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EMRA

Hurricane Haircuts: What My Peers Are Made Of

Morgan Sweere
MD, MPH

Editor-in-Chief, EM Resident
Secretary, EMRA Board of Directors
University of Florida – Jacksonville

"I could not have gotten through residency without these people."

I sat in our EMRA suite at ACEP 2024 Scientific Assembly, tearful. I am not a person who cries often. This conference taught me just how much this work and these people mean to me. I would not be the same physician without them. Being on the EMRA Board this past year has been an unexpected blessing, and one of the biggest reasons is the other board members and residents I serve with. We sat in the suite talking with each off-going board member, reminiscing on memories of hard and fun times together and simply taking a moment to recognize the strengths and contributions of each person to this organization and to our lives.

My entire residency class came to ACEP, as the third-years at our program do annually, so the week was a time of learning — but also of friendship and celebration for how far we have come. Residency is a busy time, so it is not often, aside from our class days off, that our entire class is at an event together.

As I am acutely aware of the time counting down until the end of my residency training, I recognize more so than ever before the power of a cohort. Throughout our education, we have always been part of a group like this: a group of pre-medical students, a group of medical students, and then a group of residents. At the end of it, we will finally go our separate ways, no longer trainees in medical education for the first time. There is something, to me, that makes the residency cohort particularly special and influential.

Throughout the final year of residency, I've recognized how my thought process has changed regarding the culture at work, my goals and my future, and especially my patient care. I see myself noticing things I hadn't before. "How will I handle this situation in the 'REAL' world next year?" is a question I think about daily. What kind of shop do I want to work at? What kind of physician do I want to be?

Every emergency department I've worked in has truly had an elite workplace culture. It is a unique and special place in the hospital, where the formal hierarchy I see in other departments turns instead to teamwork and family. Techs, nurses, physicians – these are all people I've learned an immense amount from over the years.

What I'd like to focus on specifically is camaraderie during residency and how much we truly learn from those we sit beside every day, our co-residents. It's more than post-shift drinks and elite Spotify playlists on shift. It's every moment in the department where we see what makes our colleagues as great as they are.

I recall early in my first year of residency, we entered hurricane season in Florida. This means residents who live in these areas are separated into hurricane teams, some who may stay in the hospital until the storm passes. During the hurricane, some unhoused citizens came to the hospital for shelter. I stood watching one of my upper level residents grab a razor and care for a man by giving him a haircut (and honestly, a pretty decent one at that). I walked up to him, asking him what he was doing. He said, **"I saw something I could do to help, so I did it."** I've thought about that moment several times over the course of my residency, because sometimes, the things we can do to help aren't medical care at all.

I saw a patient a few months ago come into the department, as I was supervising one of the lower level residents. The patient had overdosed while attempting to dye her hair, and received naloxone with EMS. She was still quite sleepy and was being observed in the emergency department. I noticed she still had bleach in her hair. I grabbed one of the techs, and we took the patient to the decontamination room to wash the bleach out of her hair. For those of you who do not bleach your hair, leaving it on your hair too long can definitely destroy your hair and make it all fall out. My co-resident came up to me, asking what I was doing. I looked at her with a smile, remembering that hurricane haircut, and said, **"I saw something I could do to help, so I did it."** Whether that made any kind of difference in the patient's life is probably debatable, but sometimes, I think the little things like that are the things the patients notice most from us as physicians.

The years of residency training test each of our resilience, without a doubt. Over the past 2.5 years, I've learned a lot of emergency medicine (and still have a lot to go!). I've also learned something else: **what my peers are made of**. Amid the craziness on each shift, I look at each of my co-residents and see traits I wish to develop in myself. I have a lot to admire in each of my co-residents: their strength, adaptability, leadership, generosity, and so much more.

I am incredibly proud to be a member of EMRA. Honored to be a part of this cohort, this specialty, this amazing group of people working hard every day to make a difference in emergency departments across the country.

I encourage each of you to take a moment next time you are on shift, at a conference, or in a lecture — just look around and pause at the greatness sitting in the chairs next to you.

References available online.

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From Burnout to Breakthrough

David Wilson, MD
University of Cincinnati
EMRA President, 2024-2025

Monitors beep, a patient calls out, "*Nurse!*" with a rasp.

It's 3:47 a.m.

Three more hours of my shift, but my body feels like it stopped working hours ago; it's my first night shift in a string, and I didn't nap beforehand. I try to focus on writing notes — GSW to the chest that went directly to the OR, flash pulmonary edema that's now comfortable on BiPAP, low-risk chest pain that's now (hopefully) safe at home. I realize I'm referring to patients by their disease and chief complaints, something I swore I wouldn't do when I started residency. I check my phone and see a text from my college friends group chat—"*Dave, when can we catch up? It's been forever!*" I close my eyes and sigh; they're all vacationing in Costa Rica, but I'll be lucky if I make it to one of their weddings in 3 months.

Welcome to emergency medicine training: rewarding, and relentless.

Residency is designed to challenge us so that, as attendings, we don't need to rise to challenges, but instead can fall to the level of our training. Long hours, unpredictable schedules, and the emotional toll of caring for people on the worst day of their lives sets us up for burnout. Yet we thrive in chaos, find purpose in crisis, and rise every day to serve. I want to reflect on the struggle of burnout, offer hope for balance, and show how meaningful change lies not in isolation, but in uniting as a community.

Burnout isn't just a buzzword in EM — it's a lived reality. I remember a particularly grueling week: long shifts, patients with unrealistic expectations, grumpy consultants, conference on my only day off. I feel lucky because my program allows plenty of autonomy and I find value in the work I do, but driving home after that last shift, I caught myself dwelling on frustrations and overlooking the human side of medicine. That scared me. I love this field — the fast pace, the problem-solving, the impact — but in trying to be a good doctor I was losing sight of what brought me to it in the first place.

I know I'm not alone in this. Burnout in EM is among the highest in all specialties. It's not just physical exhaustion; it's the emotional toll of giving without refueling. The quiet guilt of missing family milestones, friendships fading, self-care relegated to a mythical "later."

Yet I see resilience in all of us, a shared understanding that drives us to keep showing up for our patients and each other. But resilience alone isn't enough. If we keep pushing forward without addressing the causes of burnout, we risk losing not only our well-being but also the joy and passion that brought us to EM in the first place.

One lesson I've learned in residency is that we are never truly alone. It might feel that way at 3 a.m. on a chaotic shift, but step back, and you'll see the community we belong to. Emergency medicine is a family—a tribe of people who understand the unique challenges of what we do.

References available online.

This is where organizations like EMRA become essential. From mentorship and wellness initiatives to advocacy efforts, EMRA connects us, supports us, and gives us meaningful representation. When I've felt overwhelmed, my involvement with EMRA has rekindled my sense of purpose. EMRA reminds me I'm part of something bigger than myself — a movement to not just survive training, but to ensure EM remains the best specialty.

To effectively address burnout, we must address the systemic challenges causing it. This requires a unified voice and collective action. EMRA is perfectly positioned to lead that charge. Through EMRA, we have the power to advocate for what we need, not just as individuals, but as a community. We can push for policies that protect trainees, engage with leadership to improve working conditions, and create a future where EM thrives.

Here's where you come in. Do you have solutions for combating burnout, improving wellness, or advocating for trainees? Enact those ideas through EMRA. Working together, we can turn frustration into action and dreams into tangible change.

As trainees, it's easy to feel powerless. But I promise you, we are far from powerless when we stand together. United, we can create a specialty where the passion that brought us here is matched by a system that supports us.

Emergency medicine is more than a career; it's a calling. But for us to answer that call — in training and for decades to come — we must care for ourselves and each other. We must invest in our own well-being as fiercely as we invest in our patients. And we must unite to build a future where EM is sustainable, fulfilling, and resilient.

So, I ask you: What do you need? What changes do you want to see? Tell us. EMRA is here, ready to act. Let's be the generation who transforms emergency medicine.

EMRA isn't just an organization — it's *your* organization.

Your voice, your experiences, and your ideas matter. Have you ever thought, "There has to be a better way"? Bring that idea to EMRA. We are far from powerless when we stand together.

References available online.

Emergency Department Approach to LVAD Patients

Anam Chaudhry, DO
PGY3, Emergency Medicine
ChristianaCare

Jeremy Berberian, MD
Associate Director of Emergency Medicine Resident Education, Department of Emergency Medicine
ChristianaCare

Nisha Gandhi, MD
Advanced Heart Failure and Transplant Cardiology
ChristianaCare

Nearly 6.7 million Americans over the age of 20 have been diagnosed with heart failure.[1] It is estimated that more than 2,500 people with heart failure receive a Left Ventricular Assist Device (LVAD) in the United States.[2] Given the increasing prevalence, it is imperative that emergency medicine physicians know how to adequately triage and treat LVAD patients. This article will cover the basics of LVADs and provide a framework to ensure effective treatment of patients with a LVAD.

INDICATIONS FOR LVAD

Not every heart failure patient may qualify for a LVAD. As there can be a long wait on the heart transplant list, LVAD technology has been created and reformed to provide patients with durable mechanical support that enables out of hospital care while they wait for a new heart.[3] Some patients who don't qualify for transplant live with their LVADs as "destination therapy."

LVAD COMPONENTS

There are three types of LVADs with which an EM physician should be familiar: the HeartMate II (Thoratec/St. Jude/Abbott), HVAD (HeartWare/Medtronic), and HeartMate 3 (Abbott). Each of these devices function similarly and have similar components that one should be able to understand and manage.

There are four main components of the LVAD: the pump, the driveline, system controller, and power supply. The inflow cannula allows blood into the pump and the outflow allows blood to bypass the LV and enter the aorta to distribute blood to the rest of the body.

PUMP

The job of the LVAD pump is fairly intuitive: it bypasses the intrinsic contractility of the LV via the inflow cannula and the motor propels blood into the aorta via the outflow cannula.

DRIVELINE

This is only part of the LVAD that is outside of the patient's body (aside from the battery). The driveline connects the pump to the system controller and power supply.

SYSTEM CONTROLLER

The system controller serves as the main display for battery life, alarms, and additional device metrics.

POWER SUPPLY

The power supply is the battery pack for the LVAD. It includes batteries and also a power cord that plugs directly into the wall.

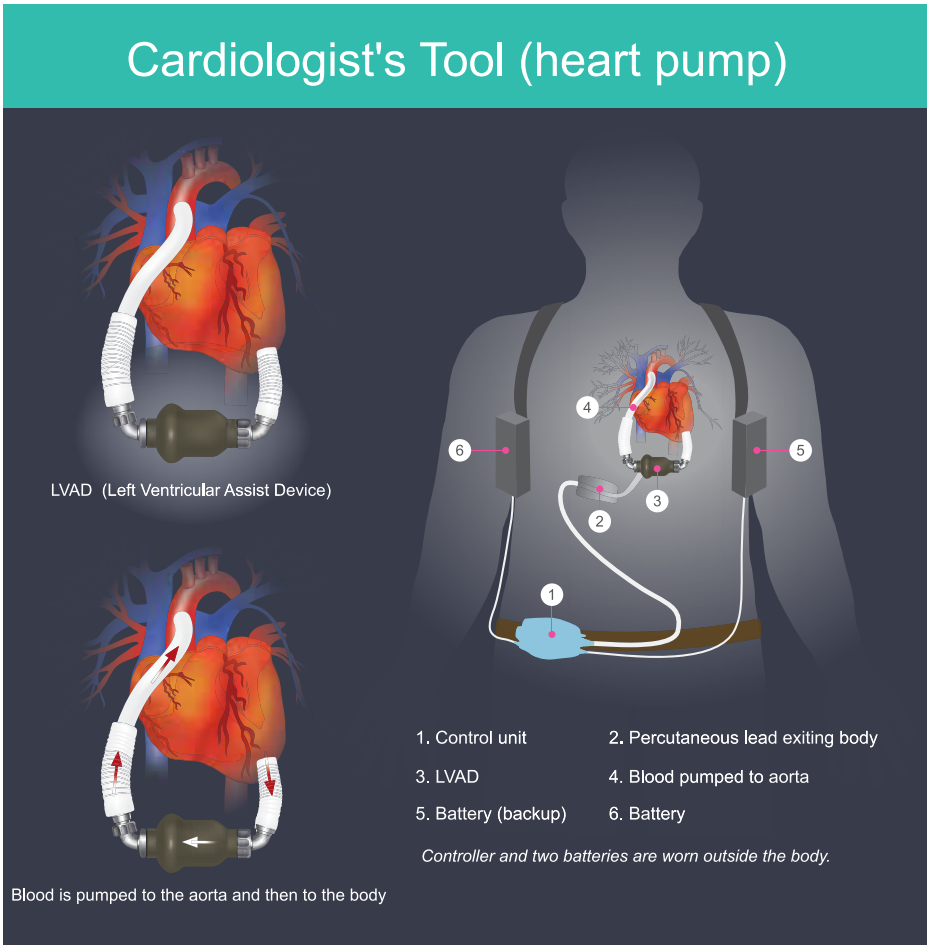


Figure 1: LVAD components [4,5]

References available online.

ED EVALUATION OF PATIENTS WITH LVAD PHYSIOLOGY

C-ABCDES OF LVAD MANAGEMENT

The initial assessment of the LVAD patient is the same as any other in the emergency department with a few caveats. The first call should be to the LVAD coordinator (most patients/families know the direct access phone number to their LVAD coordinator), and this should be done simultaneously while evaluating the classic "ABCDE" of emergency medicine. The rest of the assessment is the same with a few additions that are specific to LVAD management.

Table 1: ED Assessment of LVAD Patient[3]

	What to assess:	What to look for:
C	**C**all the LVAD coordinator	Confirm with patient/family
A	**A**ppearance **A**uscultation	Alert, warm, well-perfused Smooth hum
B	**B**attery **B**lood pressure	Charged and connected to main power source MAP 70-90
C	**C**ontroller	Alarms Cool (not overheated) Device metrics at baseline
D	**D**riveline	Intact, no evidence of damage or infection
E	**E**cho (bedside) **E**KG	Compare with prior echo: baseline ventricular size, tamponade physiology, AV disease and RV size/function

A: APPEARANCE & AUSCULTATION

As soon as LVAD patients are placed in a room, one can perform a quick visual assessment. Specifically, things to look for include making sure they are awake, alert, warm, and well-perfused. During your ED assessment of ABCs, you can check for the typical mechanical whirr of the LVAD device.

B: BATTERY & BLOOD PRESSURE

While the patient is being hooked up to the monitor and you are performing your initial ABCs, it is imperative that the LVAD is connected to a main power source. At the same time, one should quickly obtain a blood pressure. LVAD patients have minimal or zero pulsatility. Thus, a MAP is the most reliable measure of perfusion pressure. Goal MAP should be between 70 and 90 mmHg. LVAD patients are preload-dependent and afterload-sensitive.[6]

C: CONTROLLER

There are several reasons that an LVAD would alarm such as low battery, concern for a disconnection, or any other similar malfunction. The alarms to be concerned about include those regarding changes in pulsatility and flow. See Figure 2 for a broad differential for these variations.

D: DRIVELINE

The driveline should be intact without evidence of infection at the abdominal wall site. An X-ray or CT is also helpful to assess for internal fracture or infection contributing to a patient's presentation.

E: ECHO & EKG

For LVAD patients who are hypotensive with unclear etiology, a bedside echo is helpful to ascertain the primary concern and further hone a differential. The echo should start with the parasternal axis view to assess for over- versus under-distension. If possible, additional important views include a short axis view of the AV and an apical four chamber view. The differential for both situations is similar to that of a non-LVAD patient. Figure 3 provides a differential for LVAD complications based on echo.

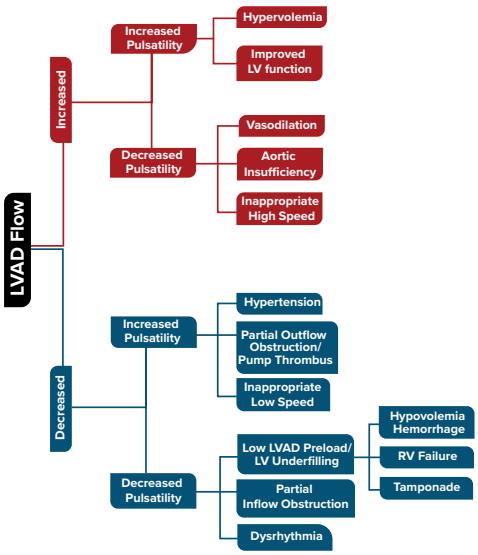


Figure 2: Causes of variable LVAD flow and subsequent pulsatility.[3]

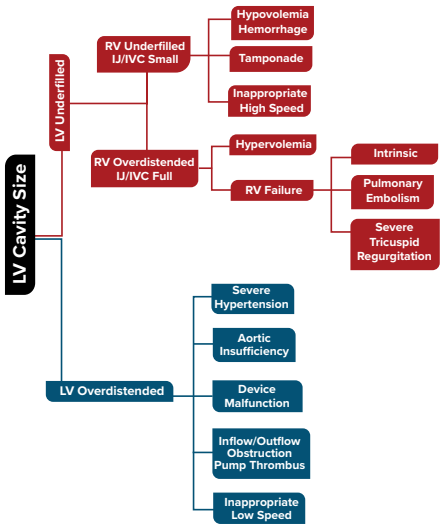


Figure 3: Differential for LVAD complications based on echo findings.[3]

References available online.

