardiotoxic effects, or even death. While serum lidocaine and MEGX levels can be obtained to evaluate for toxicity they are not readily available. We present a novel case of CNS and cardiotoxicity after suspected inadvertent intravascular injection of a small dose of lidocaine during cervical epidural injection with negative serum lidocaine and MEGX levels. It is important to be aware of the various complications of epidural injections, how they present, and the limitations of laboratory studies for diagnosis of lidocaine toxicity.

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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Limb Ischemia in a Patient with Hyperosmolar Hyperglycemic State

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A 61-year-old male with a recent diagnosis of pemphigus vulgaris was brought to the emergency department for altered mental status. He had recently started taking prednisone to manage his autoimmune disease and had a progressive decline in his mental status along with decreased oral intake. Evaluation revealed hyperosmolar hyperglycemic state (HHS) and occlusive arterial thrombosis, a rare but known complication of HHS. He was resuscitated aggressively with intravenous fluids, insulin, and heparin and admitted to the intensive care unit. Emergency physicians should remain vigilant for ischemic complications in patients with HHS. Early recognition and treatment can reduce the morbidity and mortality associated with this endocrine emergency. [Clin Pract Cases Emerg Med. 2018;2(4):348–352.]

INTRODUCTION

Hyperosmolar hyperglycemic state (HHS) is defined as severe hyperglycemia, a marked increase in serum osmolarity with clinical signs of severe dehydration in the absence of ketoacids.[1] The hyperosmolar state is a serious hyperglycemic emergency that affects patients with type 2 diabetes mellitus and is associated with significant morbidity and mortality.[2] About 1% of emergency department (ED) patients with diabetes-related conditions have HHS.[3,4] The mortality rate associated with HHS is five times higher than that associated with diabetic ketoacidosis (DKA).[5] The prognosis for patients with HHS is significantly worse at the extreme of ages and in the presence of hypoperfusion, especially coma.[6]

CASE REPORT

A 61-year-old man was transported to the ED by ambulance with a complaint of confusion. His past medical history was significant for recently diagnosed pemphigus vulgaris that was being treated with steroids. His family described gradual functional decline over the prior two weeks with no changes in his skin lesion. On the day of ED presentation, he was no longer responding verbally to his family members. He had no history of diabetes mellitus or peripheral vascular disease.

On assessment in the ED, he was found to have a waxing and waning level of consciousness and was alert to self only. His initial vital signs were heart rate of 134 beats per minute, blood pressure of 141/86 millimeters of mercury, respiration of 16 breaths per minute, temperature of 37.1°C (98.8°F), and oxygen saturation of 95% on room air. He was ill-appearing with dry mucous membranes and poor skin turgor. He was also found to have a cold, pulseless, right lower extremity. Laboratory evaluation revealed marked hyperclycemia (blood glucose, 1077 milligrams per deciliter [mg/dL]), hemoconcentration (hemoglobin, 20 grams/dL), sodium concentration of 172 millimoles/L, anion gap of 23, and acute kidney injury (creatinine, 2.47 mg/dL). Urinalysis revealed a urinary tract infection without ketones. His thyroid-stimulating hormone level and a noncontrast computed tomography of his head were unremarkable. Duplex ultrasound of the lower extremity demonstrated total occlusion of the right proximal common iliac and popliteal arteries.

Based on these results, we diagnosed HHS with limb ischemia and sepsis from urinary tract infection. The patient was treated with insulin, heparin, and broad-spectrum antibiotics in the ED. His free water deficit was calculated to be greater than 10 L. Fluid management included initial volume resuscitation

with 3 L of Plasma-Lyte. He was then transitioned to slow sodium correction with normal saline over 24 hours. He was admitted to an intensive care unit (ICU) with vascular surgery consultation for management of the limb ischemia.

The patient underwent through-the-knee amputation due to irreversible tissue damage on hospital day three. During his ICU stay, his blood glucose and sodium levels were corrected gradually and the acute kidney injury resolved. The patient did well during hospitalization and was discharged to an inpatient rehabilitation facility on hospital day 11.

CPC-EM Capsule

What do we already know about this clinical entity?
Hyperosmolar hyperglycemic state (HHS) is a clinical syndrome characterized by a state of profound dehydration, metabolic derangements, and multiorgan system failure.

What makes this presentation of disease reportable?
This case highlights a prothrombotic state accompanying a state of profound dehydration in a patient with HHS.

What is the major learning point?
Thromboembolic events should be considered in patients with HHS.

How might this improve emergency medicine practice?
Early recognition of complications of HHS allows early involvement of specialized services, which can improve outcomes in this population.

DISCUSSION

The pathogenesis of HHS is not fully understood, but the syndrome is attributed to decreased insulin sensitivity coupled with increases in the counter-regulatory hormones—cortisol, catecholamines, growth hormone, and glucagon—all of which increase hepatic and renal glucose production.[6] The hallmark of HHS is hyperglycemia leading to hyperosmolarity, which is secondary to dehydration from glucosuria and body fluid depletion caused by other precipitating factors. The level of hyperosmolarity best correlates with mental status changes in patients with HHS.[7]

In more than 50% of HHS cases, the precipitating factor is infection (e.g., pneumonia, urinary tract infection, or sepsis).[8] Other precipitants are inadequate glucose control, surgery, cerebrovascular ischemia, and myocardial ischemia.[8] Dehydration is more subtle, occurring slowly over the course of a few days. It is exacerbated by decreased oral intake, especially in the geriatric population.[9] The use of corticosteroids has been linked to the development of hyperglycemia. Patients on high-dose steroids are particularly prone to HHS.[10] The hyperosmolar state of HHS induces osmotic diuresis, leading to severe dehydration and fluid loss, which can be as high as 7-12 L, representing a 10-15% weight loss.[3] The triad of hyperglycemia, hyperosmolar state, and dehydration leads to a catecholamine surge, with increased production of cortisol and glucagon, which worsens hyperglycemia and the hyperosmolar state.[1]

HHS develops over days to weeks. Its clinical manifestation starts as weakness and lethargy, progressing to obtundation and coma. These changes correlate with the plasma osmolality and usually become apparent at levels greater than 310 milliosmoles (mOsm) per kilogram.[11,12] The physical examination usually reveals signs of dehydration (dry mucous membranes, dry axilla, decreased skin turgor, low jugular venous pressure, and hypotension[6]) with or without clinical evidence of the inciting event. The hyperosmolar state is a hypertonic state irrespective of the sodium level at presentation, because of intracellular sodium shifts. Measurable serum sodium levels can vary from hyponatremia to hypernatremia.[1]

At the onset of severe dehydration, intracellular fluid shift, and hyperglycemia, the serum sodium level does not reflect the body's actual sodium level. The true sodium level is determined by adding 1.6 to the measured serum sodium value for every 100 mg/dL of glucose. If the uncorrected sodium level is normal or elevated, the patient has already lost a significant amount of fluid. The corrected sodium level reveals the true level of hypernatremia.[13,14] Patients with HHS usually present with severe hyperglycemia (glucose >600 mg/dL). Those with end-stage renal disease can have levels exceeding 1000 mg/dL. These patients will also have hypokalemia and hypophosphatemia as a result of osmotic diuresis, leading to urinary loss of potassium and phosphate. These deficiencies are also associated with decreased oral intake.[14]

The successful treatment of HHS has five cornerstones: volume correction, glycemic control, electrolyte management, addressing the underlying cause, and managing complications (Table). Fluid correction should be started immediately, as soon as the HHS diagnosis has been established. The goals of volume resuscitation are intravascular volume repletion, restoring normal plasma tonicity, and improving end-organ perfusion. The initial intravenous (IV) fluid of choice is 0.9% normal saline, administered with the goal of achieving 50% fluid resuscitation within the first 12 hours.[3,4] After the initial resuscitation period, the patient's clinical status should be reassessed so that the fluid choice can be adjusted based on plasma tonicity and electrolyte levels.[4]

Glycemic control is achieved with IV administration of insulin after fluid repletion. The recommended dose is 0.1 units per kilogram (U/kg). The goal is to decrease osmolarity to below 310 mOsm/kg and to achieve a glucose level less than 250 mg/dL. Normally, with IV fluids and tight glycemic control, HHS patients' mental status improves rapidly. Once the blood glucose concentration is less than 250 mg/dL, the fluid can be switched to a dextrose-containing solution to avoid hypoglycemia while the patient is being bridged to subcutaneous insulin.[15] Special attention should be paid to electrolytes as well. Typically, these patients present with hyponatremia secondary to hyperglycemic osmotic force that drives water into the vascular space, causing dilutional hyponatremia. In contrast, hypernatremia denotes profound dehydration. Volume repletion normally corrects the sodium disturbance.[9,16]

Additionally, the potassium level might be elevated despite a total body deficit secondary to potassium transit to the extracellular space, creating relative hyperkalemia without the acidosis typically seen with DKA. Before starting insulin therapy, it is imperative to reassess the potassium level and the need to start potassium replacement. Starting treatment with insulin may lead to hypokalemia due to the potassium shift back into the cell that accompanies volume resuscitation.[9,17,18] Additionally, the total body phosphate level can be relatively low despite a normal or high phosphate level because of molecular physiology similar to that of hypokalemia. Phosphate should be replaced if the patient is found to be hypophosphatemic, as insulin will drive phosphate, along with potassium, into the cells. Given that phosphate is needed in all muscular contractions, its uncorrected low levels can lead to cardiac arrest.[9,19,20]

It is important to look for the factors that precipitated HHS and to correct them as quickly as possible. Ischemia of any organ can precipitate HHS. Myocardial ischemia, cerebrovascular accidents, bowel ischemia, and limb ischemia can cause catecholamine and cortisol surges that can worsen preexisting hyperglycemia. It is imperative to identify these factors and treat them.[7-10]

HHS can affect coagulation by increasing the levels of coagulation factors, especially protein C and factor VIII complex, and decreasing fibrinolytic activities.[21] In a crossover study, Stegenga and associates showed that hyperglycemia itself promotes coagulation through activation of thrombin-antithrombin complexes. The same study demonstrated that hyperinsulinemia can increase plasminogen-activating factors that decrease fibrinolysis.[22,23] Severe dehydration can induce vasoconstriction and thromboembolic events such as cerebrovascular accidents, myocardial infarction, arterial insufficiency in the lower extremities, and mesenteric ischemia.[24-26]

Severe dehydration and the prothrombotic state provoked by HHS can lead to end-organ ischemic events. This prothrombotic state can lead to acute myocardial ischemia, cerebrovascular accidents, acute mesenteric ischemia, and lower extremity ischemia. Patients who present with altered mental status and do not improve after initial resuscitative measures warrant further workup.[21-26]

Management of limb ischemia is aimed at saving limb as well as life, restoring blood flow, and preventing further cellular damage from thrombosis or embolism. While awaiting evaluation by the specialty service, patients should be started on anticoagulation with heparin. The current recommendation is a bolus of 80-150 U/kg followed by maintenance at 18 U/kg per hour. However, if heparin is contraindicated because of documented heparin-induced thrombocytopenia or antithrombin

Table. Complications associated with HHS.[3-4,8]

Complications related to the disease process	Dehydration	Hyperviscosity Electrolyte derangement Metabolic acidosis Rhabdomyolysis Malignant hyperthermia
	Prothrombotic factor activation*	Cerebral infarcts Myocardial infarction Pulmonary embolism Acute respiratory distress syndrome Mesenteric vessel thrombosis Limb ischemia Disseminated intravascular coagulation
Complications related to therapy		Hypoglycemia Hypokalemia Cerebral edema Pulmonary edema Hyperchloremic non-anion gap acidosis

*Can be worsened by profound dehydration.

III deficiency, direct thrombin inhibitors can be given. The only exception to anticoagulation is if the patient is actively bleeding.[27] It is imperative that emergent vascular surgical consultation be obtained for consideration of emergent vs. urgent revascularization if the limb is salvageable. In irreversible ischemia, amputation might be the only definitive treatment.[28]

CONCLUSION

Emergency physicians should remain vigilant for ischemic complications in patients presenting with HHS. Early recognition and treatment can improve the morbidity and mortality rates associated with this endocrine emergency.

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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Mercury Ingestion

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CASE PRESENTATION

A 30-year-old male with history of bipolar disorder and previous incident of elemental mercury ingestion in a suicide attempt, presented to the emergency department with new-onset nausea and vomiting. Abdominal radiograph showed a collection of metallic material in the appendix (Image 1), which was confirmed by computed tomography. Blood mercury level was found to be 120 micrograms per liter (mcg/L). Repeat abdominal radiograph approximately six hours later, after the patient was placed in lateral decubitus position and Trendelenburg position, showed partial spillage of the mercury out of the appendix into the cecum (Image 2). The patient was admitted for bowel irrigation with chelation therapy. Symptoms resolved after the first day, and repeat radiographs showed gradual clearance of mercury from the colon.

DISCUSSION

This case demonstrates a successful positioning maneuver of placing the patient in lateral decubitus and Trendelenburg position, which led to significant passage of the retained mercury from the appendix. No other images in the literature demonstrate this characteristic of elemental mercury. Mercury exists in three forms: elemental, inorganic, and organic. Elemental mercury can cause pulmonary toxicity when vapor is inhaled, but it has poor gastrointestinal absorption when it is ingested and is usually excreted over several days with low risk of systemic

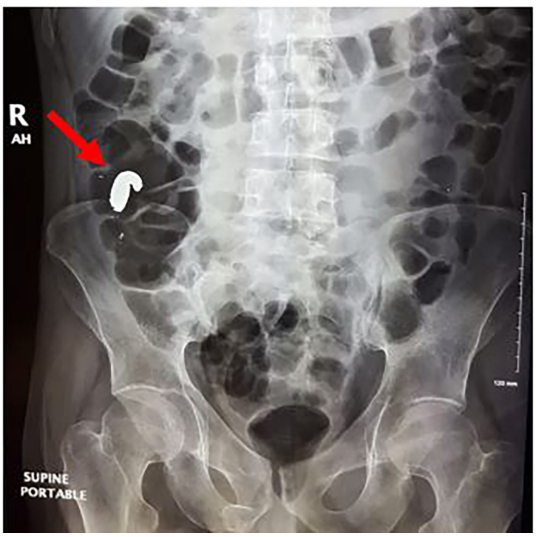

Image 1. Abdominal radiograph demonstrating high-density material in the right hemi-colonic region (red arrow), which was confirmed to be in the appendix on computed tomography.

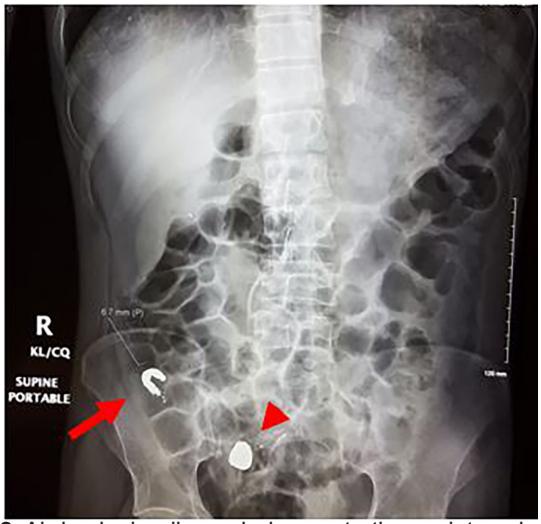

Image 2. Abdominal radiograph demonstrating an interval new collection of the mercury in the right hemi-pelvis (red arrowhead) and interval decrease in the collection of mercury in the appendiceal lumen (red arrow).

toxicity.[1,2] However, there have been several case reports of ingested mercury found to be retained in the appendix of patients, which led to the development of appendicitis.[3] Prophylactic appendectomy vs. conservative management has been described for retained mercury in the appendix.[4,5]

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?
Elemental mercury has poor gastrointestinal absorption, but it can become retained in the appendix and lead to appendicitis.

What is the major impact of the image(s)?
It demonstrates that successful patient placement in left lateral decubitus and Trendelenburg position may help dislodge retained mercury from the appendix.

How might this improve emergency medicine practice?
Conservative management with patient positioning and bowel irrigation can be used for patients with mercury retained in the appendix prior to considering surgical intervention.

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Flail Chest Resulting from a Rocket-type Firework

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CASE PRESENTATION

An 18-year-old male presented to the emergency department (ED) via ambulance after sustaining a blunt force injury to the left chest from a rocket-type firework. He received a needle thoracostomy in the prehospital setting by paramedics after he was noted to be hypotensive with absent breath sounds on the left. Initial ED vitals were temperature of 37.3°C, blood pressure 90 over palpation, heart rate 147 beats per minute, respirations 30 breaths per minute, and oxygen saturation of 89% on 15 liters of oxygen. The left anterior chest wall demonstrated a large ecchymotic burn with powder stippling and an obvious flail segment billowing paradoxically as high as seven centimeters. Subcutaneous crepitation was palpated in the soft tissues of the chest and neck. Tube thoracostomy was rapidly performed and chest imaging obtained (Images 1 and 2).

DISCUSSION

Chest radiograph revealed a left tension pneumothorax with significant subcutaneous emphysema of the left chest wall and neck. Chest computed tomography revealed additional findings of a large tear of the left pectoral muscles and defects to the underlying intercostal muscles, as well as fractures to ribs 2–4 (Image 2). The patient was taken to the operating room for surgical management.

Flail chest occurs when three or more adjacent ribs are fractured in at least two places, creating a chest wall segment that moves paradoxically from the chest wall.[1] Flail chest is a life-threatening complication of severe chest trauma with mortality rates of up to 16%.[1,2] Complications may include pneumonia (21%), acute respiratory distress syndrome (14%), and sepsis (7%).[2] In a review of flail chest injuries in the National Trauma

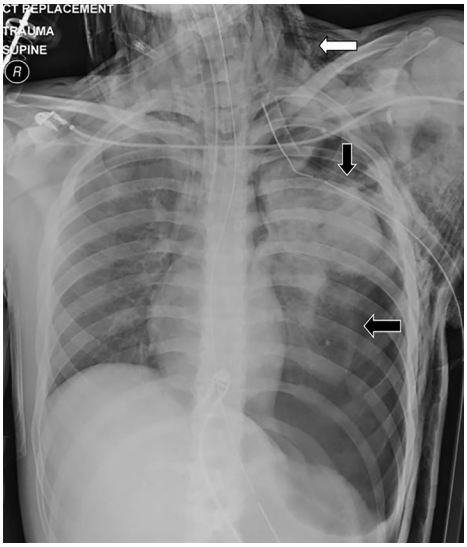

Image 1. Chest radiograph demonstrating left tension pneumothorax (horizontal black arrow) with near- complete collapse of the left lung as well as significant subcutaneous emphysema (horizontal white arrow) to the left chest wall and neck. Also noted is a chest tube to the left chest (vertical black arrow).

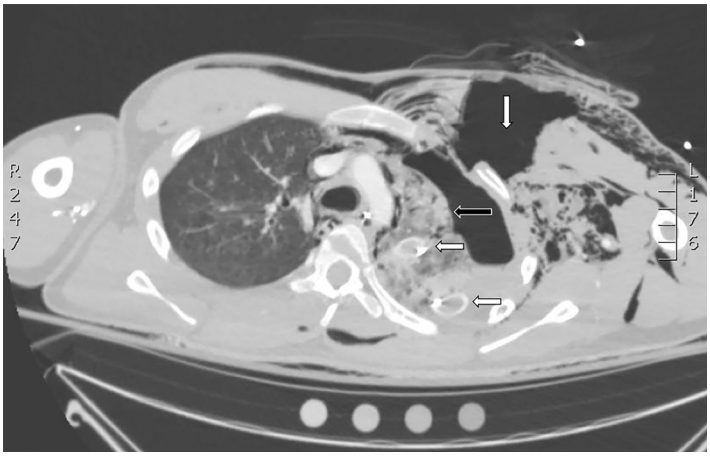

Image 2. Chest computed tomography scan at the level of the aortic arch demonstrating left-sided pneumothorax (horizontal black arrow) with high-density pleural effusion compatible with hemothorax. There is a large tear of the left pectoral muscles with distraction of the muscle tissue, and a large, air-filled, 5 cm- wide defect (vertical white arrow). Two chest tubes can be seen in the left hemithorax (horizontal white arrow). A large amount of subcutaneous emphysema tracking along the chest wall can also be appreciated.

Data Bank, we found that 59% of patients required mechanical ventilation, 82% intensive care unit (ICU) admission, 44% tube thoracostomy, and 21% required a tracheostomy. Although less than 1% of patients require operative management, it has been shown to reduce mortality, duration of mechanical ventilation, ICU and hospital length of stay.[3-5]

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?
Flail chest is a life-threatening complication of severe chest trauma with mortality rates of up to 16%.

What is the major impact of the image(s)?
The images demonstrate the significant injuries that can result from rocket-type fireworks sustained at close range.

How might this improve emergency medicine practice?
This case emphasizes the multiple associated injuries and complications to be aware of in patients sustaining close range blunt force chest trauma.

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Massive Right Breast Hematoma

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CASE PRESENTATION

A 53-year-old female with a history of hypertension, congestive heart failure, and generalized anxiety disorder taking 81 milligrams of aspirin daily presented as a trauma activation following a motor vehicle collision. She was the restrained driver of a vehicle traveling at approximately 45 miles per hour that was rear-ended by another vehicle traveling at unknown speed. Airbags were deployed. The patient was extricated by first responders.

Upon presentation to the emergency department she was complaining of severe right breast pain. She was initially tachycardic at 115 beats per minute with a blood pressure of 128/60 millimeters of mercury (mmHg). Her primary survey was intact and her secondary survey was significant for ecchymosis to her right breast, which was swollen, tense and exquisitely tender (Image 1). No further evidence of trauma was noted.

After the primary survey her right breast continued to expand and her blood pressure was noted to deteriorate to a recorded low of 99/52 mmHg despite a fluid bolus and blood transfusion. A computed tomography of the chest demonstrated a 10.5 cm x 12.7 cm x 18 cm breast hematoma (Image 2). Remarkably, there was no evidence of other concomitant injuries. Due to her consistently labile blood pressures trauma surgery elected to manage the patient operatively. A 1,500-milliliters hematoma was evacuated, consistent with the patient's state of class III shock. Origin of the bleeding was determined to be an arterial branch within the pectoralis major. The patient was taking aspirin, causing presumed platelet dysfunction, but her coagulation panel was normal.

Image 1. Photograph of patient showing massive right breast hematoma.