Table 2: LVAD Emergencies		
D	**D**riveline complications **D**evice failure	Fracture: assess with XR, consult cardiac surgery Infection: administer antibiotics Device failure: see below
E	**E**mbolism	Anticoagulation
A	**A**rrhythmia	Cardioversion vs. defibrillation, rate control, electrolyte repletion
T	**T**hrombus	Check INR, LDH, echo, consider surgery
H	**H**emorrhage	Anticoagulation reversal
S	"**S**uckdown"[6]	Consider echo, fluids

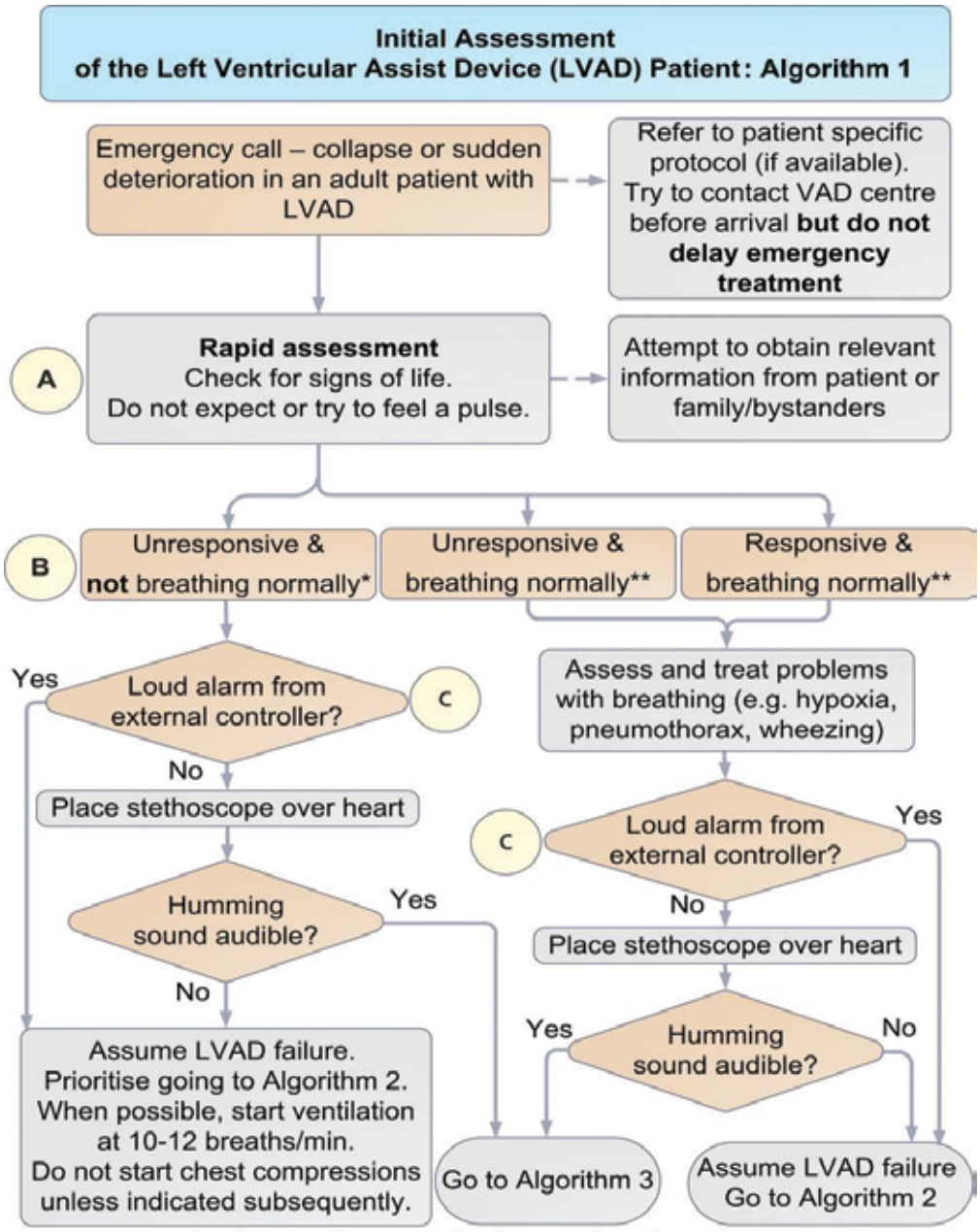
EMERGENT LVAD COMPLICATIONS

Life-threatening complications that every ED provider should be aware of in LVAD patients can be remembered with the helpful mnemonic "DEATHS," which is outlined in Table 2. All of these complications are fairly intuitive and can be managed as one would any other ED patient.

DEVICE FAILURE AND CARDIAC ARREST

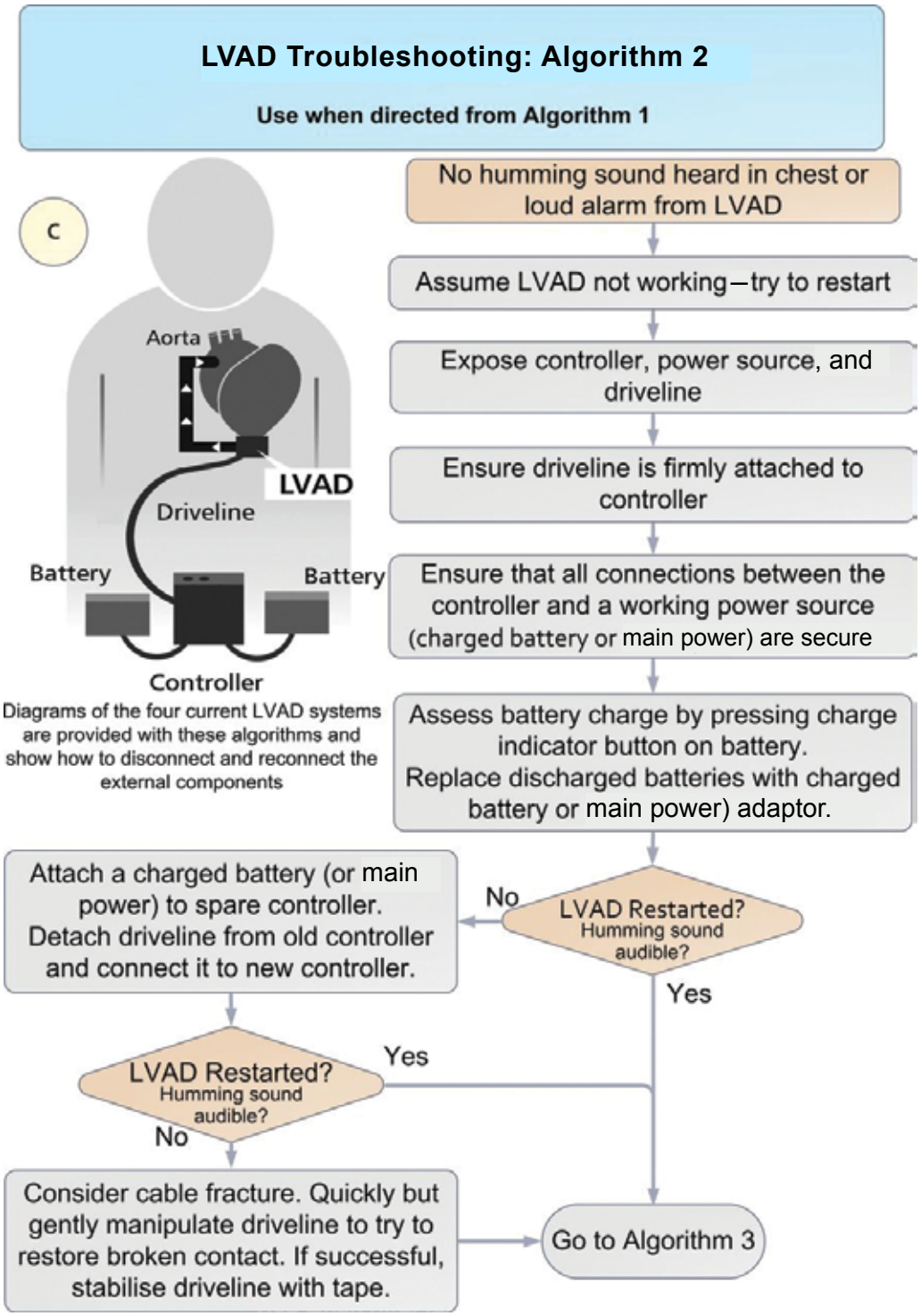
The most important and difficult aspect of LVAD management includes cardiac arrest. Cardiac arrest in an LVAD patient may occur due to device failure, or an alternative cause. As with any other cardiac arrest, compressions should be started immediately, and the LVAD team should be notified emergently as well. The ED team can proceed with standard ACLS protocol while also utilizing an echo to assess for ventricular function.

LVAD failure is the most important etiology of cardiac arrest that the ED physician should know how to manage. There are three reasons for possible LVAD failure: power, the controller, or driveline. One should check to make sure that all the external LVAD components are appropriately connected to a power source and consider manipulating the driveline. Read on for a step-by-step approach to troubleshooting situations.



Algorithm 1: Initial Assessment of LVAD Patient and Troubleshooting LVAD Issues[8]

References available online.



Algorithm 2: Initial Assessment of LVAD Patient and Troubleshooting LVAD Issues[8]

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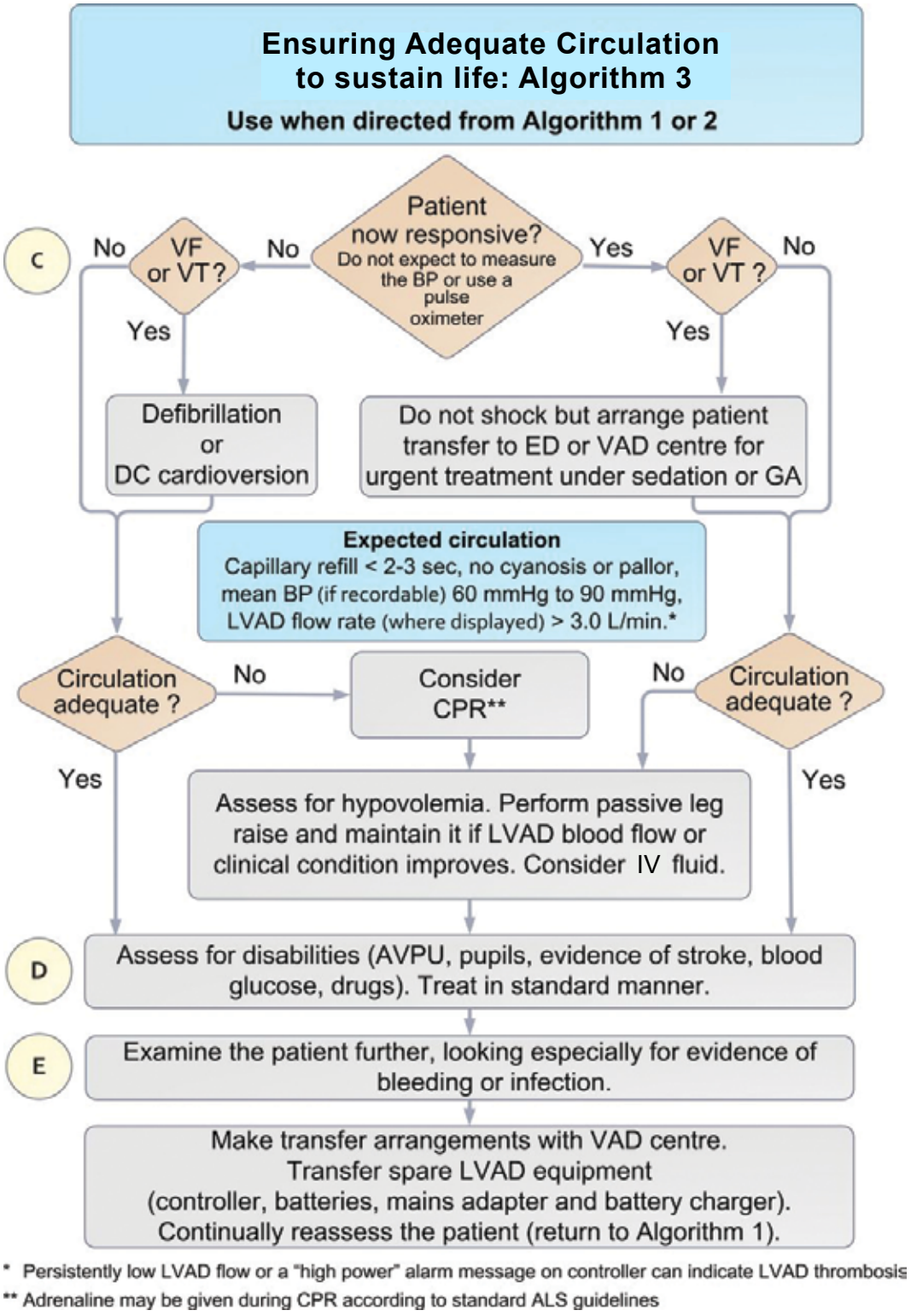

Algorithm 3: Initial Assessment of LVAD Patient and Troubleshooting LVAD Issues[8]

References available online.

Case Report: Heart Block Stemming from Lyme Disease

Puja Singh, MD
Mount Sinai Morningside-West Emergency Department
Morningside-West, New York, USA

Marian Gambrell, MD
Attending Physician
Mount Sinai Morningside-West Emergency Department
Morningside-West, New York, USA

We present a case of bradycardia and dyspnea in an otherwise healthy young male found to have EKG abnormalities inclusive of third-degree heart block.

He was determined to have secondary Lyme disease as his presenting clinical diagnosis and was admitted to the CCU and discharged later with a normal and resolved EKG with normal vital signs.

CASE

A 29-year-old male with a history of childhood asthma presents for 7 days of dyspnea on exertion that is worsened with activity and improved with rest. He went to urgent care earlier and was told to go to the ED for an abnormal EKG. He is an avid cyclist and became worried when his activity was becoming limited. He denies any paroxysmal nocturnal dyspnea, orthopnea, lower extremity edema, any pulmonary embolism risk factors, history of deep venous thrombosis/pulmonary embolism, family history of arrhythmias, coronary artery disease, congestive heart failure, or myocardial infarction.

Additionally, he became worried when 3 days prior, his Apple watch told him his heart rate was between 30-39 bpm. He endorses a sharp pain in the epigastric/midsternal area when he has shortness of breath. He is also endorsing a fever (103 t max) today, and before this had a fever for 3 days with chills, myalgias, arthralgias, and intermittent dizziness when standing up too quickly. He had no sick contacts, cough, rhinorrhea, sore throat, and he tested negative for flu and COVID twice. His EKG was obtained and was noted to be abnormal with third-degree heart block (**Figure 1**). Of note, he traveled to upstate New York for a hiking trip 1 month ago.

Additionally, he notes upon further questioning that 1 month ago, he had a bump on his left arm that was slightly itchy, which he thought was a mosquito bite and subsided with no further rashes. This rash appeared as a small circle. In the ED, the patient was noted to have stable vital signs originally, but then progressed to becoming bradycardic to 33, at which point he was brought to the resuscitation bay.

References available online.

DIAGNOSIS

Acute disseminated Lyme is an uncommon initial presentation of Lyme disease, especially with the initial rhythm showing a third-degree heart block on EKG[1]. We present a case in which the diagnosis was presumed to be disseminated Lyme disease complicated by third-degree heart block based on clinical presentation, history, EKG, and Lyme serology. His Lyme serology were all positive, with a negative troponin, BNP, and lactate. He also had a normal white blood cell count; however, he did have increased inflammatory markers. He was then admitted to the CCU for further monitoring, intravenous antibiotics including ceftriaxone, and was discharged 3 days later with a totally normal EKG (**Figure 2**), resolution of symptoms, decrease in inflammatory markers, cardiology follow-up outpatient, and oral antibiotics.

Lyme disease is the most common tick-borne disease in the U.S. and is caused by *Borrelia burgdorferi*. It is commonly spread by the ixodes tick, which also causes Anaplasmosis and Babesiosis. It is most frequently seen in the upper Midwest, Northeast, and mid-Atlantic states. Lyme has 3 stages, which include early, disseminated, and late.[2]

- **Early phase** occurs days to months after the bite and includes symptoms such as myalgias, arthralgias, fatigue, as well as the classic erythema migrans rash.
- **Acute disseminated phase** occurs weeks to months after the bite, (which is how our patient presented) and includes further dermatologic symptoms, neurologic manifestations such as cranial neuropathy (Bell's palsy), radiculopathy, encephalopathy, and cardiac manifestations such as AV block, with third-degree block being the most common.[2] The pathophysiology of the AV block is not well understood; however, it is believed to be due to transmural inflammation caused to the myocardium.
- **Late stage** includes arthritis, commonly in the knee, and more severe neurologic manifestations such as psychiatric disturbance, encephalopathy, and memory impairment.[3]

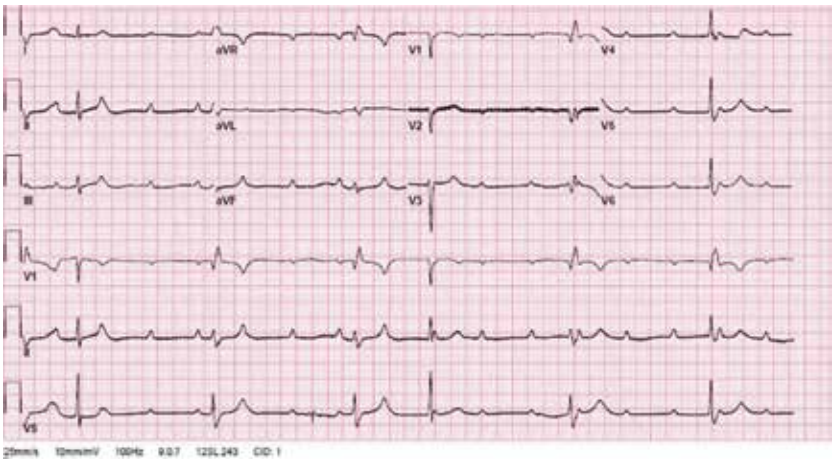

Figure 1. Initial EKG

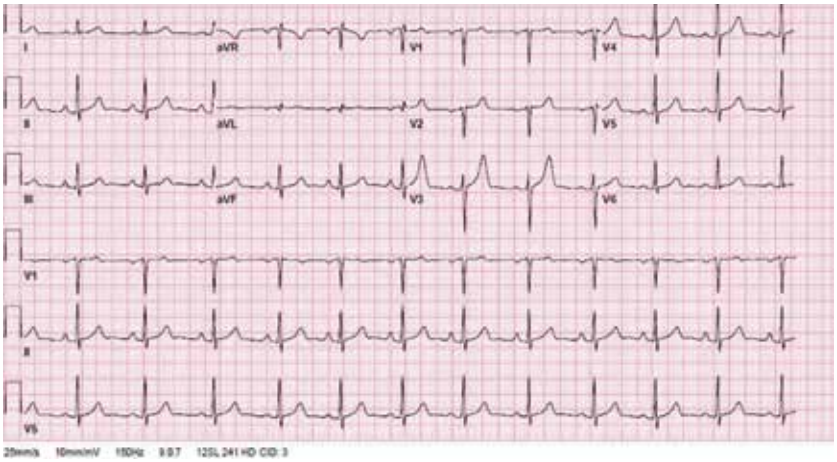

Figure 2. Repeat EKG

There is a good prognosis in the early and acute disseminated stages with treatment, with resolution between 4-42 days.

Patients with cardiac manifestations and associated heart block frequently do not require cardiac pacing, and the third-degree block typically improves within a week. The drug of choice for this phase is ceftriaxone 2g IV, continuing until the AV block resolves and the PR is less than 300, at which point treatment is switched to a 14-21 day course of oral doxycycline or amoxicillin.[1]

TAKE-HOME POINTS

- Overall, our case highlights the importance of considering Lyme disease as an etiology of acute bradycardia with heart block in patients with shortness of breath or chest pain, in all ages, in endemic areas.
- It also demonstrates the rarity of this initial presenting diagnosis in an otherwise young and healthy patient with abnormal vital signs.
- Last, it discusses the importance of recognizing this diagnosis early through a thorough travel history and physical exam to initiate early intervention and prevent unnecessary cardiac pacemaking.

References available online.

Two Sides of a Compression Coin: A Comparison of Wilkie's and Nutcracker Syndrome

Kay Nicole Tipton, DO, MS
UNC Health Southeastern
Campbell University School of Osteopathic Medicine

Neelou Tabatabai-Yazdi, DO
UNC Health Southeastern
Campbell University School of Osteopathic Medicine

Field Davis, DO
UNC Health Southeastern
Campbell University School of Osteopathic Medicine

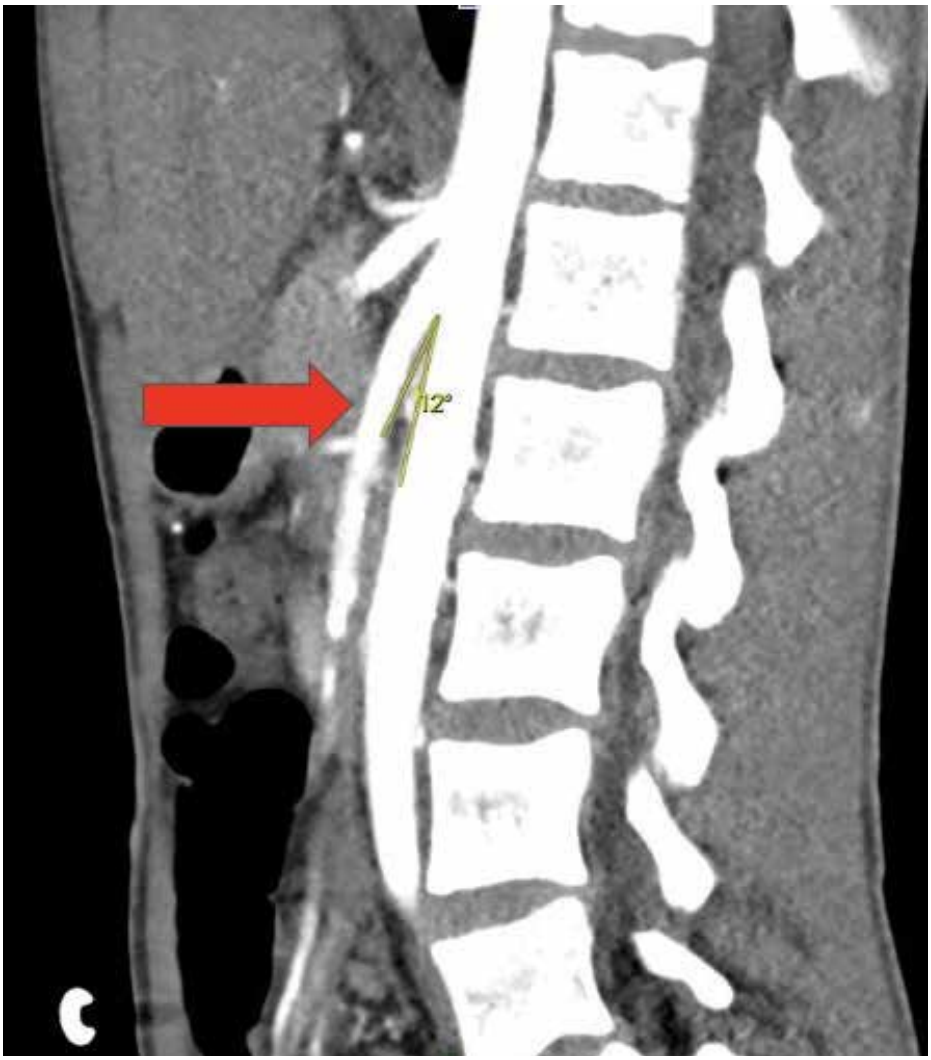

Image 1

The vague presentation of abdominal pain can lead you down many differential pathways. When combined with sudden or rapid weight loss, it can point to 2 rare syndromes caused by compression.

CASE REPORT

A 20-year-old male presents to the emergency department with a multi-month long history of abdominal pain with vomiting that occurs primarily in the morning. He is reporting worsening of this pain over the past week in his left lower quadrant, new radiation of the pain to his left groin, with inability to tolerate solid oral intake. He has had 4 other emergency department visits at our facility over the past 3 months for similar symptoms. Multiple imaging modalities have been utilized with the most recent computed tomography (CT) occurring 6 days prior and showing only thickening of the bladder wall without acute intra-abdominal findings. On this presentation, he is afebrile with normal vital signs. Physical exam reveals moderate left lower quadrant tenderness without rebound or guarding.

Laboratory evaluation was unremarkable; no leukocytosis, electrolyte abnormalities, or evidence of dehydration. Since the patient had already had a CT performed within the past week, we made the decision to obtain a testicular ultrasound to identify pathology which could be missed on CT. The ultrasound revealed a left hydrocele. This finding in combination with the history of abdominal pain, nausea, and vomiting after eating prompted evaluation with a computed tomography angiography (CTA) to exclude compressive pathology. CTA revealed an aortomesenteric distance of 5 mm, aortomesenteric angle of 12° (**Image 1**), and findings consistent with small bowel obstruction.

NUTCRACKER SYNDROME

Nutcracker syndrome refers to the clinical manifestations and symptoms as a result of the compression of the left renal vein, most often between the aorta and the superior mesenteric artery.[1] Entrapment of the left renal

References available online.

vein impairs blood flow into the inferior vena cava, subsequently leading to distention and increasing pressure of the left renal collecting system, including the gonadal vein.[2] Interestingly, not all nutcracker anatomy causes symptoms; such anatomical findings may be a normal variant.[3] In addition to anatomical variation, rare causes of nutcracker syndrome include malignancy, pregnancy, lordosis, intestinal malrotation, and rapid weight loss.[4] There are no set guidelines that dictate what manifestations and symptoms are severe enough to designate the anatomical variant as nutcracker syndrome. Nutcracker syndrome is thought to have a slightly higher prevalence in females and most symptomatic patients present during their second to third decade of life.[1]

The main clinical manifestations of nutcracker syndrome include hematuria, proteinuria, and pelvic pain in females, and varicocele in males. The pain is often described as flank or abdominal pain that radiates to the posterior pelvis and thigh and can be aggravated by movement.[1] Nutcracker syndrome symptoms are thought to be secondary to pelvic and renal congestions and increased pressure.[4] Interestingly, the pediatric population is typically asymptomatic.[5] Long-term complications include hypertension, chronic kidney disease, and left renal vein thrombosis.[6,7]

Diagnosis is multidisciplinary and often requires multiple methods of imaging, including computed tomography imaging, magnetic resonance imaging, Doppler ultrasound, and catheter venography.[2] Successful conservative treatment has been most promising in pediatric patients, likely due to increase and redistribution of adipose and fibrous tissue as they develop.[2] Medications such as angiotensin inhibitors are also used in this population to increase renal blood flow and treat accompanying hypertension.[4] More invasive interventions, including endovascular and extravascular stenting, are commonly pursued in adult populations.[4]

SUPERIOR MESENTERIC ARTERY SYNDROME

Superior mesenteric artery (SMA) syndrome, or Wilkie's syndrome, is an uncommon gastrointestinal pathology in which the transverse portion of the duodenum becomes compressed between the aorta and the SMA. This pathology, described by David Wilkie in detail in a case series publication,[8] most frequently leads to chronic symptoms that can include vague postprandial abdominal pain, weight loss, difficulty tolerating oral intake, and vomiting.[9,10] Despite most cases causing chronic symptoms, there can be acute complications, including small bowel obstruction. Dehydration and metabolic derangements secondary to anorexia and vomiting can also occur.[9]

As mentioned, the syndrome is caused by compression between the SMA and aorta; however, there are many factors that predispose a patient to this compression. Some are congenital, including an abnormal insertion point of the SMA or Ligament of Treitz leading to abnormal positioning of these structures relative to each other.[9] As the duodenum is typically protected from compression by retroperitoneal fatty tissue, rapid loss of this tissue secondary to tumor burden, anorexia, or other conditions can also predispose a person to the development of SMA syndrome.[11]

Diagnosing Wilkie's syndrome can be difficult. An abdominal computed tomography or magnetic resonance imaging test may be helpful, and certain findings are suggestive of the condition.[11] The duodenum may be dilated proximal to the site of compression. A decreased aorto-mesenteric distance or decreased aorta-SMA angle may also be seen. This angle in Wilkie's syndrome is typically between 6° to 16° in comparison to the standard angle between 38° to 65°.[9] Normal aorto-mesenteric distance is between 10 to 24 millimeters. If recent, rapid weight loss is suspected as the cause of the condition, refeeding can replenish the retroperitoneal fat tissue and relieve the condition.[11] However, if conservative treatment fails, surgical intervention may be necessary.[9]

SUMMARY

Although they produce very different symptomatology, both nutcracker and Wilkie's syndromes are caused by compression of structures most commonly between the superior mesenteric artery and aorta. These conditions, although rare, are important to recognize as emergency physicians, as they may present with chronic, recurring symptoms or acutely as the underlying cause of a small bowel obstruction or renal vein thrombosis. Even more rare is the combination of these pathologies, which can lead to increasingly complex sequelae in affected patients.

References available online.

A Case of Isolated Focal Frostbite of the Cheek over Cosmetic Dermal Filler

Sarah Spelsberg, MD, FAWM, FEWM
UNC Health Southeastern Emergency Medicine
Assistant Professor of Orthopedic Surgery, Mayo Clinic

Andrea Sharp, MD
Emergency Medicine, Mayo Clinic

Sara Filmalter, MD, CAQSM, FAWM/DiDMM
Family Medicine, Mayo Clinic Florida

Jessica Pelkowski, DNP, APRN, AG-PCNP-BC, ONP-C
Orthopedic Surgery, Mayo Clinic Florida

Abraham Boxx, BCP, NR-P, FP-C, BSN, FAWM
Former Lifemed, Alaska Medevac

We present a case of a female who sustained presumed frostbite over the right cheekbone a few weeks after undergoing dermal filler cheekbone augmentation injections.

To our knowledge, this is the first reported case of focal frostbite over dermal filler thought to be linked to loss of perfusion capability possibly induced by foreign substance filler cheekbone augmentation injections.

The most common anatomic sites of frostbite are the ears, nose, fingers, and toes.[1,3,8,9] The dermal filler injection elevates the tissue off the bone and occupies space between the tissue and bone.[2] As dermal filler facial augmentation injection becomes more mainstream and this population participates in outdoor cold weather activities, an increased risk of cold injury in thin tissue over dermal filler remains possible. Patients undergoing dermal filler augmentation injections should be vigilant about cold injury when adventuring in extreme cold environments.

CASE

A 48-year-old female sustained a 2.5 cm x 1.5 cm focal area of presumed frostbite over the right cheekbone. The patient had a history of Restylene Lyft™ cheekbone augmentation performed at the area without incident or complication 58 days prior to cold exposure. The patient is a never-smoker who has no other relevant medical or surgical history and takes no medications beyond over the counter supplements.

The patient had planned a 4-hour backcountry excursion in Wyoming, including snowmobiling, snowshoeing, and hiking. The temperature was -10°F at the start of the trip and +16°F at the completion of the excursion. Winds were variable and not sustained. The patient wore a full-face, neck and head balaclava, goggles, and a hat during the outing, as well as a full-face covering helmet during snowmobiling at up to 45 mph. The patient reported feeling generally cold but otherwise noted no focal pain, numbness, or other issues to the nose, ears, fingers, or toes.

Shortly after completing the trip, the patient noticed a 2.5 cm x 1.5 cm area of non-blanching white surrounded by erythema on the right cheekbone that felt firm to palpation. The patient reported the area to be tender "like a bruise." The patient kept the area from any further freezing cold exposure. The white lesion to the cheek was presumed to be frostbite, and the face was rewarmed in a warm shower. The white lesion turned purple and was still pliable to touch within 1 hour (**Image 1**).

There was no bleeding or blistering, but the area began to scab and formed eschar within 12 hours. There was a darker, necrotic-appearing eschar within 24 hours. No bone scan or surgical debridement was felt necessary, and topical wound care was initiated.[4] The patient took ibuprofen and a baby aspirin, but thrombolytics were not deemed necessary due to the focal nature of the injury and low likelihood of significant morbidity.[1] The palpable defect in the musculature under the necrotic area became more pronounced over time and was most noticeable at 3 weeks, after which it started to resolve.

Eschar fell off during the fourth week, revealing tender, friable, erythematous skin underneath and the palpable defect slowly resolving (**Image 2**). The patient was advised to aggressively avoid cold exposure and keep the area out of the sun. After 5 weeks, the area remained mildly erythematous with a residual palpable defect underneath but was deemed to be improving and was expected to continue to improve. Six months after the cold injury,

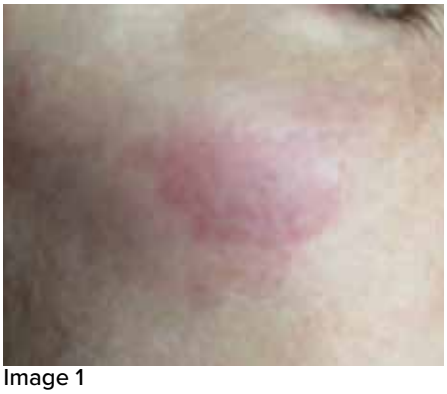
Image 1

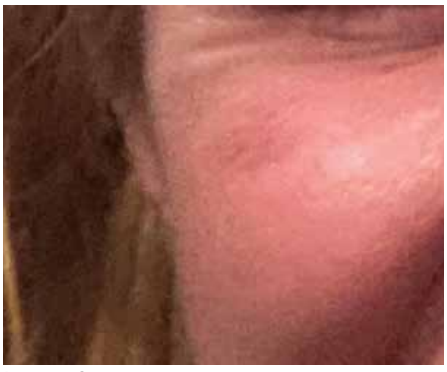
Image 2

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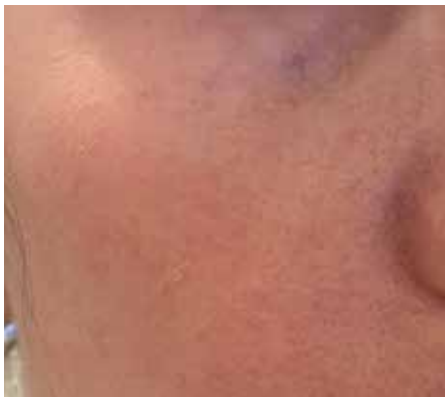

Image 3

the skin was nearly normal with some mild residual erythema (**Image 3**). To our knowledge, a case of frostbite specifically over an area of injected dermal filler, sparing the nose and ears, has not been described in the literature.

DISCUSSION

Immediate or short-term injection necrosis after dermal filler is reported in the literature as a rare but serious side effect that occurs immediately up to several hours after the injection.[2] It is usually attributed to direct injection into a vessel or vessel compression due to filler proximity and compression.[2] To our knowledge, tissue necrosis 7 weeks after filler has never been reported in the literature.[2]

In a Finnish study, the most common site of the face and head for frostbite was the ear (58%), followed by the nose (22%), and the cheeks and other parts of the face (20%).[9] This is a case of isolated frostbite of the cheek over an area of relatively recent dermal filler when the ears, nose, and the rest of the face had equivocal cold exposure but were spared frostbite injury.

Wilderness Medical Society frostbite guidelines note frostbite can be divided into 4 phases: pre-freeze, freeze-thaw, vascular stasis, and late ischemic.[1]

- **Pre-freeze phase** includes cooling, vasoconstriction, and ischemia without ice crystal formation.[1]
- **Freeze-thaw phase** includes the formation of ice crystals inside the cells during rapid freezing and outside the cells during a slow freeze.[1]
- **Freezing** leads to cellular dehydration, cell membrane lysis, and eventually cell death.[1] It can cause local abnormalities in proteins and lipids, as well as shifts in the electrolyte balance of the cell.[1]
- **Thawing** leads to reperfusion injury and inflammation.