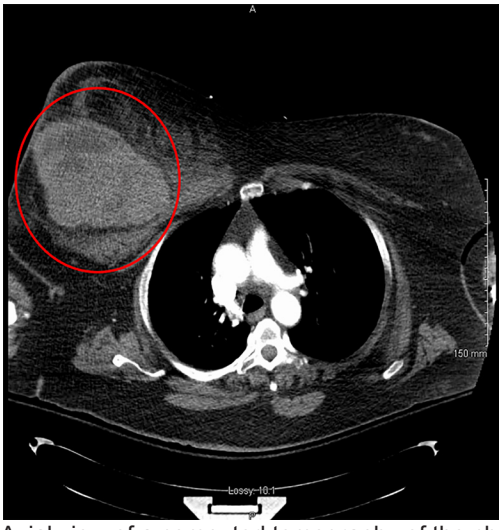

Image 2. Axial view of a computed tomography of the chest revealing large right breast hematoma (red circle).

DISCUSSION

Among cases of blunt chest trauma in females, breast hematoma is relatively uncommon, occurring in less than 2%. More than 93.5% are managed expectantly with only 6.5% requiring invasive procedures.[1] To our knowledge,

this is the only reported case of a massive breast hematoma resulting from blunt chest trauma without concomitant injuries demonstrating a state of Class III shock.[2] A similar computed tomography image has been published.[3] Nevertheless, the primary difference between this case and the case in the cited image is that the patient in the cited case had concomitant rib fractures and was treated with interventional radiology embolization, whereas the patient in this presentation was treated operatively and had no concomitant injuries. This case illustrates another compartment where hemodynamically-significant bleeding can occur in the setting of trauma.

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?
Breast hematomas are relatively uncommon, occurring in less than 2% of blunt chest trauma. More than 93.5% are managed expectantly, with only 6.5% requiring invasive procedures.

What is the major impact of the image(s)?
To our knowledge, this is the only reported case of an isolated massive breast hematoma resulting from blunt chest trauma demonstrating a state of Class III shock.

How might this improve emergency medicine practice?
It may raise suspicion among emergency providers that hemodynamic compromise may be due to an isolated breast hematoma.

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Eagle Syndrome

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CASE PRESENTATION

A 30-year-old male presented to the emergency department with intermittent neck pain, dysarthria, right facial droop, right-sided facial paresthesias and right upper extremity weakness for several days prior. Past medical history was significant for hypertension. Neurologic exam revealed a National Institutes of Health Stroke Scale (NIHSS) score of three secondary to dysarthria, right facial paralysis, and mild right upper extremity hemiparesis. Noncontrast brain computed tomography (CT) showed no evidence of hemorrhage, mass lesion, or acute infarction. CT angiography (CTA) head and neck with three-dimensional rendering demonstrated a large left styloid process and partially calcified stylohyoid ligament and large completely calcified right stylohyoid ligament consistent with Eagle syndrome (Image). The left cervical internal carotid artery also had severe focal dissection and 99% narrowing. The right cervical internal carotid artery had mild narrowing and intimal irregularity consistent with carotid dissection. Magnetic resonance imaging of the brain demonstrated scattered infarcts predominantly in a band-like pattern within the deep white matter of the left frontal lobe. The patient was admitted to the hospital after neurology consultation and started on enoxaparin. However, he decided to forego further definitive surgical management of his Eagle syndrome.

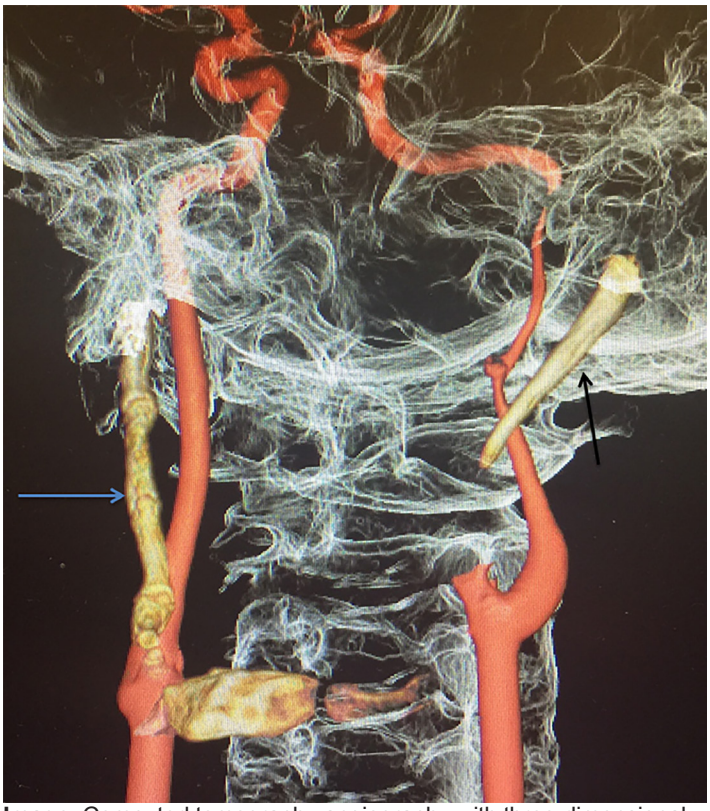

Image. Computed tomography angiography with three-dimensional rendering showing large left styloid process (black arrow) and large completely calcified right stylohyoid ligament (blue arrow).

DIAGNOSIS

Eagle syndrome is relatively uncommon with an incidence of abnormal stylohyoid length being 4% to 7.3%.[1] Classic Eagle syndrome is described as post-tonsillectomy pain, dysphagia and a foreign-body sensation, while the less-common version is related to an elongated styloid which can compress the carotid artery.[1] Transient ischemic attacks, cerebral vascular accidents and carotid artery dissections are all related to the second form of Eagle syndrome and were all seen in our patient.[1] CT represents the gold standard for diagnosis, and CT angiography can provide the clinician with further data in regard to the carotid artery. Management can be anything from conservative therapy through definitive surgical removal of the styloid process.[1]

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?
Eagle syndrome can present as an elongated styloid that can compress the carotid artery. If the latter occurs, patients are at risk of stroke and carotid artery dissection.

What is the major impact of the image(s)?
The image depicts a classic example of Eagle syndrome in which the patient suffered from stroke-like symptoms with associated carotid artery dissection.

How might this improve emergency medicine practice?
Emergency physicians should be aware that there are numerous rare causes of stroke-like symptoms, especially in the younger population.

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A Giant Vocal Cord Polyp Mimics Asthma Attack

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CASE PRESENTATION

A 38-year-old Japanese housewife who was a heavy smoker was admitted to our hospital because of upper respiratory distress that developed half a year prior to admission. She had no past medical history. On admission, her vital signs were normal. Physical examination showed hoarseness, stridor, wheezing, and orthopnoea, but no swelling of the tonsils, thyroid, or lymph nodes. A computed tomography of the neck revealed an enlarged tumor (Image A).

DIAGNOSIS

Laryngeal endoscopy revealed a giant vocal cord polyp (Image B). Direct laryngoscopic resection after tracheostomy with local anesthesia was performed, which resulted in improved symptoms. After a week, the tracheal fenestra was closed and she was discharged without complication.

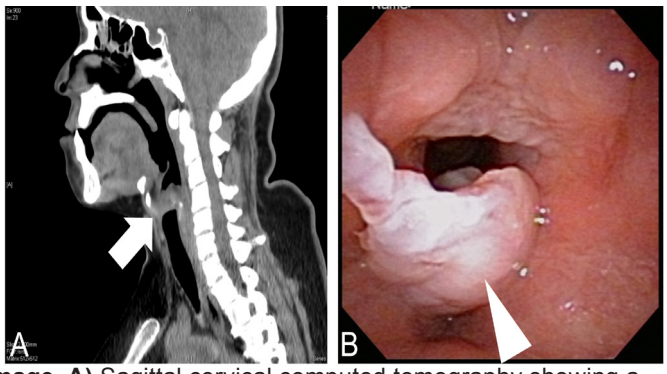

Image. A) Sagittal cervical computed tomography showing a hypodense mass below the epiglottis (arrow).**B)** Laryngoscopy showing an elevated vocal polyp (arrowhead).

CPC-EM Capsule

What do we already know about this clinical entity?
Vocal cord polyps are common lesions, and most are small; the common symptom is hoarseness.

What is the major impact of the image(s)?
Giant vocal cord polyps can mimic asthma and may cause critical airway obstruction leading to sudden death.

How might this improve emergency medicine practice?
Large vocal cord polyp can mimic asthma. Thus, physicians should consider the disease when patients present with a protracted upper respiratory distress history.

Vocal cord polyps are common lesions, with a reported lifetime prevalence of 1.31% to 16.9% of the population.[1] Mechanical or chemical irritation caused by heavy smoking can result in vocal cord polyps.[2] Most vocal cord polyps are small lesions; thus, the common symptom is hoarseness. Occasionally, larger vocal cord polyps causing partial upper airway obstruction can mimic asthma.[3,4] However, giant vocal cord polyps may cause critical airway obstruction leading to sudden death.[5]

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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Wrist Fracture in a Child Irreducible Due to Soft Tissue Interposition

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CASE PRESENTATION

An 11-year-old boy fell onto his outstretched arm. He presented to the emergency department with a deformity of his left wrist. Radiograph revealed a greenstick fracture with volar angulation of the distal radius. The distal ulnar physis was disrupted (Salter-Harris type II) and the proximal metaphyseal fragment was displaced dorsally; however, the distal radioulnar joint was intact (Image 1). Closed reduction of the distal ulna under axillary block failed. Three-dimensional computed tomography (3DCT) was performed before open reduction.

DIAGNOSIS

This type of fracture is known as a Galeazzi-equivalent fracture. Galeazzi-fracture dislocation is a well-known injury, consisting of a distal radial shaft fracture and dislocation of the distal radioulnar joint (DRUJ). It is rare in adults, and even more uncommon in children. In contrast, Galeazzi-equivalent fractures consist of a fracture at the distal radial metadiaphyseal area with complete distal ulnar epiphyseal separation instead of the more common pattern of DRUJ dislocation.[1] The ulnar physeal fracture in a Galeazzi-equivalent injury can be irreducible due to soft tissue interposition (periosteum, extensor tendons, or joint capsule). It is important to identify and analyze these fractures precisely, as growth arrest has been reported after such injuries.[2] 3DCT revealed the interposition of the extensor carpi ulnaris between the fragments, which hindered the reduction; this was confirmed intraoperatively (Image 2). The patient required

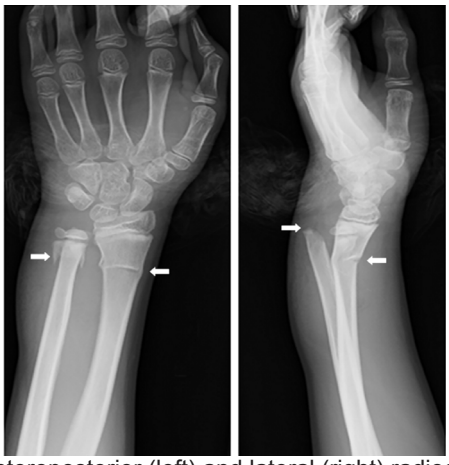

Image 1. Anteroposterior (left) and lateral (right) radiographs of the distal forearm and wrist joint. Radiographs show the radius and ulnar fractures (arrows).