During vascular stasis, there is alternating constriction and dilation of vessels, leakage from vessels, and coagulopathy in small vessels.[1] There is a multifactorial inflammatory response modulated by thromboxane A2, bradykinins, and histamine during the late ischemic phase.[1] Ice crystals in and around the tissue cause the initial damage, and disruption to the microcirculation causes cell death.[1] Cell damage caused by the ice crystals is made worse by thawing and refreezing.[1] The most common anatomic sites of frostbite are the ears, nose, fingers, and toes.[1,3,8,9] An isolated focal frostbite injury on the cheek that spares the nose and ears is uncommon.

MANAGEMENT

Topical emollients were used in this case initially and as part of ongoing wound care. They are often recommended in wound care to maintain elasticity and to keep the skin moist. While helpful in wound healing, in several studies, topical emollients were not deemed to be significantly protective in *preventing* initial or further cold injury.[5,6,7]

CONCLUSION

While there are other classification systems described in the literature,[3] our teams utilize the WMS Classification Description. Based on the WMS description of frostbite classification, this event may have been a focal fourth-degree frostbite injury due to the depth of the palpable tissue defect to the bone. The palpable muscular and tissue defect at 3 weeks was due to the dermal filler under healing but friable skin after the eschar fell off. We believe this apparent focal cold injury could have been augmented by the injected dermal filler elevating the soft tissue off the bone.

TAKE-HOME POINTS

- Frostbite is a preventable condition that occurs when local tissue heat loss exceeds the ability of body perfusion to warm the tissue.[1] To our knowledge, this is the first reported case of frostbite likely linked to loss of perfusion capability possibly induced by foreign substance filler cheekbone augmentation.
- The dermal filler elevates the tissue off the bone and occupies space between the tissue and bone.[2] As dermal filler facial augmentation becomes more mainstream and this population participates in outdoor cold weather activities, an increased risk of cold injury in thin tissue over dermal filler remains possible.
- Patients undergoing dermal filler augmentation should be vigilant about cold injury when adventuring in extreme cold environments.

References available online.

Bilateral Thalamic Infarct: A Diagnosis Not to Sleep On

Benjamin Kartub, MD
Resident Physician, Emergency Medicine
University of Tennessee Health Science Center

Abigail Cosgrove, MD
Assistant Professor,
Department of Emergency Medicine
Emergency Medicine Residency Assistant Program Director
University of Tennessee Health Science Center

Khalid Alsherbini, MD, FNCS
Associate Professor of Neurology and Neurosurgery
University of Tennessee Health Science Center
Director of EEG Department
Neurocritical Care Fellowship Program Director
Methodist University Hospital

Occlusion of the Artery of Percheron leading to isolated bilateral thalamic infarcts is a rare and difficult diagnosis in emergency medicine. Learn the symptoms that can help keep it top of mind.

CASE

A 34-year-old male presents to the emergency department with the complaint of syncope. His only relevant known medical history is hypertension, and family states that about an hour prior to arrival, he was standing on his porch when he had a syncopal episode. His vital signs are normal except for slightly elevated blood pressure, and his exam reveals no signs of trauma. His neurologic exam demonstrates bilateral ptosis and a decreased level of arousal, though the family notes he had several beers prior to passing out. You figure he may just be intoxicated and sleepy, so you plan for typical syncope-plus-head trauma orders while you let him metabolize his beers. At this time, his family member mentions that he has had a mechanical heart valve since childhood for an unknown reason and does not take his warfarin.

His presentation of acute encephalopathy, now in the setting of a significant risk factor, prompts you to consult neurology for concern of possible stroke. The CT head and angiogram are unremarkable, but an MRI is subsequently obtained and shows acute, isolated infarcts of the bilateral thalami. The neurologists question you on how this patient's presentation correlates to the imaging findings.

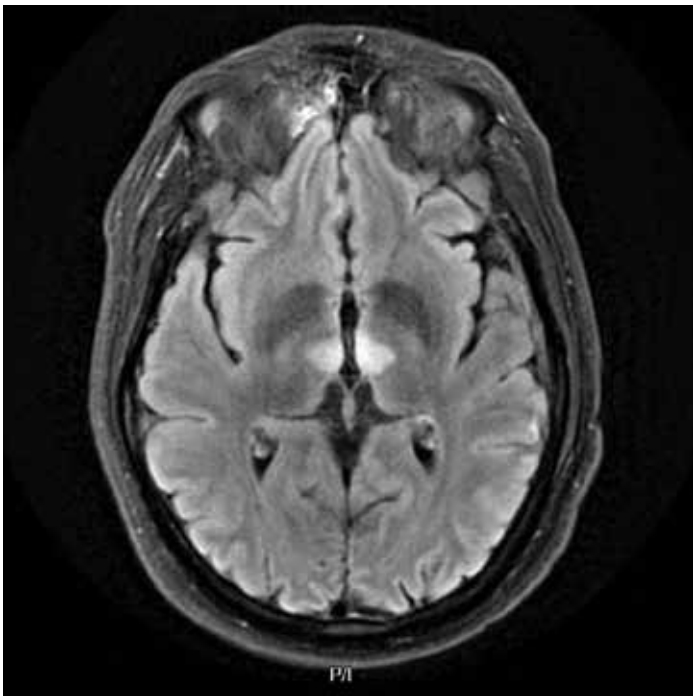

Figure 1. Imaging reveals acute, isolated infarcts of the bilateral thalami

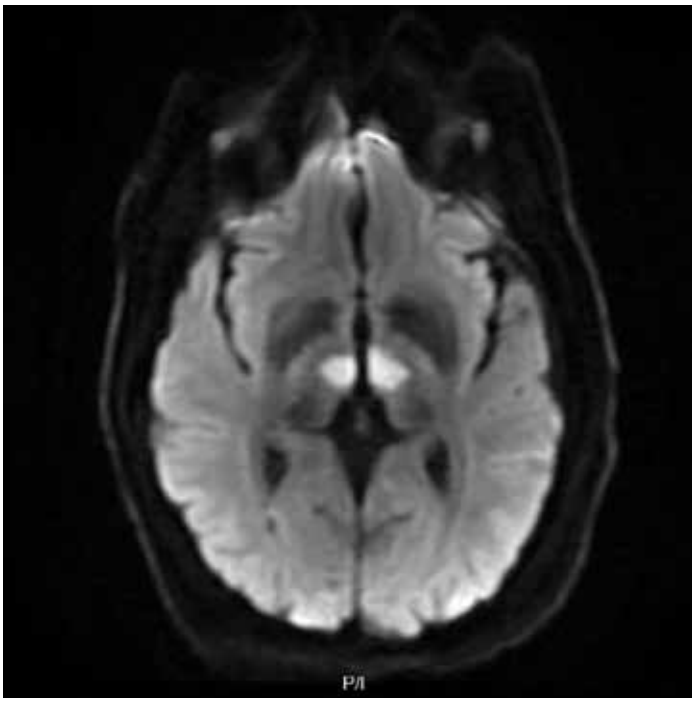

Figure 2. Imaging reveals acute, isolated infarcts of the bilateral thalami

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DIFFERENTIAL

While the differential for acute encephalopathy is broad, the differential for sudden-onset depressed mental status is conveniently narrower. Common causes include: traumatic brain injury; neurovascular pathology such as thromboembolic events and hemorrhage; seizure, particularly nonconvulsive status epilepticus, or a postictal state; intoxication from alcohol, opiates, benzodiazepines, etc.; and hypoglycemia.[1] This is in contrast to a slowly developing encephalopathy that would be expected with etiologies such as infection, certain electrolyte disturbances such as hypercalcemia, or endocrine pathology,[2] where the likely clinical course would be several days of symptoms leading to a progressively-developing encephalopathy that finally prompts presentation to the emergency department. Few of these would be likely in this patient's case. His immediate presentation rules out anything more indolent, and his history, exam, vitals, and CT make an acute ischemic event one of the more likely etiologies.

Regarding the findings on the patient's MRI, bilateral thalamic infarction has a limited differential. It would, logically enough, need to occur in the setting of a symmetric process such that the typical unilateral thromboembolic infarct that we are taught to think of with a subcortical lesion is not enough to explain the bilaterality. A basilar artery infarct or cerebral venous sinus thrombosis could theoretically cause these findings, though these are much more likely to be asymmetric and affect additional vascular territories. Non-vascular lesions of the bilateral thalami include Wernicke's encephalopathy, Wilson's disease, Creutzfeldt-Jakob disease, osmotic myelinolysis, and bilateral malignancies,[3,8] though these all have their own history and associated findings that make them unlikely in this case and, again, would not occur as rapidly as in this patient. One relatively unknown pathology that could be responsible for this patient's acute encephalopathy and his surprising MRI finding is infarction via the Artery of Percheron.

ARTERY OF PERCHERON

The Artery of Percheron is a rare anatomical variant of vascular supply to the thalami. Normal vascular supply is through the posterior cerebral artery (PCA) just as it branches off from the basilar artery, where both PCAs send off perforating arteries that supply their respective thalamus.[4,5] The Artery of Percheron is a variant where the perforating arteries to both thalami (the paramedian segment specifically) arise from a single arterial trunk that comes off only one PCA[6,7] and are in the same vascular territory. This anatomy explains how a single embolic event can infarct such specific bilateral structures without involving broader vascular territories as would be expected in a larger basilar artery infarct.

THALAMUS

The thalamus is a gray matter structure above the midbrain and beneath the lateral ventricles. It is composed of many different nuclei, and the various nuclei serve as the relay centers for sensory information coming back to the brain or for motor information going out to the body. It is also part of the reticular activating system that regulates consciousness and wakefulness, and it plays a role in cognition and behavior. Thus, infarcts to the thalamus can present with a wide range of deficits depending on an individual's vascular supply and the specific nuclei involved. The paramedian thalamic infarcts from Artery of Percheron occlusion are likely to display deficits in oculomotor function, language, and consciousness.[8] This patient, with his low level of arousal and bilateral ptosis, had neurologic findings consistent with thalamic infarct.

CLINICAL PEARLS

Occlusion of the Artery of Percheron leading to isolated bilateral thalamic infarcts is a rare event. Its presence is mainly isolated to case reports in the neurology literature, and no emergency medicine resource appears to make mention of its existence. This is a rare and difficult diagnosis that typically requires MRI for definitive diagnosis. Vigilance is needed to identify the potential of this stroke pattern in patients with altered mental status and few other findings. The prompt recognition of this rare etiology of stroke in a patient with unresolving encephalopathy led to early enough diagnosis for therapeutic intervention.

CASE RESOLUTION

The patient had an unremarkable hospital course. He was given tenecteplase due to his presentation within the thrombolytic window and was appropriately anticoagulated for his mechanical valve. Over the subsequent days his level of alertness improved with modafinil, though at the time of his discharge he still had some degree of ptosis and required frequent verbal or physical stimuli to remain fully alert and awake.

Notably, the prognosis for thalamic stroke is considered favorable, though recovery from deficits in level of arousal and behavioral changes have been reported to be worse compared to motor or sensory deficits.[3]

References available online.

When a Headache Is Not Just a Headache

Moshe Bengio, DO
Aventura Hospital and Medical Center

Glenn Goodwin, DO
Aventura Hospital and Medical Center
EM Residency Graduate

Abhishek Roka, MD
Medical Clerkship Director
Aventura Emergency Medicine Residency

Michelle Marin, MD
Attending Physician, Pediatric Emergency Medicine

Acute, persistent headache in a pediatric patient with no recent trauma can have a broad differential and, in most cases, should raise red flags. This case demonstrates the evaluation and management of cerebral abscess in a child.

CASE

A 12-year-old male presented to the pediatric emergency department for intractable and worsening left frontoparietal headache during the past 6 days, unresponsive to ibuprofen. The patient also reported neck stiffness, vomiting, and photophobia. Of note, he had extensive medical history, including surgical pulmonary valve repair shortly after birth.

The patient's vital signs were within normal limits. Neurologic exam and visual acuity were unremarkable. There was neck stiffness and a positive Jolt test, but Kernig's and Brudzinski's were negative. A comprehensive laboratory workup yielded normal values. His headache was partially relieved with ketorolac and metoclopramide; however, given his medical history and duration of symptoms, a CT brain was ordered.

The non-contrast brain CT revealed bilateral ambiguous defects (**Figure 1A**), necessitating MRI and pediatric neurosurgical consultation. The MRI brain with contrast demonstrated bilateral ring enhancing lesions and a third tiny focus. See **Figure 1 (B and C**). These lesions were consistent with abscess formation, but malignancy could not definitively be ruled out.

CEREBRAL ABSCESS OVERVIEW

Signs and Symptoms

The classic triad of a cerebral abscess is unilateral headache, fever, and focal neurologic deficits.[1] Similar to the above patient, cerebral abscesses may present

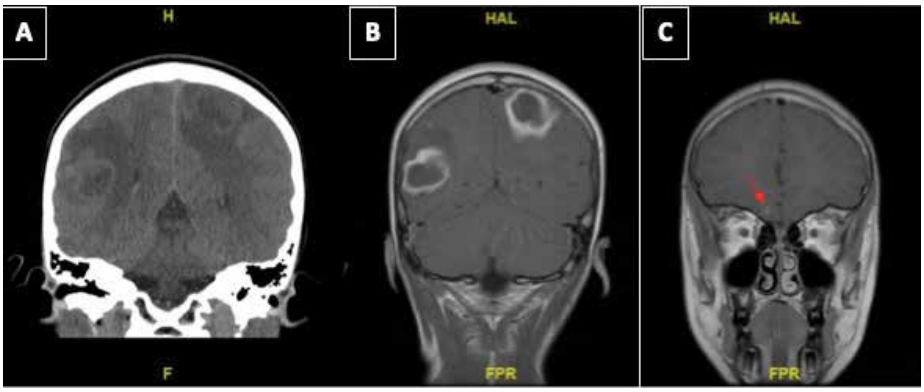


Figure 1.
A. *CT imaging with right and left hemisphere hypodense lesions with surrounding vasogenic edema.* ***B.*** *T1 weighted MRI brain with contrast demonstrating peripheral with thick rim of enhancement of the lesions with low T1 signal and vasogenic edema. There is also questionable leptomeningeal enhancement.* ***C.*** *T1 weighted MRI brain with contrast demonstrating a gyrus rectus lesion.*

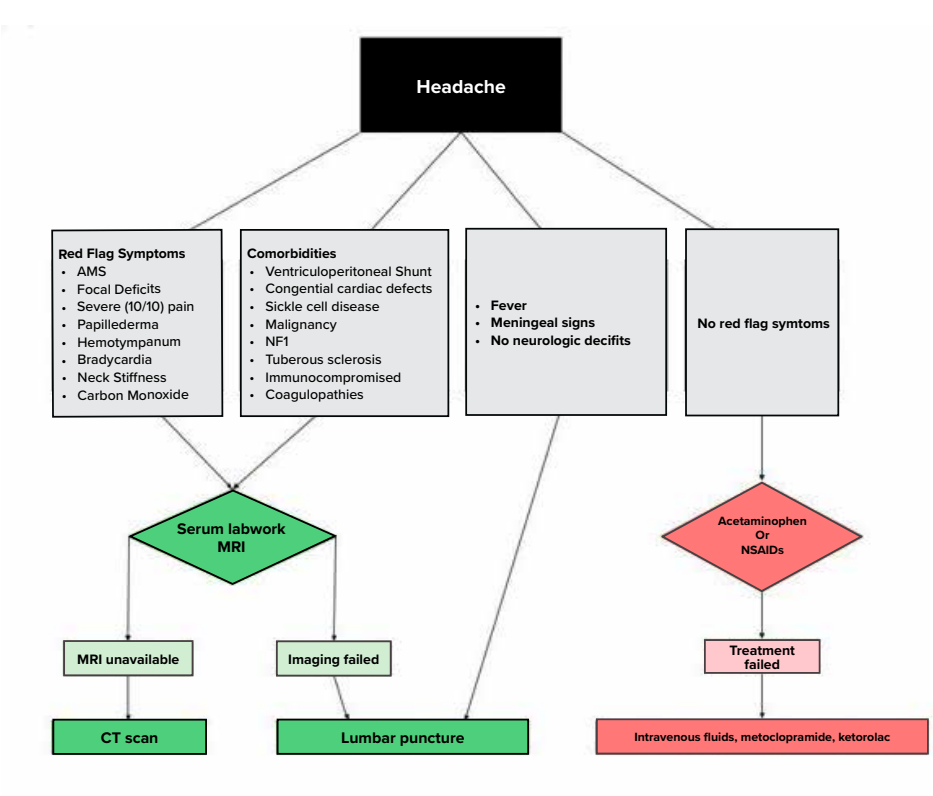


Figure 2. Differential flowchart for pediatric headache

References available online.

TABLE 1. Predisposing factors, causative agents, and management of cerebral abscess, respectively. RIPE = Rifampin, Isoniazid, Pyrazinamide, Ethambutol. TMP-SMX = Trimethoprim Sulfamethoxazole

PREDISPOSING FACTORS (Column A)	CAUSATIVE AGENT (Column B)	EMPIRIC ANTIBIOTICS (Column C)
Neonate	*Proteus* *Enterobacter* *Citrobacter*	Cefotaxime & Ampicillin
Immunocompromised	*Fungi* *M. tuberculosis* *Nocardia*	Ceftazidime, Vancomycin & Metronidazole +/- Amphotericin (fungal), RIPE (tb) TMP-SMX (Nocardia) therapy
Congenital heart disease	*S. viridans* *Microaerophilic streptococci* *Haemophilus*	Ceftriaxone & Metronidazole +/- Vancomycin
Middle ear infection	*Streptococci* *Enterobacteriaceae* *Pseudomonas*	Ceftriaxone & Metronidazole
Sinus infection	*Streptococci* *S. aureus* *Enterobacteriaceae*	Ceftriaxone & Metronidazole +/- Vancomycin
Oral cavity infection	*Mixed anaerobic flora* *Streptococci* *S. aureus* *Enterobacteriaceae*	Penicillin + Metronidazole or Ampicillin-sulbactam
Post-traumatic	*S. aureus* *Streptococci* *Enterobacteriaceae*	Ceftriaxone & Vancomycin

without the complete triad. Other clinical symptoms typically include papilledema, vomiting, and neck stiffness, as well as focal neurologic changes, fever, and seizures.[1] All of these findings are headache red flags and should receive an expanded workup to include imaging.[2] A brief pediatric headache algorithm is provided in **Figure 2**.

Pathology

Cerebral abscesses begin with an initial focal cerebritis without pus or capsule formation (similar to our patient's third focus in **Figure 1C**).[3] These focal lesions worsen over 10-14 days to form an encapsulated suppurative lesion, now referred to as a cerebral abscess.[3]

The most common organisms include *S. pneumoniae* and *viridans*, followed by *S. aureus, S. epidermidis*, and *group B streptococcus (*GBS).[4] However, one should consider predisposing factors for the most likely respective causative organism (**Table 1**).[1] Others not included in the table are neurocysticercosis from *Taenia solium* and other parasites such as *Paragonimus*, *Schistosoma japonicum,* and *Entamoeba histolytica*.[1, 5-7] These organisms typically aren't considered unless the patient has been to the respective endemic areas.

Risk Factors

Direct inoculation is high risk for an intraparenchymal abscess and can include penetrating trauma or nearby infection. See **Table 1**, column A.[1] Another risk factor is hematogenous spread, which will likely present with multiple foci.[1] The likely organism is based on the age and health status of the patient, in addition to the source of infection, succinctly summarized in **Table 1**.

Diagnosis

CT scans have a relatively low sensitivity for cerebral abscess and may not pick up early cerebritis.[8] Nonetheless, CT is still generally the reasonable, initial imaging modality in the ED due to ease of access. MRI is the diagnostic modality of choice, however, based on its impressive diagnostic value: "The sensitivity, specificity, positive predictive value, negative predictive value, and diagnostic accuracy of echo-planar diffusion-weighted MRI in the diagnosis of intracerebral abscesses was 85.64%, 95.88%, 93.82%, 90.14%, and 91.56%, respectively."[9]

A lumbar puncture (LP) is not recommended with space occupying lesions due to a theoretical risk of tonsillar herniation and adds little diagnostic value.[10-12] Despite the classical teaching, not all ring-enhancing lesions are abscesses. The differential diagnoses for ring-enhancing lesions is broad and memorable with the MAGICAL DR mnemonic (**Table 2**).[13]

TABLE 2. Differential diagnosis for ring enhancing lesions

M	Metastasis
A	Abscess
G	Glioblastoma
I	Infarct or inflammatory (tuberculoma, neurocysticercosis, aspergillosis)
C	Contusion
A	AIDS (toxoplasmosis or cryptococcus)
L	Lymphoma (immunocompromised patient)
D	Demyelination
R	Radiation necrosis

Pharmacological Treatment

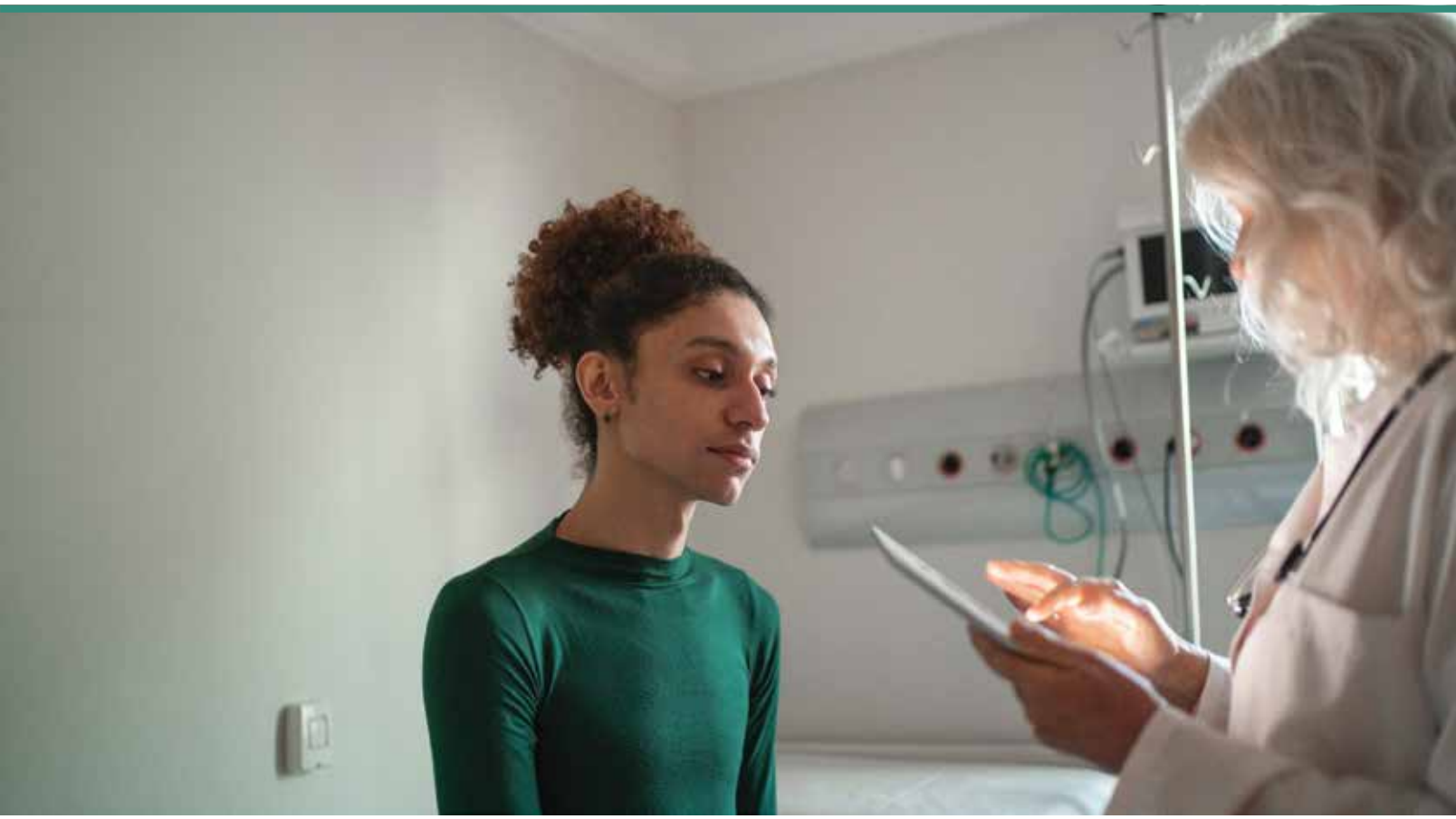
Consider supportive pain medications like acetaminophen or NSAIDs while pending workup results. In patients unresponsive to these medications, add a combination of intravenous fluids (20 mL/kg, ketorolac (0.5 mg/kg, max 15 mg), metoclopramide (0.2 mg/kg, max 10 mg), and, if needed, magnesium or steroids.[14-16] Appropriate antibiotic regimens are described in **Table 1**, column C. The lesions will often require neurosurgical intervention.

CASE CONCLUSION

Given the broad differential diagnosis of ring enhancing lesions and the lack of classic findings for cerebral abscess, including neuro deficits on physical exam, antibiotics were held until after the lesions were confirmed, intraoperatively, to be abscesses.

The patient received levetiracetam while awaiting neurosurgery. The abscesses were eventually drained, and he made a full recovery without neurologic compromise. The patient was referred to cardiology on discharge to investigate the pulmonary valve repair, as this was theorized to be the source of the septic emboli, but the patient was lost to follow-up.

References available online.



Bit-by-bit: Decoding Nonbinary Patients in the Emergency Department

Lachlan Driver, MD
Harvard Affiliated Emergency Medicine Residency
Department of Emergency Medicine
Mass General Brigham

Alyson J. McGregor, MD, MA, FACEP
Associate Dean of Clinical Faculty Affairs and Development
Professor Department of Emergency Medicine
University of South Carolina School of Medicine Greenville
Prisma Health

Though there has been some research on lesbian, bisexual, and gay health in the care of emergency department patients, as well the beginning of literature on transgender/gender non-conforming (TG/GNC) as we begin to understand the roles of sex and gender on population health, little to no studies have been devoted to addressing the specific health care needs of the growing nonbinary (NB) patient population. Thus, it is likely one of the least studied LGBTQIA+ subgroups, along with having worse health outcomes than their cisgender binary and sometimes transgender binary counterparts.[1]

Scandurra et al. found in their 2019 systematic review of 11 studies that health disparities and outcomes in nonbinary and binary transgender were mixed, although overall consistently worse than cisgender individuals.[1] Of note, the ongoing PRIDE Study is the first long-term, large-scale health study of individuals in the U.S. who are LGBTQIA+, that is trying to tease out the effects of being LGBTQIA+ on people's physical, mental, and social health, and includes collecting information on NB identities and their health.[2]

Overall, there are no significant studies specifically devoted to treating NB patients in the ED and how that may be disparate from approaching binary TG patients. As a specialty, we need to be working to ensure that our patient care, education/training, and hiring practices reflect our values of inclusivity when it comes to nonbinary and gender-fluid individuals, which may be critical steps in the next few years. As part of that, patients should be asked to provide chosen names and their pronouns on forms along with other patient identifiers, such as sex assigned at birth. Thus, a multi-step process should be followed to capture information about sex and gender identity.[3,4]

GROWING IN NUMBERS

An estimate from a February 2021 Gallup Poll places the growing LGBTQIA+ population in the U.S. at approximately 5.6% of the population, or 18.5 million

References available online.

people.[5] Of those, other estimates show that 1.2 to 2 million people (6.5% to 11% of the LGBTQIA+ population) classify themselves as NB, with over 50% being between 18 and 29 years old.[5,6]

Additionally, the Gallup Poll also found that 11.3% of LGBTQIA+ adult respondents self-identify as transgender.[5] This is consistent with other recent estimates that report that 1.4 million or more individuals in the U.S. identify as transgender, accounting for around 0.6-2.7% of the population.[7,8] From the 2015 U.S. Transgender Survey, it can be said that 60% of TG people began to feel that their gender identity was different than what is on their birth certificate before age 10, with only 6% being over 21 years old.[9]

Additionally, the 2015 U.S. Transgender Survey showed that approximately 35% of the 28,000 transgender respondents to the survey self-identified as NB,[9,10] and 56% of NB people begin transitioning between the ages of 18-24.[9] NB individuals are born with bodies that may be phenotypically and genotypically male, female, or intersex (a genetic pattern of sex chromosomes or a response to sex hormones that differ from typical XX, XY physiology), but their innate gender identity is something different than exclusively man or woman. They may lie on the man-woman spectrum and view themselves as both, lie outside the spectrum with a different or third gender identity, or may have no gender identity at all, as is the case with agender individuals. Though frequently cited to be under the TG umbrella and although studies are limited, it has been shown that approximately 43% of NB people identify as TG, meaning they have transitioned from their assigned gender at birth to a NB status — one of neither exclusively male nor female.

This has led to many NB LGBTQIA+ people to not self-identify solely as TG but specifically with a TGNB status.[6,11]

VISIBILITY VS. INVISIBILITY

Certain personal identities are visible, while some are invisible. For instance, one's sexuality could be considered an invisible identity since there is no specific finding that could pinpoint to an individual's sexual or romantic orientation, regardless of whether they are queer/gay/straight/lesbian/bisexual/other. On the other hand, sex, gender, and racial identities can be externally visible, while gender identity with its corresponding expression could be either visible or invisible, particularly for NB individuals, as there is no one idea of what a NB person "should look like," or what sex or gender they were assigned at birth.

Thus, it can be challenging to be "seen" by society and health care providers. For instance, if a patient is assigned female at birth, it is often assumed that their purpose of transitioning, whether it is surgically, hormonally, or socially, is to achieve the status of man; to move from the box from "W" to "M". That may not be the case for many NB individuals. Many health care organizations still request patient gender and sex as entirely binary, often only ask for either sex or gender, and do not offer alternatives for patients to report themselves as having NB gender.[12] Due to NB gender markers being often unavailable in the medical record, patients are frequently at risk for being gendered incorrectly and therefore misunderstood by their medical providers. Additionally, identifying patients in this subgroup can be challenging as NB vernacular is relatively new.

IMPACTS ON HEALTH OUTCOMES

There is often confusion and frequent misunderstandings, both on the part of patient and clinician.[13] In fact, in a study of TG and NB university students, Goldberg et al., found that NB persons reported more misgendering by health providers — in other words, being called by incorrect pronouns, or providers making incorrect assumptions about their patients having certain hormone status or organs. Additionally, NB participants were 76% more likely than binary TG participants to be "misgendered sometimes or often" by health care providers.[1,14] They also receive lower rates of support from family and friends compared to TG counterparts, have higher levels of anxiety and depression compared to binary TG individuals, and have higher rates of smoking and drug use, all of which could impact medical and mental health needs when presenting to the ED.[1,15-18]

A 2018 study by Clark et al. found that NB participants had 62% higher likelihood of non-suicidal self-injury compared to binary TG individuals.[16] They were also more likely to report increased alcohol use than binary TG and smaller odds of having a family doctor than their binary TG counterpart.[1,16] All of these could contribute to more frequent and more severe health outcomes when these patients eventually present to the ED.

Additionally, interviews of NB patients by Lykens et al. in 2018 showed that NB individuals frequently felt misunderstood as their health care teams were often only familiar with the binary concept of medical care.[19] This is congruent with Goldberg et al., who found that NB students reported increased misgendering and less trans-affirming care by health care providers, compared to binary TG students.[18] NB patients felt pressure to conform to binary medical narratives, self-modified the care they received, or went without medical care altogether.[19] Additionally, 86% of nonbinary respondents to the U.S. Transgender Survey reported that, regarding their gender identity, "people do not understand so they do not try to explain it".[9] Approximately two-thirds reported being NB is often judged as being a phase or not a true identity, and 43% of NB respondents feared violence, while 20% of NB patients have avoided medical care due to fear of being disrespected or mistreated by a health care provider.[9]

On the other hand, there has also been some evidence that binary TG individuals have worse health outcomes related to emergency medicine care compared to NB patients. Though, as above, NB patients tend to present with more non-suicidal self-harm, they are less likely to attempt suicide.[20] NB patients avoid medical care due to fear of being disrespected or mistreated by treatment team at lower rates compared to binary TG men, 20% vs 31%, respectively.[9] Additionally, HIV status appears to vary greatly by gender identity, with trans men and NB people having rates of 0.3-0.4%, whereas approximately 3.4% of trans women report being HIV positive.[9]

An interesting 2016 study of over 3,000 LGBTQIA+ participants by Smalley et al. showed that binary TG and nonbinary individuals have very different

References available online.

risk-behaviors, with NB having lower risk of sexual risk-taking, substance use, and medical risk-taking. Additionally, the same study showed that while many of the subcategories under LGBTQIA+ are typically condensed into one category, they show variable risks when examined individually.[21]

ROLE OF EM PHYSICIANS AND DEPARTMENTS

In general, emergency medicine physicians should be aware that not all identities are necessarily visible (e.g., sexuality and NB gender are not always visible to others), but all are valid. Given that EM physicians often treat children, it is important to know that 60% of TG/GNC people began to feel that their gender identity was developing before age 10.[9] Additionally, NB patients may have a different medical narrative than binary TG individuals, they are also at higher risk of self-harm but lower risk of suicide attempts compared to TG binary patients.[16,20] There is also evidence that NB patients report higher risk of excessive alcohol use, as well as smoking and drug use, though some smaller studies disagree.[21] In general, NB patients and trans men tend to have lower rates of being HIV positive compared to trans women; NB patients also have lower rates of having a primary care provider, thus may have higher likelihood of presenting to the ED when unwell.[1,16]

Compared to other gender-expansive patients, NB patients experience increased misgendering and less trans-affirming care by health care providers, which can lead to distrust and lack of following clinical discharge instructions.[1,14] In general, NB patients feel pressure to conform to a binary narrative.[19] Finally, NB patients do undergo hormone and surgical affirmation though at lower rates than TG binary individuals.[8,9]

Emergency medicine departments should make it a policy to consistently ask about gender, pronouns, chosen name, sex assigned at birth on (non-emergent) presentation, hormonal status, and organ inventory. Departments should document these findings in the EMR when possible, as this will allow for further research in this poorly studied population. In general, EDs should encourage health care teams to communicate and use everyone's correct pronouns/chosen name – and be willing to offer your own. This is important: though there are no known studies collecting information about the proportion of NB or LGBTQIA+ residents as a whole, in the Moll et al. paper from 2021, authors showed that the majority of respondents surveyed at residency programs across the country were aware of known LGBTQ+ faculty and residents at their programs.[22]

Additionally, departments should screen for self-harm in the NB population, as well as depression/anxiety, and substance use. They should educate and have designated areas in the curricula for these discussions for all members of the health care team, as well as registration, on how to correctly gather information and affirm patients' identities.