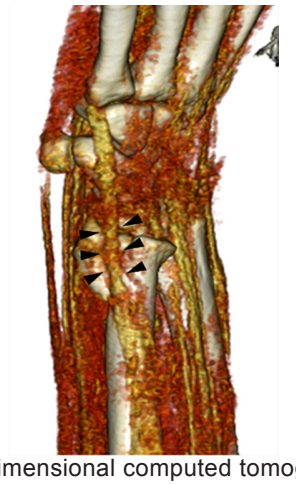

Image 2. Three-dimensional computed tomography shows the interposition of the extensor carpi ulnaris between the fragments (arrows).

open reduction and fixation of the ulnar physeal fracture with two Kirschner wires. He has regained wrist range of motion, with no complications at two-year follow-up.

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?
The ulnar physeal fracture in a Galeazzi-equivalent fracture can be irreducible due to soft tissue interposition, such as periosteum, extensor tendons, or joint capsule.

What is the major impact of the image(s)?
The interposition of the extensor carpi ulnaris (ECU) between the fragments, which hindered the reduction was revealed by three-dimensional computed tomography (3DCT).

How might this improve emergency medicine practice?
This case report reveals the ECU interposition of this Galeazzi-equivalent fracture by 3DCT and shows the difficulty of closed reduction in the emergency department.

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Donut Sign on Magnetic Resonance Angiography: Interpret with Caution

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CASE PRESENTATION

We present two cases of cerebrovascular accidents. Case #1: A 24-year-old man presented with open fractures of the left femur and tibia after a motor vehicle collision. Within two hours, he developed left facio-bracial paresis. Although he arrived in the window period for thrombolysis, polytrauma precluded thrombolysis. His modified Rankin Scale (mRS) score at admission was five. Case #2: A 26-year-old man presented to the emergency department after eight hours with hemiplegia and global aphasia. His admission mRS score was four. Stroke workuprevealed hyperhomocysteinemia (>114 μmol/L).

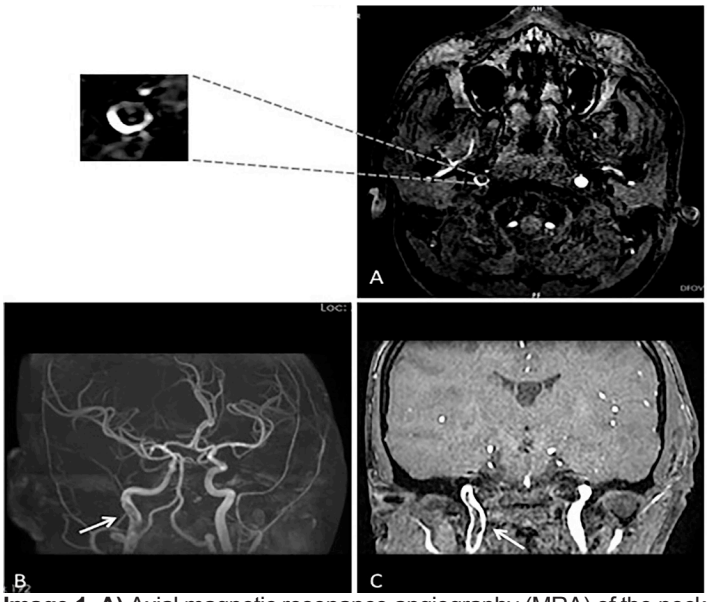

Image 1. A) Axial magnetic resonance angiography (MRA) of the neck shows thrombus in the right internal carotid artery (ICA) (donut sign). **B)** Maximum intensity projection image shows filling defect in the right ICA distal to the bifurcation of common carotid artery (white arrow). **C)** Coronal MRA shows thrombus in the right ICA (white arrow).

DIAGNOSIS

In Case #1, considering the background of trauma and imaging suggestive of luminal filling defect, we suspected an internal carotid artery (ICA) dissection.[1] The proposed mechanism was the hyperextension of the neck due to decelerating forces during trauma, causing stretch of ICA over the cervical vertebrae. This resulted in shear stress and intimal tear, acting as a nidus for the thrombus formation.[2] In Case #2, hyperhomocysteinemia is the risk factor for thrombus formation leading to stroke. After four weeks, there was a complete resolution of thrombus and the mRS score improved to two in Case #2 with oral antiplatelet and anticoagulation therapy, while Case #1 was lost to follow-up. In both cases, family refused to consent for mechanical thrombectomy.

In the literature, luminal thrombus in the ICA has been described radiologically with various analogies such as "donut sign," "crescent sign," or "string sign."[3] Although these radiological signs guide us to localize the lesion, they should be interpreted with caution. In Case #1, maximum intensity projection (MIP) image of magnetic resonance angiography (MRA) head showed a filling defect in the right ICA (Image 1B), while in Case #2 there was no definitive filling defect seen in MRA-MIP of the left ICA (Images 2B and 2C). However, thrombus was identified in the axial source images of the neck in both cases (Images 1A and 2A). The probable explanation for the false negative MIP images in Case #2 could be the flow-related alteration in image enhancement. For instance, intraluminal thrombus may become inapparent if surrounded by hyperintense blood flow and a falsely normal-appearing blood vessel can be reconstructed (Image 3).[4] Therefore, it is always advisable to refer to the individual source images while interpreting the MIP image.[4]

In summary, these images emphasize two practical points: 1) limitations in the interpretation of MRA-MIP image (false negatives); and 2) the significance of evaluating source images of head and neck in stroke workup.

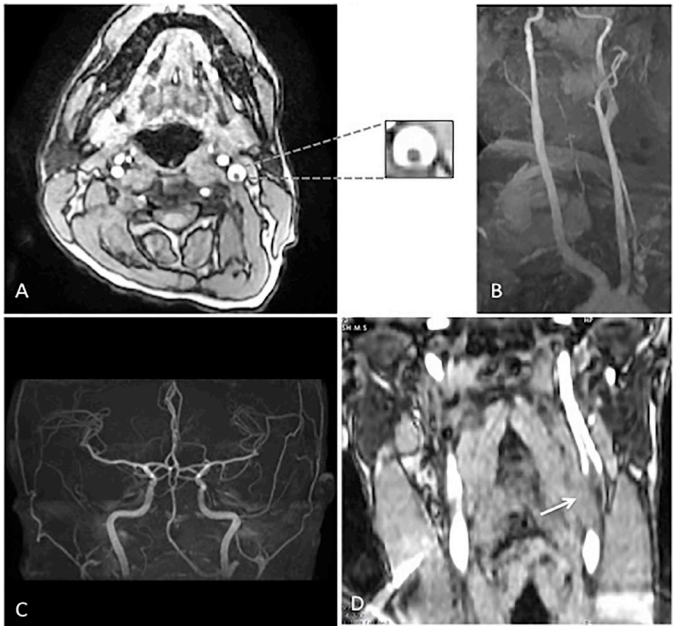

Image 2. A) Axial magnetic resonance angiography (MRA) of the neck shows thrombus in the left internal carotid artery (ICA) (Donut sign), also note an increase in diameter of vessel. **B,C)** Maximum intensity projection images of neck and intracranial vessels shows 'no definitive filling defect' in left common carotid artery or ICA. **D)** Coronal MRA shows thrombus in the left ICA (arrow).

CPC-EM Capsule

What do we already know about this clinical entity?
Cerebrovascular accidents are the most common neurological emergencies. Magnetic resonance imaging (MRI) and MR angiography are the recommended modalities to identify stroke etiology.

What is the major impact of the image(s)?
These radiological images highlight the major limitation of maximum intensity projection images in detecting the intraluminal thrombus.

How might this improve emergency medicine practice?
Knowledge of basic MRI interpretation and its limitation will aid the emergency physician in prompt diagnosis, etiological differentiation and subsequent management of stroke.

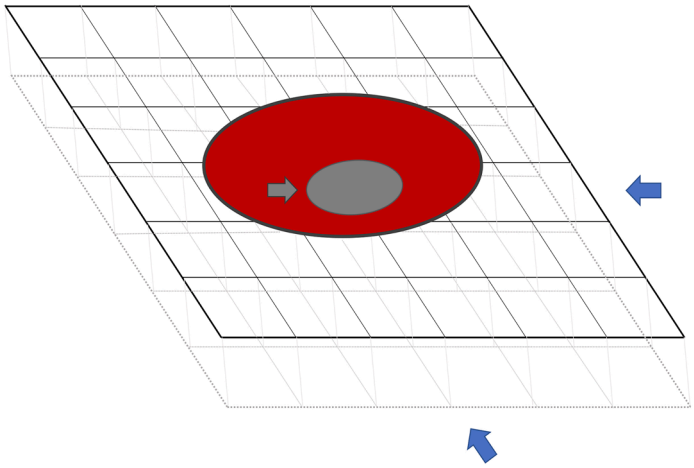

Image 3. Schematic representation of cross-section of internal carotid artery on voxels. No matter what direction the maximum intensity projection (MIP) reprojection algorithm is oriented (as shown by blue arrows), the hyperintense flowing blood (shown as red) may obscure the intraluminal thrombus (grey arrow) on final MIP.

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 Knee Fracture with Underestimated Severity

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CASE PRESENTATION

A 13-year-old girl presented to the emergency department (ED) after her right knee was forced into valgus after making contact with the opposing goalkeeper while playing soccer. At the scene, she had experienced immediate severe knee pain and was unable to bear weight. Anteroposterior radiographs of the knee revealed a minimally displaced fracture to the lateral femoral condyle (Image 1). Computed tomography (CT) revealed injury of the distal femoral epiphyseal growth plate (Salter-Harris type 4), and the point near the epiphyseal closing was tender in the patient (Image 2). Three-dimensional CTs are useful in delineating the coronal shear component (Image 3). Knee arthroscopy revealed severe complications including posterior cruciate ligament ruptures, medial collateral ligament injury, and longitudinal tear of the lateral meniscus anterior horn, in addition to suspicion of these injuries on preoperative magnetic resonance imaging (MRI). The patient underwent open reduction and internal fixation (ORIF) to achieve anatomic reduction.

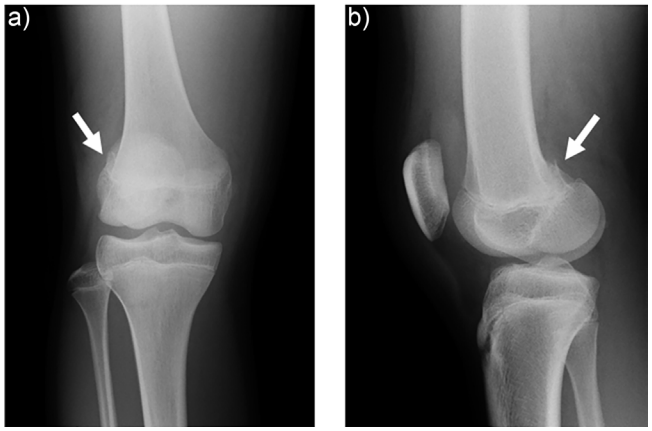

Image 1. Anteroposterior a) and lateral b) radiograph show the injured knee with a minimal fracture of the lateral femoral condyle (arrow).