There are many resources when it comes to optimizing health outcomes of TG/GNC individuals; the LGBT Health Education Center at Fenway Institute has developed best practice guides, such as their "Do Ask, Do Tell" toolkit, which advises collecting gender identity, sex assigned at birth, chosen name, and pronouns used for optimal patient care.[23] Additionally, Society for Academic Emergency Medicine published an online guide on teaching and discussing the topic of Sex and Gender Minorities in the ED.[24]

Table 1

INFORMATION	IMPLICATIONS FOR EM PHYSICIANS
Not all identities are necessarily visible (e.g., sexuality and NB gender)	All identities are valid
60% of TG/GNC people began to feel that their gender identity was developing before age 10	This is particularly important in treating the pediatric population
NB patients may have a different medical narrative than binary TG individuals	All patients should be viewed through a unique lens; NB patients do not have the same risk factors as binary TG patients
NB patients are at higher risk of self-harm but lower risk of suicide attempts compared to TG binary patients	Be vigilant about screening for self-harm and its sequela as well as suicidal ideation in these patient populations
Some studies showed NB patients report higher risk of excessive alcohol use, as well as smoking and drug use	Consider substance use disorders in both NB and binary TG patients
NB patients have lower rates of having a primary care provider	NB patients may have higher likelihood of presenting to the ED when unwell. It is important to make sure they get an appointment when they get referred to a PCP
NB patients experience increased misgendering and less trans-affirming care by health care providers	These events can lead to distrust and lack of following clinical discharge instructions, thus it's important to spend the time to explain instructions and return precautions
NB patients undergo hormone and surgical affirmation though at lower rates than TG binary	Consider side effects of hormone therapy (when relevant) in patients' chief complaint, (e.g., VTE in someone taking estrogen)
NB patients feel pressure to conform to a binary narrative	If a patient is NB, ask what NB means for them, what pronouns they use, and what they'd like to be called; don't assume they're binary

References available online.

BACKGROUND

Though there has been some research on lesbian, bisexual, and gay health in the care of emergency department (ED) patients, as well the beginning of literature on transgender/gender non-conforming (TG/GNC) as we begin to understand the roles of sex and gender on population health, little to no studies have been devoted to addressing the specific health care needs of the growing nonbinary (NB) patient population.

METHODS

Authors explore the pertinent literature and the current vernacular pertaining to the LGBTQIA+ community, with a particular focus on nonbinary patients in the ED, contrasted with LGB as well as transgender patients' health outcomes and risk factors. We discuss the educational implications and make actionable suggestions to improve care of gender-expansive individuals in the ED.

RESULTS

Emergency medicine physicians should be aware that not all identities are necessarily visible: Sexuality and NB gender are not always visible to others, but all are valid. NB patients may have a different medical narrative than binary TG individuals. NB patients experience increased misgendering and less trans-affirming care by health care providers and, in general, feel increased pressure to conform to a binary narrative. EDs should consistently ask about gender, pronouns, chosen name, sex assigned at birth, hormonal status, and organ inventory. Departments should document these findings in the EMR when possible, as this will allow for further research in this poorly studied population.

CONCLUSION

There are no significant studies specifically devoted to treating NB patients in the ED and how that may be disparate from approaching binary TG patients. As a specialty, we need to be working to ensure that our patient care, education/training, and hiring practices reflect our values of inclusivity when it comes to nonbinary and gender-fluid individuals.

PERTINENT TERMS DEFINED

Sex: a person's genotypic/phenotypic presentation, typically assigned male, female, or intersex at birth, typically corresponds to XX or XY or other combinations of chromosomes. Typically written as sex assigned at birth (SAAB), assigned female at birth (AFAB), or assigned male at birth (AMAB).

Gender identity: one's deeply held, invisible concept of self in relation to being a woman, man, both, or neither (e.g., agender or third gender) in context of one's society or social environment. This identity may be concordant or discordant with sex assigned at birth (woman — female, man — male if concordant).

Gender expression: the external and more visible manifestations of conveying one's gender identity, expressed through pronouns used, chosen or given name, clothing, hairstyle, behavior, voice, and/or body shape/characteristics, in the context of social gender expectations.

Transgender (TG): an umbrella term describing individuals whose gender differs from expectations associated with their sex and gender assigned at birth.[25]

Cisgender (CG): Those individuals whose internal sense of gender identity corresponds to their sex and gender assignments at birth.

Gender diverse/non-conforming (GNC): term used to signify that one's gender identity or expression differs from cultural expectations, which could include transgender, nonbinary, and sometimes cisgender individuals.

Nonbinary (NB): frequently cited as part of transgender identity (though not always[6]), those individuals do not fully see themselves in traditional man/woman categories. They may lie on the man-woman spectrum and view themselves as both, lie outside the spectrum with a different or third gender identity, or may have no gender identity at all.

Transition: the non-linear, non-obligatory process of TG/GNC individuals taking steps to have their outward presentation more closely aligned with their internal gender identity, which can include social (name/pronoun use), hormonal (estrogen/testosterone therapy), and/or surgical aspects (e.g., top/bottom surgery).

Sexual orientation: "An inherent or immutable enduring [...] sexual attraction to other people",[26] not to be confused with an individual's sexual practices, which may or may not match up with what they consider their sexuality.

Romantic identity: An inherent romantic attraction to other people, which should not be confused with an individual's romantic practices, which may or may not match up with their romantic identity

EMERGENCY MEDICINE DEPARTMENTS SHOULD:

- Consistently ask about gender, pronouns, chosen name, sex assigned at birth on (non-emergent) presentation, hormonal status, and organ inventory
- Document findings in the EMR, as this will allow for further research in this poorly studied population
- Encourage healthcare teams to communicate and use everyone's correct pronouns/chosen name — and be willing to offer your own
- Screen for self-harm in the NB population, as well as depression/anxiety, and substance use
- Educate and have designated areas in the curricula for these discussions for all members of the healthcare team, as well as registration, on how to correctly gather information and affirm patients' identities
- Use toolkits, such as the "Do Ask, Do Tell" by Fenway Health or "Sex and Gender Minorities" by SAEM to educate yourself and others.[23,24]

References available online.

Case Report: A Unique Role for Bedside Ultrasound in a Patient with Progressive Unilateral Leg Swelling

Michaela Banks, MD, MBA
Louisiana State University
Spirit of Charity - New Orleans

George Alexander Knight, MD
Internal Medicine
Tulane University School of Medicine

Heather Murphy-Lavoie, MD
Clinical Professor of Emergency Medicine
LSU School of Medicine

This case of unexplained, worsening pain and swelling in a bounceback patient was solved with bedside ultrasound and a thorough history that revealed social determinant red flags.

A 38-year-old male presented to the emergency department with 2 weeks of right lower extremity swelling. He stated that he went to an outside emergency department twice the week prior, where two deep vein thrombosis (DVT) ultrasound studies were negative. He presented to our emergency department a few days later due to increased right lower extremity pain, progressively worsening leg swelling, and an inability to bear weight on the right leg due to pain. The patient stated his pain was most prominent near his groin, which had been there in the prior months.

Chart review revealed a history of HIV with intermittent adherence to antiretroviral therapy for the past year due to various stressors in his life. He was unsure of his last CD4 count but had recently re-established care at the hospital's HIV clinic.

On physical examination, the patient had bulky, painful lymphadenopathy bilaterally, with significantly more lymphadenopathy on the right leg as well as lesions scattered on the lower extremities. A bedside ultrasound of the lymph nodes revealed irregularly shaped, asymmetric lymph nodes.

Upon asking the patient if he had ever been diagnosed with Kaposi sarcoma, he revealed that he thought he had a biopsy within the past year that was positive for Kaposi. Unfortunately, our electronic medical record (EMR) did not have access to the records at the hospital where he reported having the biopsy. The patient also revealed that he had experienced housing instability and had been incarcerated over the past few years, making it difficult for him to regularly follow-up with his infectious disease physician and take his prescribed medications.

MANAGEMENT

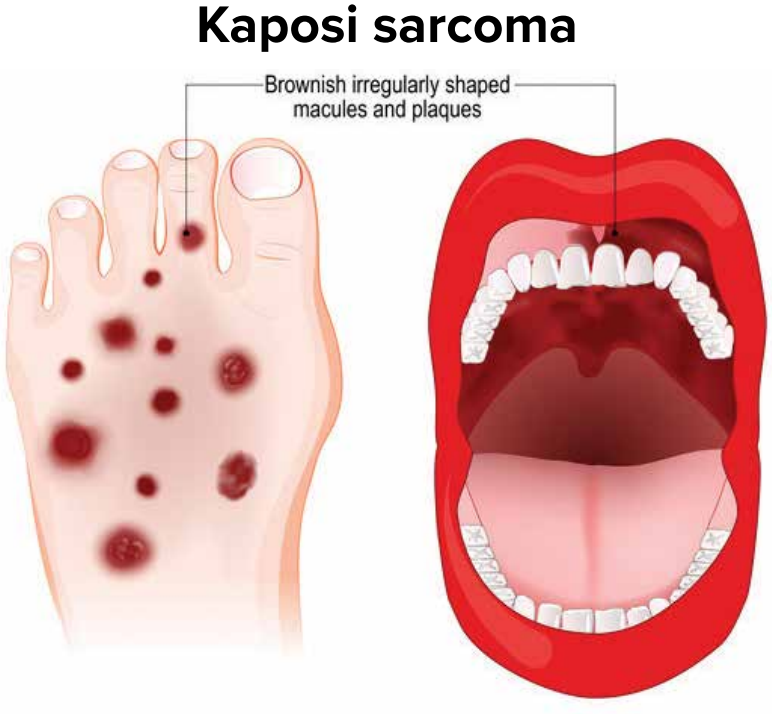
Our differential diagnosis was lymphogranuloma venerum (Chlamydia trachomatis L1-L3), disseminated Kaposi sarcoma, lymphoma, syphilis, and deep space abscess. The team ordered an immune deficiency panel, treponema pallidum antibody, gonorrhea/chlamydia amplification, and started the patient on doxycycline.

Due to the patient's inability to ambulate as well as his lack of stable transportation and reliable accessibility to the health care system, he was admitted to the hospital.

DISCUSSION

Use of Ultrasound

In the radiology literature, benign lymph nodes are described as oval shaped with a thin, hypoechoic cortex (<3mm) and a hyperechoic hilum. Abnormal features of lymph nodes are: a round or asymmetric shape, calcification, ill-defined capsular margins, or a thickened cortex.[1,2] These findings would make one more concerned about a malignant lymph node rather than



Classic Kaposi sarcoma affects the skin on the lower legs and feet

Kaposi sarcoma affects the mouth

References available online.

a reactive, benign lymph node.[3] While it was difficult to assess the cortex and hilum from our images, we felt confident classifying the shape of the lymph nodes as asymmetric. The use of bedside ultrasound made us more concerned about an infiltrative process, specifically Kaposi sarcoma in this patient. Other ultrasound findings that can be found in Kaposi sarcoma include: pleural and pericardial effusions, ascites, and hyperechoic lesions on the liver and spleen.[2]

Kaposi Sarcoma

Kaposi sarcoma (KS) is a malignant systemic disease that originates from the vascular endothelium and traditionally presents in 4 clinical forms. The 2 most well-known forms are in patients who are immunocompromised, such as those who have AIDS or are on immunosuppression after an organ transplant. Human herpesvirus 8 (HHV-8) is present in all forms of KS and interferes with normal cell functions.

Lesions of Kaposi sarcoma are vascular lesions that appear pink or purple and can arise on the skin, and mucocutaneous surfaces such as the mouth. Additionally, there can also be involvement of the lymph nodes, lungs, spleen, and liver. Lesions and lymph nodes that are suspicious for Kaposi should be biopsied for diagnosis of the disease.

Skin lesions due to KS are treated by local excision or liquid nitrogen. In patients with systemic KS due to HIV, highly active antiretroviral therapy (HAART) can be used with regression or full treatment of the disease seen. Severe cases of KS are treated with chemotherapy.[4]

CASE CONCLUSION

During the patient's hospital admission, an MRI of the pelvis showed "Ill-defined, matted soft tissue in the right inguinal region and retroperitoneum." A repeat skin biopsy was done, which showed Kaposi sarcoma. The patient was also found to have pulmonary nodules that were concerning for KS.

The patient was restarted on HAART as well as prophylactic medications through infectious disease consultation due to a CD4 count < 50. Prior to discharge from the hospital, the patient was scheduled for multiple paclitaxel infusion sessions to treat his disseminated Kaposi sarcoma.

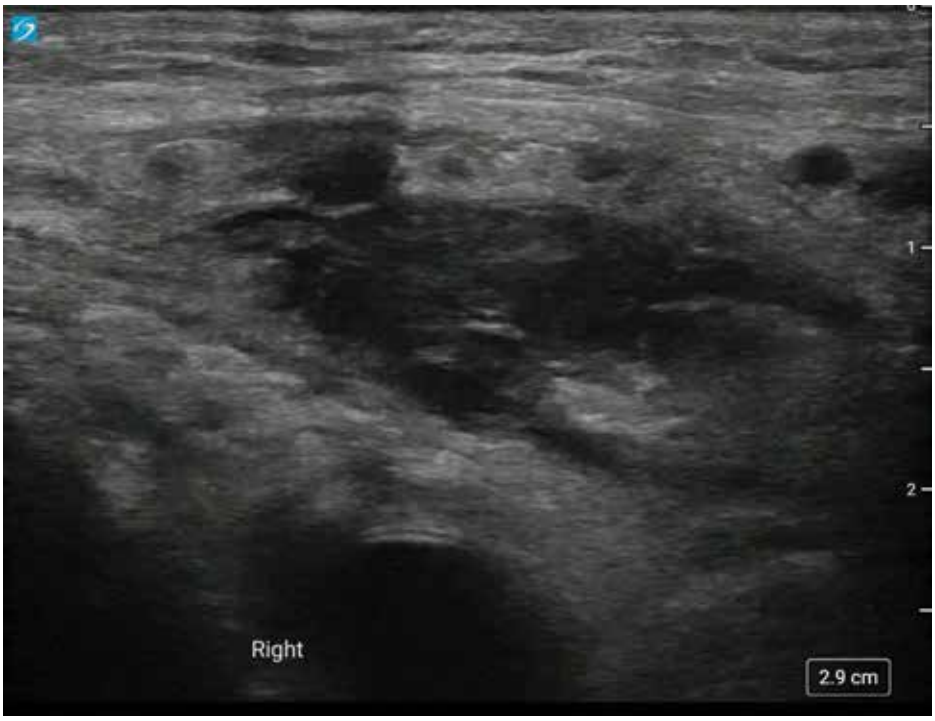


Image 1. Irregularly shaped, asymmetric lymph nodes seen on POCUS

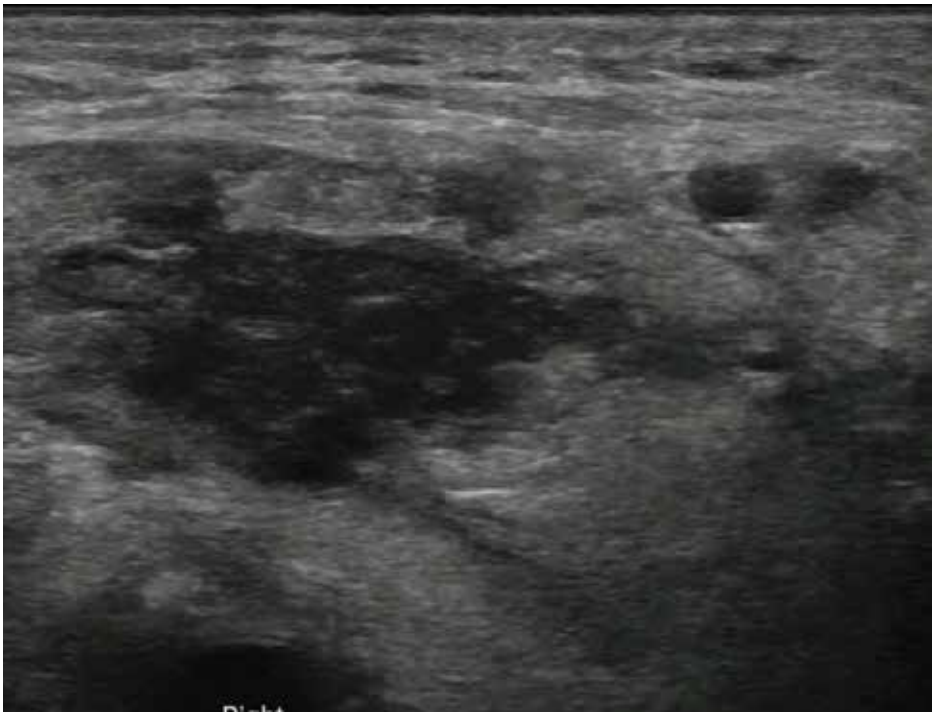


Image 2. Bedside ultrasound showing lymph node irregularities

References available online.

Acute Right Eye Pain and Vision Loss: A Workout Gone Wrong

Corey Michelson, MD
UMass Chan Medical School

Philippe Stanier, MD
Attending Physician
UMass Chan Medical School

Sydney DeArmond, PA
UMass Chan Medical School

Patients with suspected ocular trauma are at risk of vision loss — and emergency physicians must recognize the danger. This is one situation where an emergent ophthalmology consult is critical, as you work to measure and control intraocular pressure.

CASE PRESENTATION

A 67-year-old male with a past medical history of medication-controlled hypertension presented to the emergency department following a right eye injury sustained while working out. The patient was using an exercise band during a home workout when the band snapped, recoiled, and struck him in the right eye. He arrived with severe right eye pain and near-total visual impairment with shadow distinction. He otherwise denied photophobia, pain with extraocular movement, or headache. He never wore contacts or corrective lenses and had no prior ocular history. On physical examination, several minor superficial abrasions were noted on the right eyelid, and his right eye was erythematous. His right pupil was 5mm and non-reactive. His left eye was within normal limits, EOM intact and without pain. No proptosis was identified.

An exam under fluorescein stain identified several minor corneal abrasions with a negative Seidel sign. On tonometry, the right eye's intraocular pressure (IOP) was 56 mmHg, and the left eye's IOP was 10 mmHg. A diagnosis of traumatic glaucoma was made.