DIAGNOSIS

Coronal fractures of the femoral condyle, first described by Hoffa in 1904, are uncommon clinical entities, typically seen in adults after high-energy trauma.[1] Historically, poor outcomes have been reported in the literature with non-operative treatment. ORIF has been shown to produce good, long-term clinical results in adults. Hoffa fractures appear to be even more uncommon in a skeletally immature patient. Nevertheless, they should also be treated with ORIF to achieve anatomic reduction, stable internal fixation, and early active mobilization.[2,3] Plain radiographs are not sufficiently sensitive to detect Hoffa fracture fragments, and there is a risk of underestimation. Emergency physicians should not be hesitant to order CTs for determining accurate diagnosis. To confirm ligamentous and cartilaginous injuries, MRI and knee arthroscopy are useful. In the ED, the emergency physician should initially fix the patient's knee using an above-knee splint, and consult an orthopedic surgeon for subsequent ORIF.

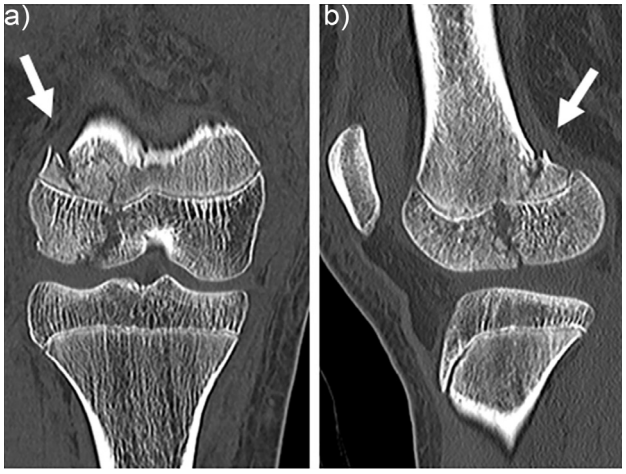

Image 2. Coronal (A) and sagittal (B) views on computed tomography show injury to the distal femoral epiphyseal growth plate (Salter-Harris type 4 and tenderness at the point near the epiphyseal closing) (arrow).

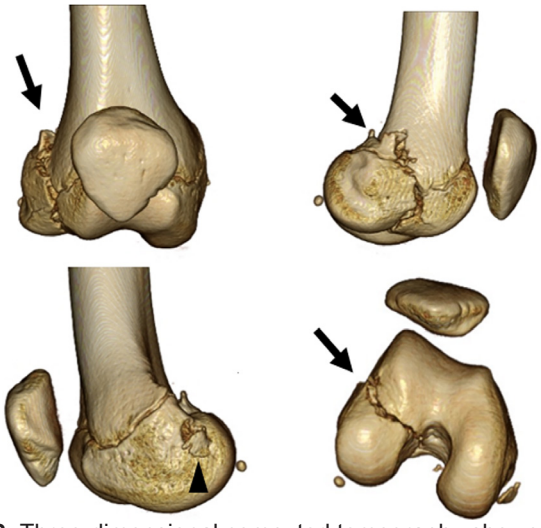

Image 3. Three-dimensional computed tomography showed that the injured knee had a displaced coronal fracture of the lateral femoral condyle (arrows) and avulsion of the attachment of medial collateral ligament (arrowhead).

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?

Hoffa fracture is uncommon, and poor outcomes have been reported with conservative treatment. Plain radiographs are not sufficiently sensitive and there is a risk of under diagnosis.

What is the major impact of the image(s)?

The computed tomography (CT) image in this report demonstrates injury of the distal femoral epiphyseal growth plate and a displaced coronal fracture of the lateral femoral condyle, which are underestimated on plain radiographs.

How might this improve emergency medicine practice?

This case report demonstrates emergency physicians should not hesitate to order CT to accurately diagnose Hoffa fracture.

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Malignant Catatonia Mimics Tetanus

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CASE PRESENTATION

A 70-year-old Japanese man with untreated depression but no history of trauma had fallen seven days prior to hospitalization. After the incident he developed disturbance of consciousness, and his speech gradually became incoherent due to masseter spasm. His vital signs on admission were as follows: blood pressure 97/53 mmHg; pulse 99 beats per minute; body temperature 37.8 °C; respiratory rate 15 breaths per minute; SpO_2, 99% without oxygenation; Glasgow Coma Scale Eye opening 3, Verbal response 3, Motor response 2. Physical examination revealed a back abrasion, stupor, and spasmodic laughter (Image). Blood tests including markers of inflammation and creatinine kinase, urinalysis, cerebrospinal fluid, blood cultures, imaging, and electroencephalography findings were normal. Administration of human tetanus immunoglobulin, tetanus toxoid, and penicillin did not improve the patient's symptoms. On day two, blood tests were normal; thus, we administrated 5 mg diazepam. After that, we observed remarkable improvement in the patient's consciousness, trismus, and fever.

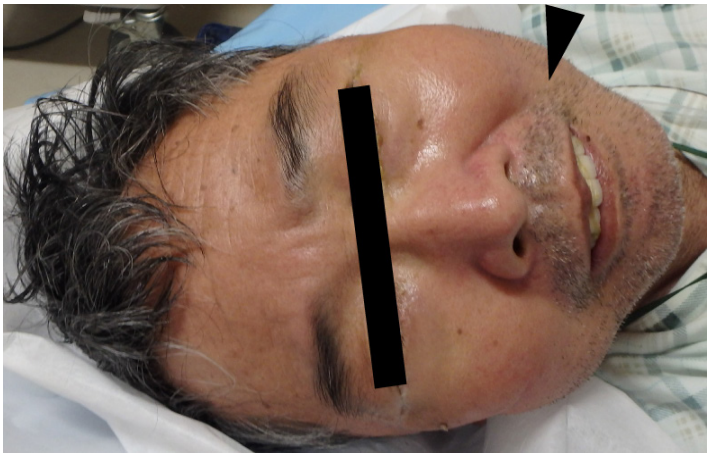

Image. Patient shows sardonic smile (arrowhead) and stiff neck with fever and coma.

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

DIAGNOSIS

Catatonia is found in 10% of psychiatric inpatients, but malignant catatonia (MC) is rare.[1] Catatonia is mainly caused by primary psychiatric, neurologic, metabolic and drug-induced disorders, as well as brain injury.[2] Catatonia is most commonly characterized by mutism, stupor, posturing, and hypokinesis.[3] Fever and autonomic dysregulation due to MC often lead to fatal consequences,[4] with a mortality rate exceeding 50%.[5] Evidence suggests that MC represents a disturbance of dopaminergic and gamma-aminobutyric acid receptors,[6] as administration of 1 – 2 mg lorazepam typically leads to rapid resolution of symptoms within two hours.[7] Such treatment should be used within 24 hours after excluding alternative diagnoses.[8] Because diagnosis is often difficult and delayed,[9] administration of low-dose benzodiazepines (e.g., five mg diazepam) may be warranted in patients with a history of psychological disorders presenting with MC symptoms.

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CPC-EM Capsule

What do we already know about this clinical entity?
Malignant catatonia (MC) often leads to fatal consequences. Administration of low-dose lorazepam typically leads to rapid resolution of symptoms; thus, definite diagnosis is crucial.

What is the major impact of the image?
Because MC resembles tetanus, diagnosis is often difficult and delayed.

How might this improve emergency medicine practice?
Administration of low-dose benzodiazepines may be warranted when patients presenting with MC symptoms have a history of psychological disorders and normal blood, urine, cerebrospinal fluid testing and imaging.

Adult Male with Left Arm Pain and Swelling

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CASE PRESENTATION

A 64-year-old male with a history of intravenous drug abuse presented to the emergency department (ED) with left arm pain and swelling for four days. Left upper extremity exam revealed diffuse swelling, erythema and tenderness in the mid-distal forearm. A point-of-care ultrasound (POCUS) was performed to characterize the suspected abscess for incision and drainage; however, imaging revealed a severely enlarged radial artery, suspected to be a pseudoaneurysm with an approximate diameter of 3.71 cm (Image 1, Video). Computed tomography of the extremity revealed an aneurysmal radial artery (Image 2). The patient was transferred to the operating room where the diagnosis was revealed.

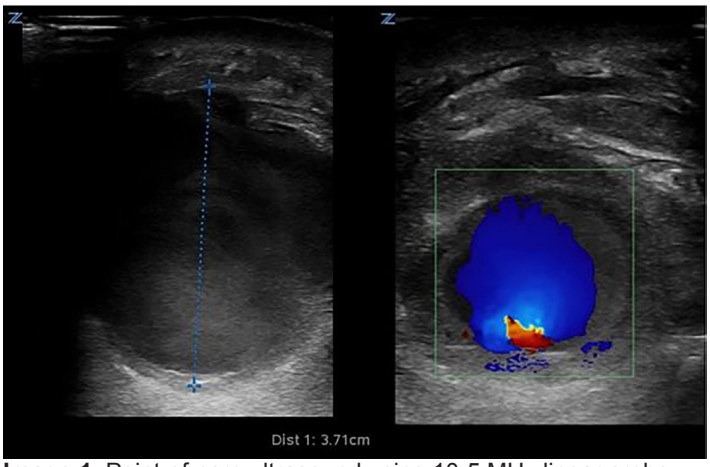

Image 1. Point-of-care ultrasound using 10-5 MHz linear probe demonstrating a large 3.71 cm aneurysmal dilation of left radial artery (left). Color Doppler revealing significant pulsatile and turbulent flow (right).

DIAGNOSIS

Radial Artery Mycotic Aneurysm

Mycotic aneurysms are rare, with annual prevalence of 0.03% of injection drug users who present to the ED with complications from injection.[1] Far more common is the development of an abscess or cellulitis at the injection site. However, due to the lethality of attempted blind incision and drainage of an aneurysm or pseudoaneurysm, it is critical to properly distinguish these from an abscess or cellulitis.

POCUS is a safe, accurate, and cost-effective modality to distinguish between these diagnoses, and can alter management by 73% when compared to clinical judgment.[2] The sonographic appearances of an abscess and cellulitis have been well described.[3] An abscess most commonly has an anechoic or hyperechoic spherical appearance with lobulated or irregular borders as well as possible posterior acoustic enhancement.[4,5] The use of color Doppler to identify any blood flow within an anechoic structure can be a critical step to avoid a catastrophic incision into a blood-filled structure. The patient was taken to the operating room where surgeons revealed a purulent aneurysmal radial artery that was treated with ligation and debridement, parenteral antibiotics, and wound vacuum dressings.

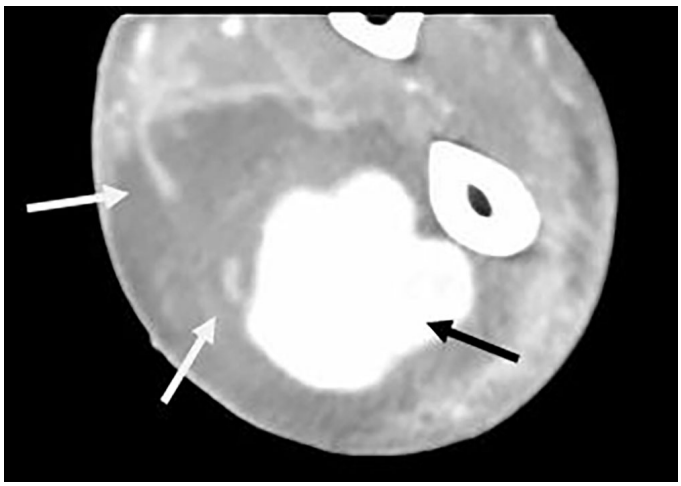

Image 2. Subsequent computed tomography with intravenous contrast demonstrating a 3.2x3.4x5.2 cm radial artery aneurysm (black arrow) within the anterolateral compartment of the distal forearm, as well as diffuse cellulitis of anterior forearm soft tissue with phlegmonous changes (white arrows).