CASE DISCUSSION

Traumatic glaucoma is a form of secondary glaucoma that can develop following trauma to the eye and orbit. More common after blunt trauma, it can nevertheless occur following penetrating injury. Traumatic glaucoma may also develop after chemical, thermal, electrical, or radiation injuries.

Often, traumatic glaucoma develops immediately following trauma to the eye, although on occasion, it may present days to weeks following the initial injury. The disease progresses when increased aqueous humor collection within the eye leads to increased IOP and damage to the optic nerve. Increased IOP may develop after ocular trauma through several mechanisms: disruption of the trabecular meshwork after globe trauma, bleeding into the anterior chamber (hyphema), or traumatic, inflammatory changes to the iris leading to compromise of the blood-aqueous barrier.

A complicating factor in this case was the apparent posterior lens subluxation noted on ultrasound (Image 1). Posterior displacement of the lens can lead to rupturing of the zonular fibers which hold the lens in place. Subluxation typically occurs when 25% or more of the zonules are torn. Full lens dislocation (ectopia lentis) may occur with complete disruption of the zonules. It is thought that the posteriorly dislocated lens may have promoted intermittent angle closure or that the vitreous prolapse (vitreous leak into the anterior chamber and

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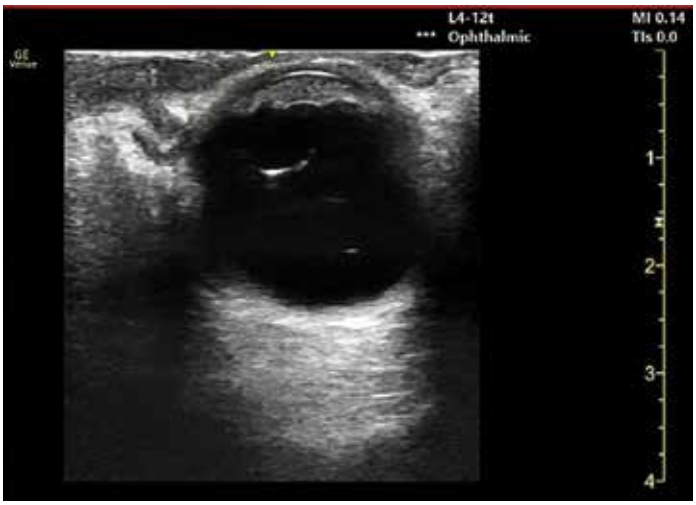
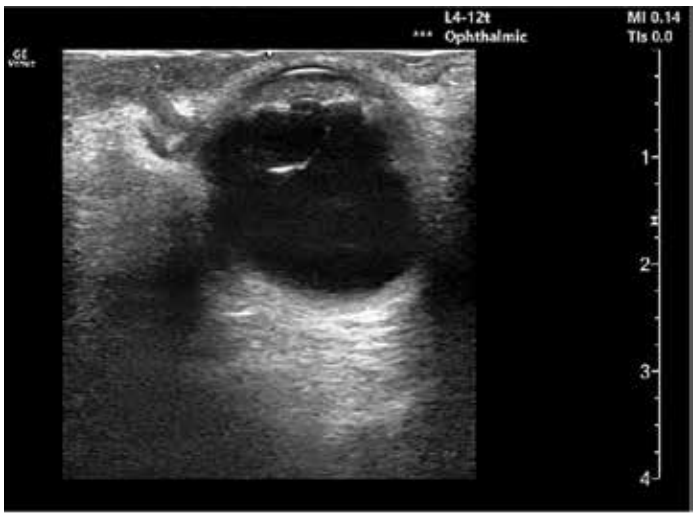




Images 1 and 2. Ultrasound images of the affected eye noting a posteriorly subluxed lens with mild vitreous hemorrhage

pupil) may have disrupted the trabecular meshwork, disrupting clearance of aqueous humor. Subluxed or dislocated lenses may also cause local trauma and irritation to the nearby iris, leading to the release of pigments into the vitreous, which may also work to disrupt proper aqueous absorption.

Once the diagnosis was made, treatment was promptly initiated (see below), and ophthalmology was consulted.

VISION-SAVING TREATMENT IN THE ED

In the emergency setting, treatment of traumatic glaucoma must focus on reducing IOP while establishing a plan for definitive care. Medical treatment aims to accomplish this by increasing aqueous humor outflow and reducing aqueous humor production. The patient was administered IV acetazolamide to reduce aqueous humor production. Dorzolamide/Timolol (combined topical carbonic anhydrase inhibitor and beta-blocker) and Brimonidine 0.15% (alpha agonist) eye drops were administered every 15 minutes, along with serial tonometry measurements. Despite these measures, the IOP remained elevated in the affected eye, and this same regimen was continued for another hour. It is postulated that the IOP remained elevated despite medical therapy because the posteriorly dislocated lens may have been intermittently and transiently obstructing the resorption of aqueous humor.

Following the second hour of medical therapy, intraocular pressures improved but remained elevated at 30-40 mmHg. At this time, a decision was made to transfer the patient to a center with additional ophthalmological capabilities for further management. While awaiting transfer, the patient was provided additional medical therapies, including prednisolone and Cyclopentolate eye drops, and was administered a one-time dose of IV mannitol over 45 minutes with a continued goal of normalizing intraocular pressure at the recommendation of our ophthalmology consultants.

On outside hospital arrival, right intraocular pressure was measured at 21 mmHg. He was observed for several hours with serial pressure checks and ultimately discharged home with a plan for IOP re-check in 24 hours and non-emergent outpatient surgical management.

He was instructed to continue prednisone, brimonidine, acetazolamide, and dorzolamide/timolol, to sleep with head of bed elevated, and to avoid NSAIDs and any straining or heavy lifting.

TAKE-HOME POINTS

- Measuring intraocular pressure in patients with suspected ocular trauma is essential. However, it is critical to exclude globe rupture first, as measuring intraocular pressure in this case can be harmful. Elevated intraocular pressure (usually greater than 30mmHg although diagnosis can be made with >21 mmHg), along with eye pain and a minimally reactive pupil, is concerning for acute traumatic glaucoma.
- This is one situation where an emergent ophthalmology consult is critical. The goal for ED physicians is to reduce IOP until ophthalmology decides on a definitive care plan. Early and effective therapy is crucial in optimizing ocular nerve perfusion and limiting potential permanent vision loss. Medical intervention aims to increase aqueous humor outflow while reducing production.
- Ocular ultrasound is a valuable tool in identifying additional contributory pathologies such as lens subluxation/dislocation or detachment, vitreous hemorrhage or detachment, or retinal detachment.

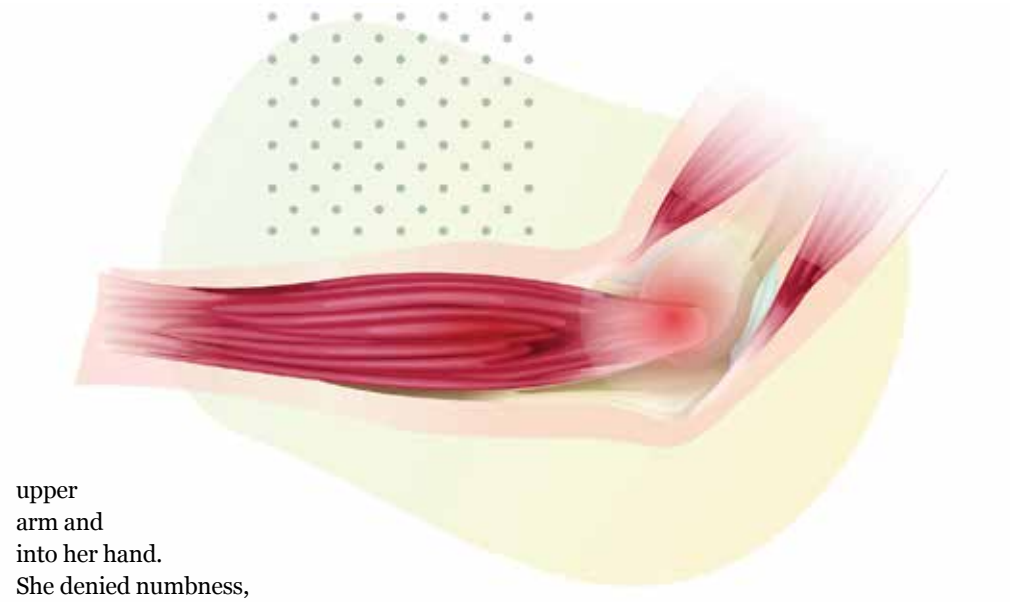
References available online.

Morel-Lavallée Lesion: A Case Report

Rana Akkad, DO
Creighton University School of Medicine/
Valleywise Health Medical Center (Phoenix)
Emergency Medicine Residency

Sarah Taetz, MD
John Peter Smith Health Network

Geoffrey Comp, DO, FACEP, FAWM
Creighton University School of Medicine/
Valleywise Health Medical Center (Phoenix)
Emergency Medicine Residency



Morel-Lavallée lesions (MLLs) are soft tissue internal degloving injuries that develop following high-velocity trauma, most commonly, motor vehicle collisions.

Impact transmitted into the soft tissue causes a shearing force that separates the hypodermis from the underlying fascia, resulting in the disruption of lymphatics and capillary beds, and ultimately the development of a fluid collection. This fluid collection becomes necrotic and elicits a chronic inflammatory response.[1]

Because they often present several days following the traumatic impact, MLLs often go undiagnosed or have a delayed diagnosis, which impedes prompt treatment. This can lead to the development of a chronic lesion and increases the risk of complications including life-threatening infections. Thus, it is important to recognize the signs of MLLs, which include swelling, ecchymosis, fluctuance, and hypermobility of the skin.[2] MLLs are usually associated with fractures of the greater trochanter, femur, or pelvis, but have been reported in the knee, gluteal region, and abdomen, with few reports in the literature of upper extremity MLLs.[1]

CASE REPORT

A 42-year-old female with no pertinent medical history presented to the emergency department with a chief complaint of 1 day of left forearm pain. The patient stated that she was on a boat, when a rope became wrapped around her forearm, and then pulled with extreme force, causing a soft tissue twisting injury. The patient initially noted some swelling and erythema, and tried over-the-counter analgesics, but continued to experience progressive swelling and discomfort. At the time of presentation, the patient was complaining of pain radiating to her upper arm and into her hand. She denied numbness, tingling, or any other symptoms.

The patient was a well-appearing middle-aged female in no acute distress. Vital signs demonstrated a blood pressure of 111/67, heart rate of 89 bpm, respiratory rate of 16, oxygen saturation of 98%, and temperature of 36.9°C. The patient rated her pain level as a 5 on a scale of 1-10.

On physical exam, there was an obvious deformity and pain to palpation of the left upper extremity near the proximal left wrist and distal forearm. Range of motion was limited in wrist flexion and extension as well as elbow flexion and extension secondary to pain. Patient had an 18 cm x 13 cm area of swelling and erythema to the volar aspect of the left forearm with cutaneous abrasions from the rope. Compartments were soft, and radial pulses were 2+ bilaterally. No crepitus was appreciated. Sensation was intact. The extremity was warm, with a capillary refill of less than 2 seconds. There was normal range of motion of the shoulder, hand, and fingers.

A two-view X-ray of the left forearm and a two-view X-ray of the left wrist were obtained to evaluate for an acute fracture or dislocation. No acute fractures or dislocations were observed, joint spaces were preserved, and alignment was maintained. Soft tissues appeared normal.

The patient was given oral narcotics for pain.

On reassessment, the patient continued to have pain out of proportion to the exam. Therefore, a CT scan of the left upper extremity with IV contrast was ordered to assess for further injury. The CT scan described soft tissue and subcutaneous fat edema with a small amount of fluid along the palmar aspect of the forearm measuring up to seven mm in thickness. No vascular injury was detected.

Blood work was obtained to evaluate for evidence of rhabdomyolysis or acute kidney injury. Total creatine kinase was slightly higher than the reference range at 587 U/L (normal 30-135 U/L). Basic metabolic panel revealed no electrolyte abnormalities and normal creatinine and GFR. The patient continued to have pain in the emergency department and was given IV narcotics for pain.

Based on the CT scan findings as well as the mechanism of injury, the patient was diagnosed with a Morel-

References available online.

Lavallée lesion. The surgical team was consulted and evaluated the patient. They agreed with the diagnosis and admitted the patient for further evaluation and treatment.

Treatment consisted of pain management and an evaluation by hand surgery, who recommended operative intervention. The patient went to the OR the next day for a left forearm exploration, washout, fasciotomy, and incision and drainage with packing. The patient received antibiotics prior to surgery. Intraoperative wound culture did not grow any organisms.

Prior to discharge, the patient met all of physical therapy's mobilization requirements and was taught how to perform wound care. The patient was discharged from the hospital on day 3 with oral antibiotics, pain medications, and a follow-up appointment in the hand surgery clinic within 1 week.

DISCUSSION

This case is of interest as it highlights that Morel-Lavallée lesions can be found in the upper extremity and should be considered when the mechanism of injury involves a shearing force. This patient's initial presentation suggested that she had a fracture of either her radius or ulna secondary to the swelling and obvious deformity of her forearm. Therefore, an X-ray was done first to evaluate.

Once the patient's X-ray came back negative for fracture, suspicion was higher for other underlying pathology given the degree of swelling and pain. Although in this case the patient had no fracture, it is important to note that the patient could still have a MLL in concurrence with a fracture. Literature review shows that MLLs have been diagnosed in both traumas with and without fractures.[3] Had this patient presented with a fracture, it is possible that her swelling and deformity would have been attributed to a fracture rather than an MLL.

Missed or delayed diagnosis of this MLL could have led to complications and permanent contour deformity.

It is important to note that MLLs are underreported and are often misdiagnosed, therefore the published rate of incidence is most likely an underestimation. This makes it difficult to establish an overall incidence, as well as the rate of occurrence in tandem with fractures.

A thorough literature review was conducted using the following keywords: Morel-Lavallée lesion, emergency department, upper extremity, internal degloving, degloving injuries, upper, arm, shoulder, chest, abdominal, neck, head, spine, spinal, wrist, or hand. This resulted in 21 articles being found. Of these, 3 were in reference to case reports of Morel-Lavallée lesions in the upper extremity. Two of these cases were diagnosed outside of the emergency department. One report was diagnosed 10 months after injury during a follow-up with hand surgery.[4] The second report was diagnosed during follow-up with orthopedic surgery after an ED visit.[3] It is unclear how the third report was diagnosed, as it is a radiology case report.[5]

Our case report is unique, as it was an upper extremity MLL diagnosed by emergency physicians during the patient's first evaluation.

This resulted in prompt identification and treatment of the patient's lesion, minimizing chances of infection and permanent deformity. Furthermore, this patient was promptly seen by the hand surgeons on call and was taken to the OR. Clinical course was uncomplicated. Ramaseshan et al. reported about 33-44% of Morel-Lavallée lesions are misdiagnosed.[6] This rate of misdiagnosis is suspected to be higher in upper extremity lesions, as they are quite rare and therefore may not be in a physician's primary differential. Vanhegan et al. reviewed 204 Morel-Lavallée cases in 29 published papers and found the incidence of Morel-Lavallée lesions based on location was reported to be the greater trochanter or hip 30.4%, thigh 20.1%, pelvis 18.6%, knee 15.7%, gluteal region 6.4%, lumbosacral 3.4%, abdominal wall 1.5%, calf or lower leg 1.5%, head 0.5%, and unspecified 2.0%.[7] Given the distribution reported above, it can be deduced that the unspecified reports may encompass the upper extremity MLLs.

Morel-Lavallée lesion presentations differ between patients and cases. Presentations may also differ based on the chronicity of the lesion. They can present as painful lesions or even nonpainful lesions secondary to hypoesthesia from damage to cutaneous nerve branches.[8] They can mimic various disease processes such as contusions, DVTs, cellulitis, fractures, soft tissue injuries, or abscesses. It is important to consider Morel-Lavallée lesions when working up patients with traumatic injuries, as misdiagnosis may lead to complications such as infection, necrosis, permanent deformity, and even death. There is a reported 1.7% comorbidity rate.[9] Morel-Lavallée lesions have also been reported to cause hemorrhagic shock and death secondary to active bleeding into the lesion.[8]

Gold standard for diagnosing Morel-Lavallée lesions is MRI; however, CT scans and even point of care ultrasound have been found to be useful.[10] Early detection can alter the management of MLLs and positively change clinical outcomes, thus it is important for emergency physicians to consider MLLs when evaluating and treating traumatic injuries.

CONCLUSION

Although MLLs are relatively uncommon, this may be confounded by the number of MLLs going undiagnosed. Thus, early recognition of MLLs is imperative to ensure prompt treatment. If left untreated, MLLs can lead to chronic and debilitating sequelae including infection, skin necrosis, and impaired function and mobility.[11] Furthermore, it is important for emergency physicians to maintain a high index of suspicion for MLLs in cases involving blunt trauma, even when the injury is in locations that are uncharacteristic of MLLs, such as the upper extremities and in cases where fractures are not present.

References available online.

The Deadliest Genital Ulcer Is Not Infectious: A Case Report of Penile Calciphylaxis

Andrew Good, DO **Agata Dow, MD** **Chris Kilen, DO** **Katherine Jones**
Department of Emergency Medicine,
Henry Ford Wyandotte Hospital

Calcific uremic arteriolopathy (CUA) is a systemic disease, and the arteriolar calciphic depositon leads to vascular stenosis, thrombosis, and infarction of tissues around the body.

This can be seen anywhere in the body and is most commonly seen in distal extremities, in subcutaneous tissues with high adipose contents.[1] Pain is typically exquisite. End-stage renal disease (ESRD) itself is a predictor of high mortality, which is tripled in the presence of calciphylaxis.[1] Mortality of CUA in ESRD is 33%-50% at 6 months, and with penile involvement this mortality is estimated to be 50% at 3 months.[1-3] Ulceration of calciphylactic lesions increases the mortality rate to above 80%,[2] and superimposed infection is not uncommon.