Video. Radial artery mycotic aneurysm.

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?
Mycotic aneurysm is a rare but serious presentation among intravenous drug abusers presenting with seemingly classic skin and soft tissue infection at injection sites.

What is the major impact of the image(s)?
Point-of-care ultrasound (POCUS) of suspected abscesses prior to incision and drainage is an effective method of securing this diagnosis and ensuring you are not incising something else.

How might this improve emergency medicine practice?
This case further supports the use of POCUS in helping emergency providers to avoid rare but potentially catastrophic complications during routine abscess evaluation and management.

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A Brush with Danger

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CASE PRESENTATION

A five-year-old male presented to the emergency department (ED) after falling off a stool while brushing his teeth, resulting in a penetrating oropharyngeal injury. The toothbrush was impaled deeply into the left lateral soft palate with only the handle of the toothbrush visible protruding from the mouth. The patient was hemodynamically stable and was comfortable in the arms of his mother after a single dose of fentanyl. A computed tomography angiography (CTA) with three-dimensional reconstructions was performed without sedation, which showed the tip of the toothbrush terminating adjacent to the alveolar and pterygoid branches of the external carotid artery, but with no visible injury to these vessels (Image). Associated subcutaneous emphysema and muscle edema were also noted. The otorhinolaryngology service was consulted, and the toothbrush was successfully removed in the operating room without complication.

DISCUSSION

Although the distance from the tonsillar fossa to the internal carotid artery is approximately only 25 millimeters,[1] most children with penetrating oropharyngeal trauma can be managed expectantly. Rarely, these injuries can damage the internal carotid artery leading to thrombosis or dissection of the affected vessel risking a cerebrovascular accident.[2,3] In a previous single-center cohort of children with penetrating palate trauma, the risk of stroke was 0% (95% confidence interval 0-2.5%).[4] Although most patients with penetrating palate injuries do not require imaging, CTA is the first-line imaging modality for the ED clinicians with a sensitivity between 90-100% for vascular injury when compared to angiography.[5]

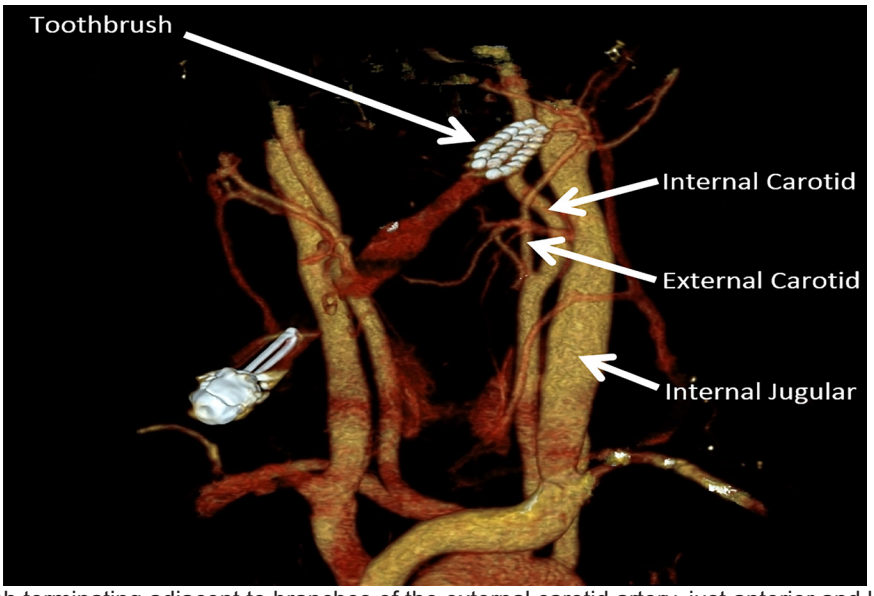

Image. Tip of the toothbrush terminating adjacent to branches of the external carotid artery, just anterior and lateral to the left internal carotid artery and adjacent to the internal jugular vein.

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?
Children with penetrating oropharyngeal trauma are at risk for vascular injury, although vascular complications are rare.

What is the major impact of the image(s)?
The image demonstrates the proximity of the vasculature to the soft palate and highlights the potential for vascular injury.

How might this improve emergency medicine practice?
Most penetrating oropharyngeal injuries do not require imaging, but if the exam or mechanism is concerning, computed tomography angiography should be the first-line imaging modality.

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Point-of-care Cranial Ultrasound in a Hemicraniectomy Patient

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CASE PRESENTATION

A 74-year-old male presented to the emergency department with right-sided weakness and confusion and was found to have a left parietal intraparenchymal hemorrhage with cerebral edema and left-to-right midline shift on non-contrast computed tomography (CT) of the head. Increase in cerebral edema and expansion of the hematoma caused clinical neurological decline necessitating a left-sided hemicraniectomy with clot evacuation. A cranial ultrasound was performed two days after surgery to assess for progression of cerebral edema and intracranial hemorrhage. A transtemporal approach in axial plane was used to visualize intracranial structures through the craniectomy window (Images 1 and 2). Physiological structures such as the falx cerebri, lateral ventricles, midbrain,

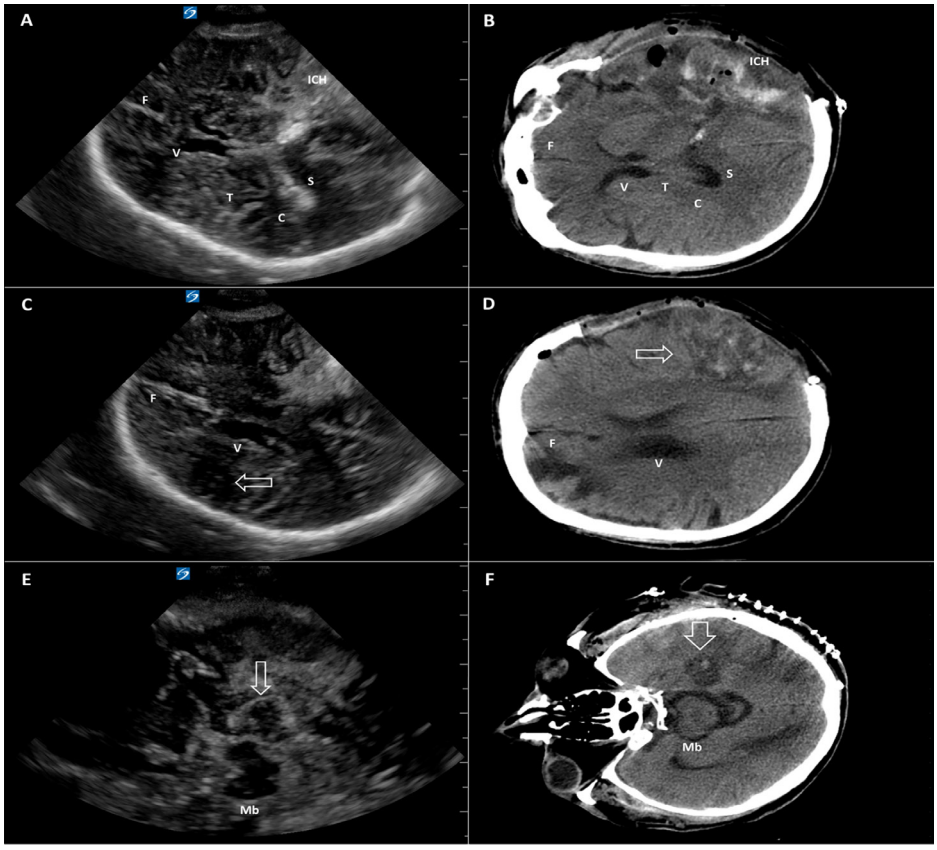

Image 1. Cranial ultrasound through the left transtemporal window (left column, panels A, C, E), with corresponding cross-sectional anatomy on a non-contrast computed tomography of brain (right column, panels B, D, F) in a 74-year-old male with a left hemicraniectomy. Intracranial hemorrhage (ICH) and hypodense ischemic areas are indicated by white arrows.
F, falx cerebri; *V*, lateral ventricles; *Mb*, midbrain; *C*, choroid plexus; *S*, splenium of corpus collosum; *T*, thalami.

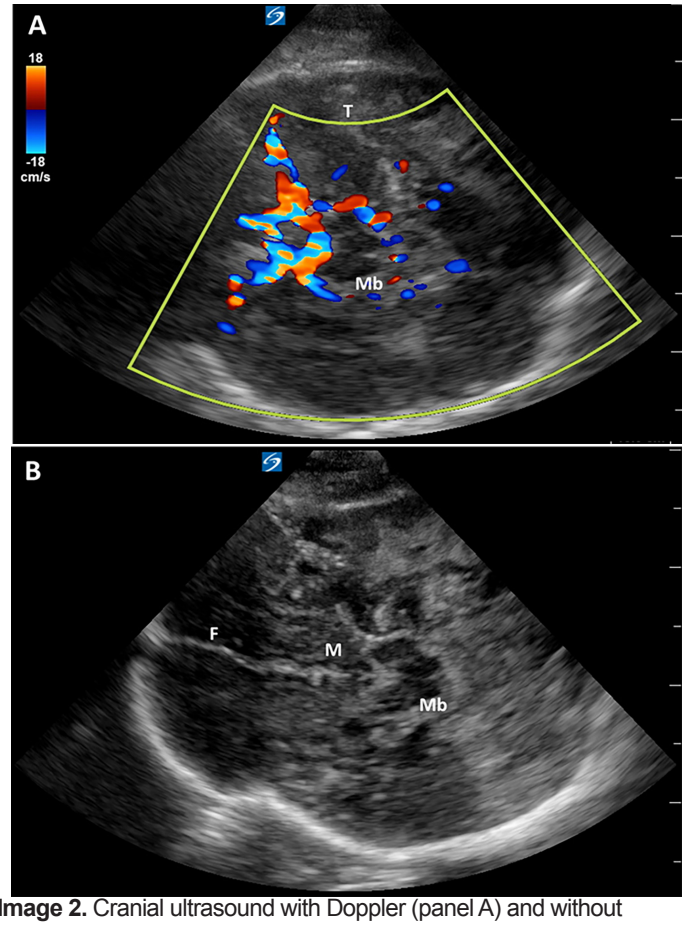

Image 2. Cranial ultrasound with Doppler (panel A) and without Doppler (panel B) through the left transtemporal window in a 74-year-old male with a left hemicraniectomy with additional anatomical details. *T*, thalami; *Mb*, midbrain; *F*, falx cerebri; *M*, mammillary bodies.

CPC-EM Capsule

What do we already know about this clinical entity?
Point-of-care ultrasound (POCUS) is widely used in the emergency departments (ED) as a tool to visualize anatomy without exposing patients to potentially harmful radiation.

What is the major impact of the image(s)?
The ability to view anatomical structures and pathology of patients with hemicraniectomies using POCUS may decrease radiation from repeat computed tomography.

How might this improve emergency medicine practice?
POCUS scans on patients with hemicraniectomies will allow for faster assessment of structures, pathology and cerebral perfusion in the ED.

Cranial ultrasound has potential applications in point-of-care assessment of intracranial pathology in neurocritical care patients. This application has promising use in directing therapy in patients who are otherwise unstable for transport and may provide a noninvasive, radiation-free diagnostic tool for serial neuroimaging.

mammillary bodies, choroid plexus, splenium of corpus callosum, thalami, and circle of Willis were visualized with incredible anatomical detail. Pathologies such as intracranial hemorrhage, focal ischemic areas, and vasogenic edema, as well as encephalomalacia, were identified with close correlation to the non-contrast head CT. The patient is currently recovering in the neurocritical care unit with supportive care.

DISCUSSION

Visualization of intracranial structures by ultrasound in adults is limited by the presence of skull, although ultrasound imaging can occur through temporal windows. Point-of-care ultrasound allows for the assessment of midline shift, brainstem, and ventricles, and Doppler allows visualization of cerebral perfusion patterns.[1,2] Patients with a hemicraniectomy have better temporal windows available since a portion of their skull has been removed. In such patients, ultrasound can provide a non-invasive method to serially assess midline shift, intracranial hematomas, and focal ischemia at the bedside.

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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Chief Complaint: "There Is Something Burning in my Mouth"

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CASE PRESENTATION

A 50-year-old Hispanic male with a history of diabetes presented to the emergency department with a painful maxillary mass for 12 days. He had been previously treated with antibiotics without improvement. Review of systems was significant for fever, diaphoresis, weight loss, and malodorous breath. Physical exam revealed poor dentition, mild tenderness to palpation of the maxillary sinuses and a 2.5 x 4 cm yellow, rubbery lesion on the hard palate (Image 1). The mass was pliable and adherent. Computed tomography of the face revealed irregularities of the hard palate, subcutaneous emphysema, and chronic sinusitis (Images 2 and 3).

DIAGNOSIS

Rhinocerebral mucormycosis, an infection of the nasal and paranasal sinuses, is the most common presentation of the mucormycosis spectrum.[1] Five hundred cases are reported in the United States each year.[2] The fungi are found in dead and decaying matter such as soil but thrive in acidic glucose-rich environments.[1,3] Infection begins with fungal seeding of the sinuses in an immunocompromised host (e.g., patients with malignancy, chronic steroid use, acquired immunodeficiency syndrome, and diabetes), who are predisposed due to decreased phagocytic activity of neutrophils and monocytes.[1,3] From the sinuses, the fungus spreads to the orbits, oropharynx and mouth.[1] When left untreated, *Mucor* can extend into the brain, cranial nerves, lungs, gastrointestinal system and kidneys, leading to vaso-occlusive thromboemboli, tissue infarction, and necrosis.[1] Patients often present with indistinct symptoms such as headaches, low-grade fever, weakness, purulent nasal drainage, nasal congestion, nose bleeds, sinusitis, oral ulcers, and facial and periorbital pain.[1]

Our patient promptly received intravenous antifungals, including amphotericin B upon admission. Flexible laryngoscopy showed necrotic changes. A bilateral inferior maxillectomy was performed and a prosthetic palatal obturator

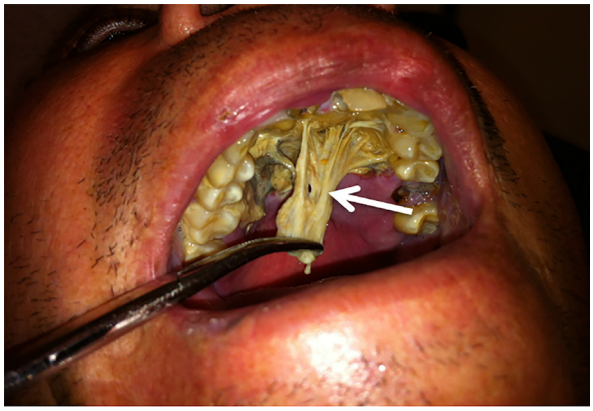

Image 1. Demonstration of yellow, rubbery lesion found on the hard palate (white arrow) of the patient that upon biopsy revealed non-septated hyphae resembling Rhizopus species.

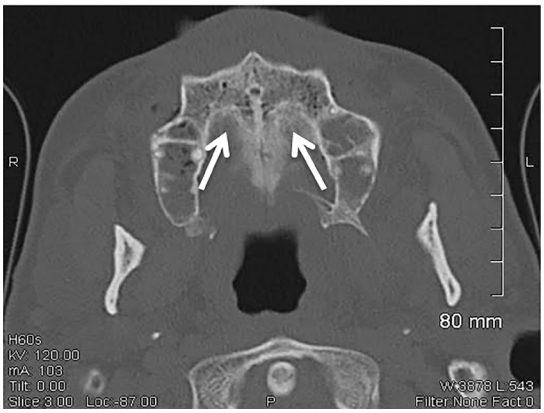

Image 2. Axial view of a computed tomography scan of facial bones showing cortical irregularity of the hard palate, including submucosal emphysema (white arrows).

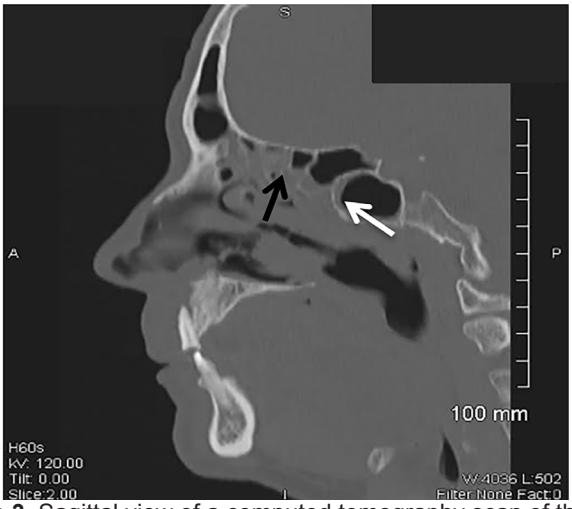

Image 3. Sagittal view of a computed tomography scan of the facial bones revealed extensive acute and chronic sinusitis of the sphenoid (white arrow) and ethmoid sinuses (black arrow).

was fitted for the patient. He remained on intravenous amphotericin B and later switched to oral posiconazole for completion of the six-month treatment.

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?
Rhinocerebral mucormycosis is the most common presentation of the mucormycosis spectrum and is most commonly found in immunocompromised individuals.

What is the major impact of the image(s)?
The images displayed are a visual demonstration of Mucor's invasive abilities as well as the extent of bone destruction that it can cause.

How might this improve emergency medicine practice?
This case presentation reflects the significance of keeping a broad differential diagnosis, as a missed opportunity to diagnose this rare illness can result in death.

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A Case of Necrotic Skin Lesions on the Abdomen

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CASE PRESENTATION

A 42-year-old female with a history of type II diabetes, partial left nephrectomy, and fibromyalgia was transferred from an outside hospital for concerns of a painful rash on her abdomen and flanks. She was admitted for sepsis and acute kidney injury at the outside hospital three weeks prior to arrival, and was discharged one week later on subcutaneous enoxaparin for deep vein thrombosis prophylaxis. She noticed bruising and rash to her bilateral lower abdomen one week after discharge with progressive pain. She presented to an outside emergency department (ED) for rash and pain control. Abdominal computed tomography showed diffuse body wall edema with no subcutaneous air. The local consulting surgeon did not believe the patient had necrotizing fasciitis but was unsure of diagnosis of the rash. She received piperacillin/tazobactam, vancomycin, and one unit of packed red blood cells prior to transfer. Upon arrival to our ED, physical exam showed tender necrotic firm lesions to her bilateral lower abdomen and flanks with surrounding erythema (Images 1 and 2).

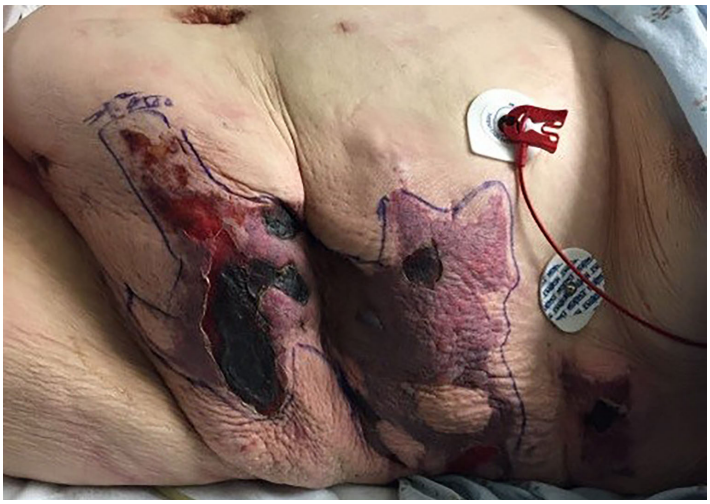

Image 1. Photo of patient's left lower abdomen and flank showing areas of erythema and centralized necrotic lesions representative of calciphylaxis outlined by surgical marking pen.

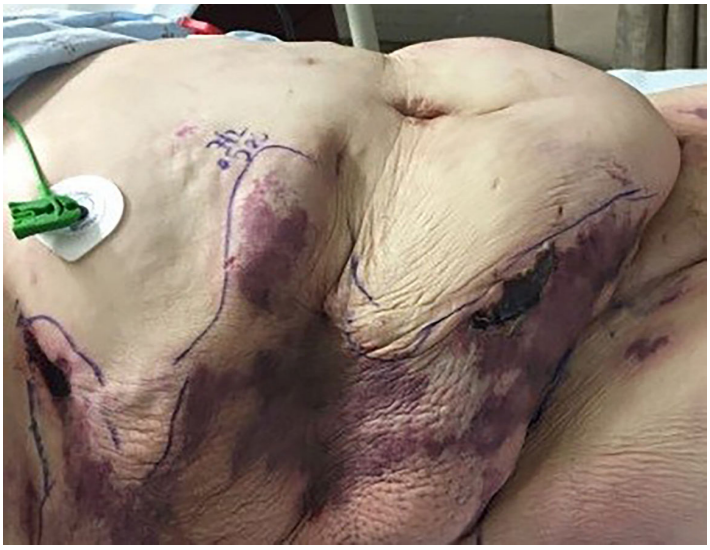

Image 2. Photo of patient's right lower abdomen and flank showing areas of erythema and centralized necrotic lesions representative of calciphylaxis outlined by surgical marking pen.

DISCUSSION

Diagnosis: Non-uremic Calciphylaxis

Calciphylaxis is a rare, life-threatening vascular syndrome characterized by calcification of microvasculature causing thrombosis and resultant soft tissue ischemia and necrosis. Diagnosis is made clinically, supported by skin biopsy demonstrating arterial calcifications and occlusions without vasculitis. It is most commonly seen in patients with end-stage renal disease or imbalance in calcium homeostasis with an incidence of 35 cases per 10,000 patients.[1] In a review of 36 cases of nonuremic calciphylaxis, the majority of patients were found to have hyperparathyroidism or recent glucocorticoid use as a risk factor.[2,3] Consequently, its occurrence in our patient, who had no known risk factors for nonuremic calciphylaxis, was an extremely rare event.

In consultation with general surgery and dermatology, we concluded that infectious or vasculitic etiologies were the most likely causes. A wound-edge biopsy demonstrated areas of vascular calcification, intimal hyperplasia, and scattered soft

tissue calcinosis consistent with calciphylaxis. Dermatology recommended treatment of the lesions with sodium thiosulfate, 0.25% acetic acid irrigation, and collagenase ointment. She was admitted and completed a course of vancomycin and ceftriaxone and subsequently was discharged home without 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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CPC-EM Capsule

What do we already know about this clinical entity?
Literature has described calciphylaxis frequently in patients receiving dialysis or with uremia, and it is associated with high mortality.

What is the major impact of the image(s)?
These images, which show the visual appearance of calciphylaxis in a patient without end-stage renal disease, will aid in the recognition of this entity and its inclusion in differential diagnosis.

How might this improve emergency medicine practice?
This is a rare presentation that mimics other life-threatening conditions. Greater awareness of calciphylaxis will lead to earlier diagnosis, consultation and treatment, as outcomes are historically poor.

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Blinking Bug Bite

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CASE PRESENTATION

A three-year-old female presented to a community emergency department with a one-day pruritic rash on her knee. The patient and the parents noted that the rash blanched intermittently and that this blanching appeared to be what they called a "blinking" bug bite. Physical examination revealed a normal child with no heart murmur and two bullous lesions around the left knee that blanched in a pulsatile fashion, corresponding to the femoral pulse (Images 1 and 2, Video).

DIAGNOSIS

A review of the medical literature revealed a single "Images in Clinical Medicine" from the *New England Journal of Medicine*, which revealed a severe dermatitis of the lower extremities that had similar presentation.[1] No other cases were noted in the literature. The wounds were anesthetized and debrided for concern of a staph infection. The patient was placed on antibiotics and healed uneventfully. The case presented here represents an example of a physical manifestation of Quincke's sign, not related to aortic insufficiency but to the rarely-noted effect of intense arterial dilatation in the bullous inflammation of the affected subcutaneous area. Quincke's pulse is a physical finding of aortic insufficiency and, as in this case, focal arterial dilatation. Here, the arterial dilatation in the area of the bite led to an inability of arterioles to maintain sufficient pressure during diastole, resulting in the pulsating blanching and flushing that produced a "blinking" bug bite.

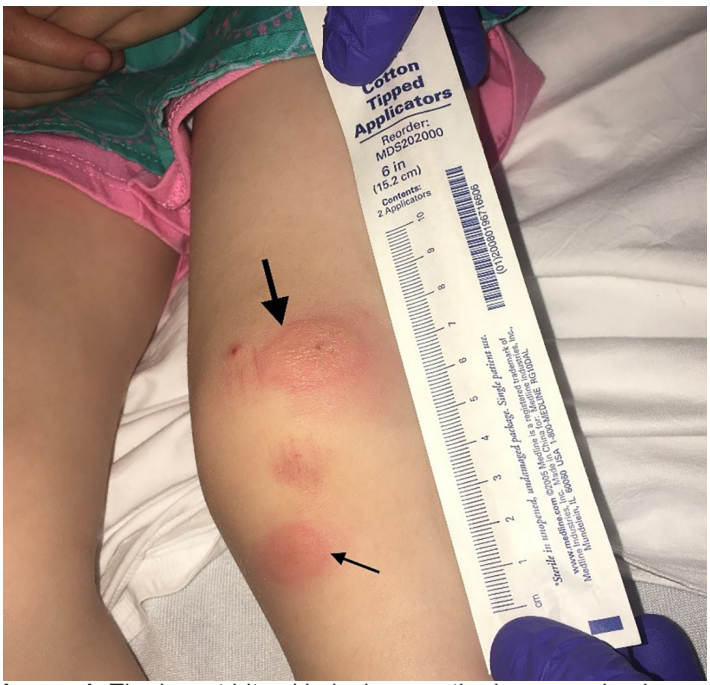

Image 1. The insect bite skin lesions on the knee varying in size (arrows).

Video. This recording demonstrates the blinking bug bite, a manifestation of Quincke's pulse.

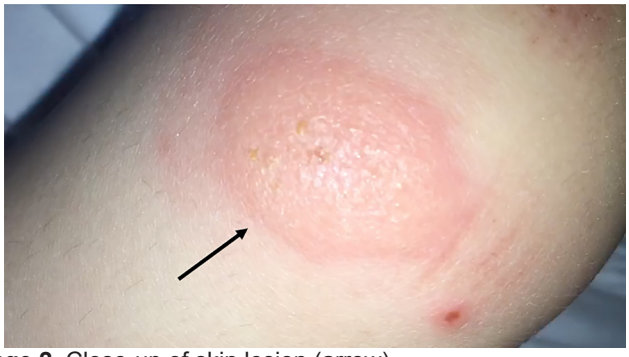

Image 2. Close-up of skin lesion (arrow).

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?
Google Scholar and PubMed reveal only one case report of a "blinking" bug bite. However, searchable online blog posts reveal similar descriptions of these bites.

What is the major impact of the image(s)?
The images here demonstrate a classic example of the "blinking" bug bite, which has not been well described in the medical literature.

How might this improve emergency medicine practice?
These images and video may act as a reference for emergency physicians to more easily diagnose this benign condition.

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This Rash Puts You in the ICU

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CASE PRESENTATION

A 33-year-old female with a history of psoriasis presented to the emergency department with a diffuse, pruritic skin rash that had been progressive for two days. She complained of associated subjective fever, chills, and myalgias. Her exam revealed a diffuse erythematous, blanching, non-tender rash to the face, body, and extremities (Images 1 and 2). The rash did not involve mucus membranes, but there was involvement of the palms and soles. There was scaling over the extensor surfaces and sparing of the flexor surfaces. The patient had been admitted to the hospital several weeks prior for a similar rash requiring intensive care unit (ICU) admission, steroids, and methotrexate.

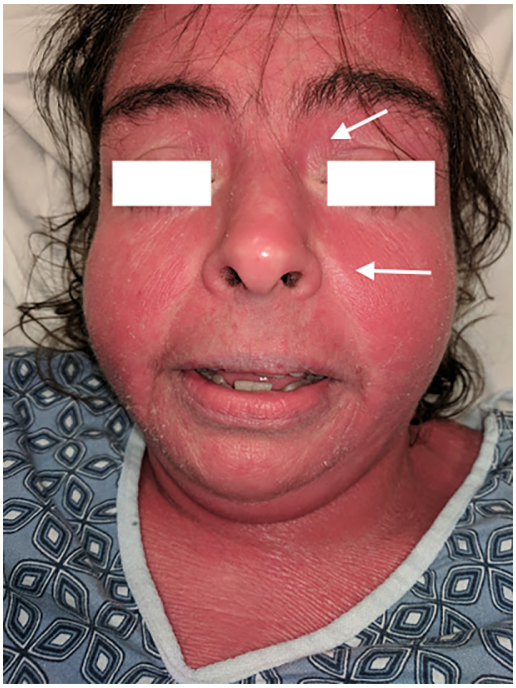

Image 1. Erythroderma flare facial rash notably spares the periorbital area and nasolabial folds (arrows).

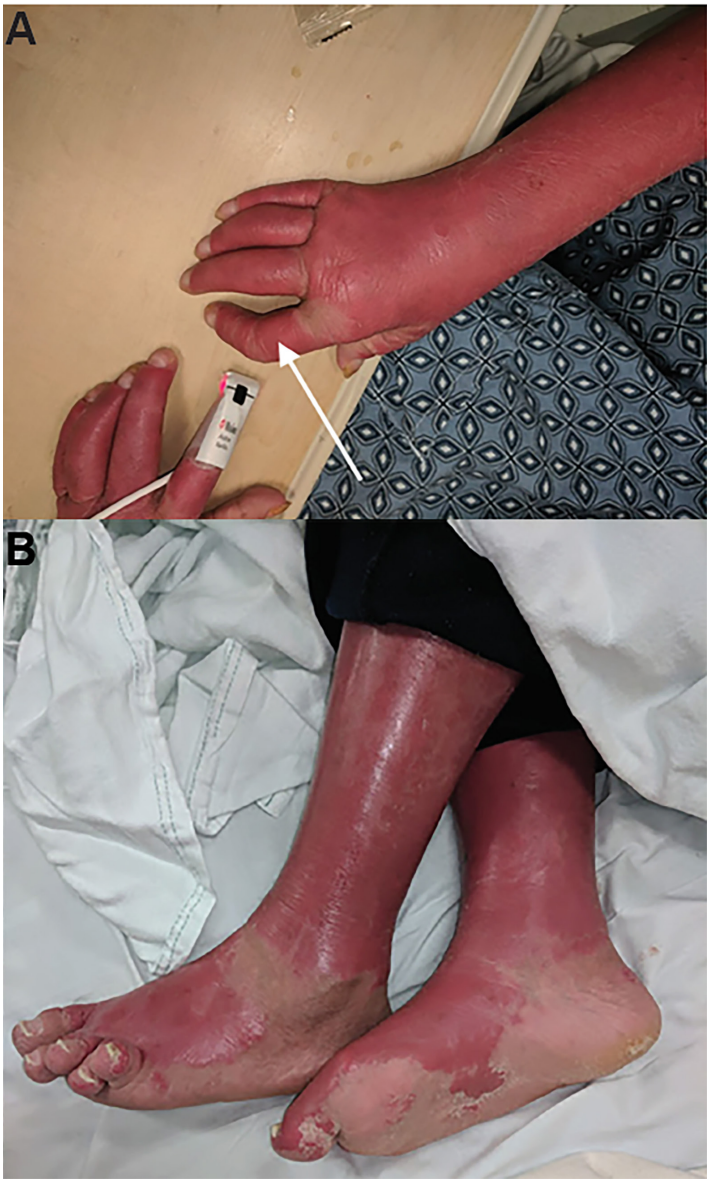

Image 2. Erythrodermic psoriasis flare to the upper (A) and lower (B) extremities demonstrating dactylitis and Dupuytren's contracture (arrow) consistent with long-term psoriatic arthritis.

DIAGNOSIS

Erythroderma (erythrodermic psoriasis flare). Erythroderma, or "red skin," is a severe cutaneous condition that presents with diffuse erythema involving greater than 75% of the body's surface and skin exfoliation.[1] It classically spares the periorbital regions and nasolabial folds. This patient also demonstrates findings consistent with long-term psoriatic arthritis such as Dupuytren's contractures and dactylitis.[2,3] While a prior visit required ICU admission, during this visit her vital signs were normal and she had an uneventful hospital course after the resumption of corticosteroid 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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CPC-EM Capsule

What do we already know about this clinical entity?
The majority of cases of erythroderma are secondary to exacerbation of a prior skin condition such as psoriasis. Important triggers include recent withdrawal of corticosteroid or antipsoriatic therapies, infection, and stress. Patients experience impaired temperature regulation and increased insensible losses. Increased skin breakdown makes these patients prone to superimposed cutaneous infections.

What is the major impact of the image(s)?
These images should aid in physician recognition of a potentially life-threatening rash. Patients with severe erythroderma flare as depicted here require supportive care in the form of temperature monitoring, fluid resuscitation, and electrolyte repletion. In severe cases, these patients may require admission to a burn care unit.

How might this improve emergency medicine practice?
Appropriate recognition of, management, and disposition of patient's suffering from erythrodermic psoriasis flare.

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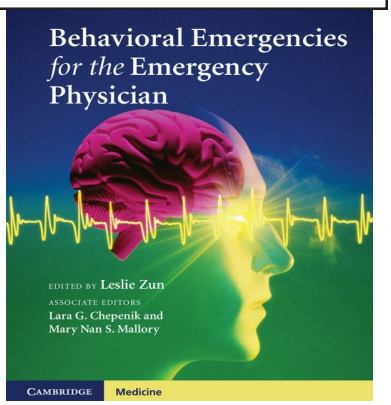

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