It should be noted that not all cases of CUA are associated with ESRD; independent risk factors include warfarin therapy, primary hyperparathyroidism, malignancy, and alcoholic liver disease.[1] This is a rare pathology, and undifferentiated cutaneous lesions have a vast array of possible etiologies.[4] Because clinical uncertainty is frequent, this subsect of patients represents a vulnerable population who require prompt team evaluation and subsequent medical optimization.

CASE REPORT

A 51-year-old man with a past medical history of ESRD, diabetes mellitus, hypertension, and peripheral vascular disease presented with an exquisitely painful penile ulcer that had been rapidly expanding for the past 2 weeks. The patient further reported a chronic right calcaneal ulcer that was weeping fluid recently. He presented to the emergency department for 2 weeks of painful ulcer expansion. The patient has been put on calcium supplements and vitamin D for treatment of ESRD related hypocalcemia and anemia.

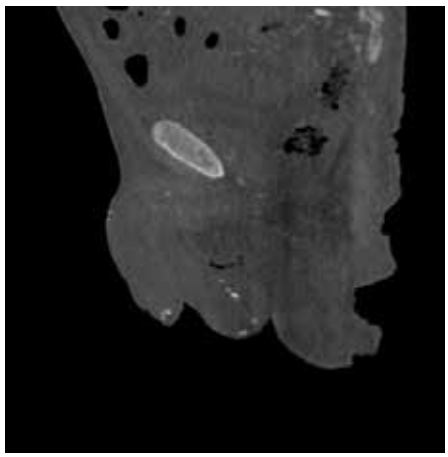
Vitals include a blood pressure of 148/88 mm Hg, pulse of 89 beats/minute, temperature of 98.2°F (36.8°C), respirations rate of 20 breaths/minute, oxygen saturation of 100% on room air, and body mass index of 21.02 kg/m^2. Physical examination noted indurated, disfiguring, and exquisitely tender ulceration of the entire glans penis, with near destruction of urethral meatus (**Image 1**).

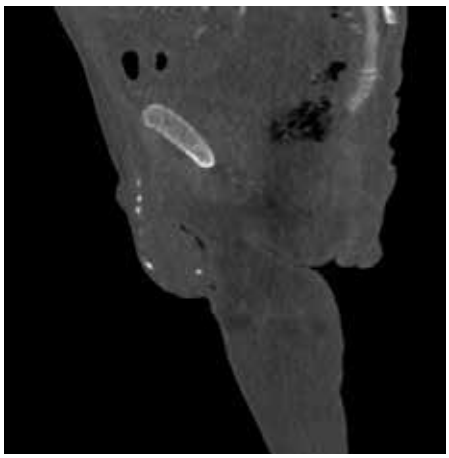
While in the emergency department, the patient required multiple doses of morphine for control of his severe and recalcitrant penile pain. Vancomycin and cefepime were administered, given his infected calcaneal ulcer and history of extended-spectrum beta-lactamase Enterobacter in previous wound cultures. The patient's lesion was not a classic, violaceous ulcerating eschar indicating high suspicion for calciphylaxis; concern for infectious, neoplastic, vasculitic versus calciphylactic etiologies remained on the differential diagnosis.

CTA with bilateral runoff was obtained, given the patient's history of peripheral vascular disease and infected extremity ulcer. The imaging noted severe diffuse generalized arterial vascular calcification throughout all visualized arterial vascular structures including the aorta as well as the large, medium and small arteries (**Images 2a and 2b**). As noted, the degree/extent of arterial vascular calcifications was severe, essentially involving all arterial vascular structures diffusely.

Infectious disease consultation recommended cessation of antibiotics with herpes simplex swabbing, later determined to be negative. Squamous cell carcinoma was high enough to merit biopsy, which was performed by his urologist. Biopsy results indicated foci of dystrophic calcification as well as ulceration with fibroinflammatory

Image 1. Penile ulceration





Images 2a and 2b. Penile and testicular calcifications seen in sagittal view of computed tomography of the abdomen/pelvis

References available online.

exudate, granulation tissue, and acute inflammation of the dermis. Staining was negative for bacterial and fungal organisms.

The patient was not treated with sodium thiosulfate or hyperbaric oxygen therapy, and he declined penile amputation as a potential treatment modality. Instead, symptomatic control and goals of care discussion were prioritized. The patient's pain improved on hydrocodone/acetaminophen 5/325 mg with morphine 2 mg every 4 hours as needed while in the hospital.

He was discharged on gabapentin 300 mg daily with subjectively improved, though not resolved, symptomatic control. One month following his presentation for penile pain, he presented in volume overload from missed dialysis. While in the hospital, he unfortunately went into ventricular fibrillation and expired.

DISCUSSION

Penile pain has primarily infectious and traumatic etiologies, which may dampen clinical concern for calciphylaxis, a diagnosis with a three-month median survival.[3] Calciphylaxis affects an estimated 4% of ESRD patients and of these patients, only 6% have penile involvement.[1] Resultantly, if clinicians are not considering the diagnosis, they are very likely to miss it. Clinicians should consider calciphylaxis, especially if the patient has a history of ESRD, hyperparathyroidism, warfarin use, malignancy, or other risk factors.[1] Calciphylactic lesions are typically acutely violaceous, subcutaneous nodules or plaques. They later evolve into necrotic, dusky ischemic necrosis. This will likely occur in conjunction with other integumentary lesions; however, it may occur as an isolated lesion like in the case presented above.

Clinicians may obtain basic chemistry, calcium, and parathyroid hormone levels to evaluate for signs of renal failure, hypercalcemia, or hyperphosphatemia in conjunction with evaluation of risk factors. Regardless, diagnosis is largely clinical. The differential diagnosis may include neoplastic, infectious, vasculitic, iatrogenic, atherosclerotic, or autoimmune etiologies. It may resemble squamous cell carcinoma, sexually transmitted infection, cellulitis, warfarin skin necrosis, cholesterol embolism, or vasculitis. Gabel et al. noted in a study that of 119 patients diagnosed with calciphylaxis, 73.1% were initially misdiagnosed — with the vast majority receiving antibiotics for presumed cellulitis.[4] A multidisciplinary team is therefore recommended for proper diagnosis and treatment.

To definitively diagnose calciphylaxis, a biopsy visualizing calcific arteriolopathy is required; however, it is not encouraged. Conservative management is typically recommended, because trauma to calciphylactic lesion risks worsening of the patient's pain, extension of the lesion, infection, and ulceration. Infection of these lesions is the primary cause of high mortality and ulceration carries greater than 80% mortality rate.[1,2,5,6] Varying treatment success has been noted in multiple case reports, including sodium thiosulfate, hyperbaric oxygen, and penectomy.[7-10] Floege et al. reported a significant reduction in the development of calciphylaxis with initiating cinacalcet when patients begin dialysis; however, this study has been criticized for a poor attrition rate and further studies are required to determine measurable benefit.[11] Large-scale evaluation of each of the above treatments has found mortality benefit.[1]

The patient presented in this paper is an unfortunate example of the swift mortality following penile calciphylaxis. Without any treatment modalities demonstrating mortality benefit, goals of care discussion is perhaps the most productive intervention available. Symptomatic control should be attained through acetaminophen and opiate medications.[12] Morphine and codeine are at risk of accumulation in reduced kidney function, so hydromorphone, fentanyl, or methadone are preferred.

CONCLUSION

Penile calciphylaxis is a harbinger of looming mortality, indicating need for a clear goals of care discussion. Biopsy is not routinely recommended, as this can increase risk for ulceration and superinfection and worsen outcomes. No treatments have established mortality benefit, though cinacalcet, sodium thiosulfate, and penectomy have shown benefit in case reports. Pain control is the mainstay of therapy, with acetaminophen, hydromorphone, and fentanyl as the primary recommended medications. This pathology is best cared for by a multidisciplinary team.

Attestation

The Henry Ford Institutional Review Board does not require approval for case reports with the exception of genitalia. The photographs obtained in this photo were obtained with informed consent and knowledge they may be used for publication and educational purposes. This consent is submitted with the associated manuscript.

References available online.

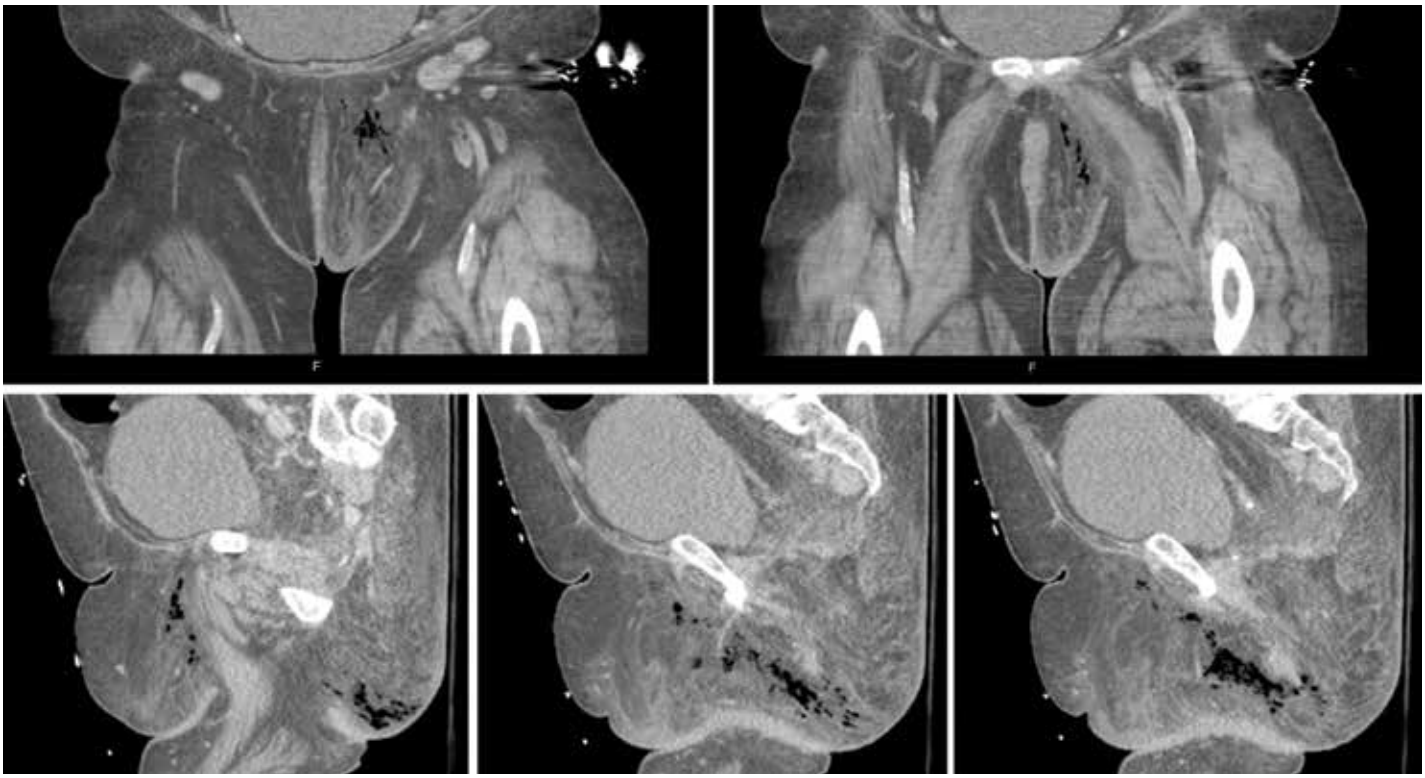


Image 1. CT scan of our patient on presentation. A. & B. Frontal slices demonstrating pneumolabium. C., B., & E. Axial slices demonstrating subcutaneous emphysema tracking through the perineum.

Pneumolabium: A Case Report of COVID Turned Fournier's Gangrene

Miranda Teixeira, MD
University of Tennessee Health Sciences Memphis Emergency Medicine

Phillip Jordan, MD
Eastern Virginia Medical School Emergency Medicine

This paper underscores the essential role of comprehensive history-taking and physical examination in emergency departments to mitigate the risk of diagnostic anchoring.

INTRODUCTION

When a patient comes into the emergency department with a diagnosis listed as the cause of their ailment, clinicians can inadvertently narrow their focus influencing treatment decisions. Before seeing the patient, orders are in, and the workup is begun based on the triage note. This report discusses a case where reliance on initial triage information, which suggested a respiratory infection, could have obscured a separate life-threatening condition. By incorporating comprehensive questions and performing a diligent physical exam, the clinicians course-corrected and provided the appropriate lifesaving treatment.

CASE PRESENTATION

A 67-year-old white woman was brought to the ED by EMS, with a chief complaint listed as COVID. Upon arrival, she was visibly short of breath and ill-appearing. The patient was hypotensive at 89/50 mmHg, hypoxic with an oxygen saturation of at 87% on room air, a respiratory rate of 27, a temperature of 37.6°C, and normal mentation. She reported testing positive for COVID 5 days prior and since then has had nausea, fatigue, and decreased oral intake. The patient is ambulatory at baseline, with a BMI of 37.12 kg/m^2, and reports handling all her daily activities independently. However, for the past 3 days she has had worsening weakness, prompting her call to EMS. The patient has a history of diabetes mellitus, coronary artery disease, DVT, and a remote history of thyroid, colon, and ovarian cancer in remission. The patient also states she has not been taking her medications for the past few days.

While gathering history, the patient states that her buttocks and lower back have been hurting, and she thinks the cyst on her vulva has reoccurred, which has required operative drainage previously.

References available online.

On exam, the patient had an erythematous, indurated area, without fluctuance of her left hemi-vulva that was painful to palpation. Upon rolling the patient, the team found a similarly inflamed left buttock region extending 7 cm laterally from her intergluteal cleft. This appeared continuous with the inflammation on her vulva tracking through the perineum and surrounding her left perianal region. There were no areas of ulceration, palpable crepitus, or necrosis noted on exam and no drainage from the area. The patient's pain appeared out of proportion to her exam.

After the physical exam, our concern swiftly transitioned from a COVID-19 related ailment to concern for necrotizing soft tissue infection, particularly Fournier's gangrene, as the source of her deterioration.

DIAGNOSTICS AND INITIAL TREATMENT

During the evaluation, the patient developed an increasing oxygen requirement. Although the patient remained alert and conversational during this period, she remained hypotensive despite 2L of lactated ringers. Therefore, norepinephrine vasopressor therapy was initiated, and broad-spectrum antibiotics were administered. Blood cultures, lactate, CBC, CMP, and a CT of the abdomen and pelvis were obtained.

A preliminary interpretation by the emergency physician demonstrated obvious pneumoperineum with subcutaneous emphysema tracking along the left gluteal fold with left sided pneumolabium (see **Image 1**).

General surgery was immediately consulted for arrangement of emergent surgical debridement now that the diagnosis of Fournier's gangrene was clear. The general surgeon performing surgical debridement observed the presence of "dishwasher fluid" and notable lack of bleeding during debridement, both of which are consistent with Fournier's. Necrotic tissue encompassing the left labia majora extending to the left thigh, down to the left buttock below the anal verge was excised down to the adductor muscle. Post-procedure, the patient was transferred from the operating room to the ICU, where she continued to require high doses of vasopressor therapy — eventually necessitating a return to the OR for additional debridement for a total area of 1650cm^2.

DISCUSSION

Fournier's gangrene is a necrotizing soft tissue infection (NSTI) representing less than 0.02% of hospital admissions per year.[1] The prevalence in females is exceedingly rare, at a rate of 0.25 cases per 100,000 female patients.[1] Despite its rarity, prompt identification and surgical consultation is paramount due to a 1 in 5 mortality rate.[2]

Increasing the difficulty of the diagnosis, patients with necrotizing infections may present unaware of the severity of their infection due to a phenomenon known as "la belle indifference" in which the involved tissue destruction results in insensate regions of infection.[3] Despite the extensive area of obvious infection on exam, this patient seemed unaware of its severity. An astute history and physical exam led to the rapid recognition of this often life-threatening condition.

TOXICOLOGY, PHARMACOLOGY

Case Report: A Review of Metformin-Associated Lactic Acidosis

Madeleine Silverstein, MD; Avirale Sharma, MD; Vivek Medepalli, MD; Andrea Nillas, MD; Jedidiah Leaf, MD
University of Texas Southwestern Medical Center

Knowing a patient's pharmacologic history is key to an accurate diagnosis and appropriate treatment.

CASE PRESENTATION

A 68-year-old male with a history of type II diabetes mellitus, hypertension, hypercholesterolemia, and hypothyroidism presented to the emergency department with lightheadedness that began 2 days prior. He reported associated nausea, vomiting, shortness of breath, and non-bloody diarrhea. Patient denied fevers, chills, cough, congestion, chest pain, dysuria, or back pain. He had been working outdoors for the past few days. Patient reported compliance with his medications as prescribed including glipizide, levothyroxine, metformin. He denied any recent sick contacts, travel, new foods, or antibiotic use. Initial vitals were heart rate of 82 beats per minute, blood pressure of 171/67 mmHg, temperature of 37.2°C (oral), SpO2 of 100% on room air. Physical exam was remarkable for dry mucous membranes, mild tachypnea, and mild abdominal discomfort. Mental status was within normal limits.

Initial laboratory results showed a complete blood count remarkable for a leukocytosis of 16 x 1,000/mm^3 with neutrophilic predominance. Complete metabolic panel revealed sodium of 142 mmol/L, potassium of 5.7 mmol/L, chloride of 90 mmol/L, CO2 of 3 mmol/L, anion gap of 49, blood urea nitrogen of 82 mg/dL, creatinine of 11 mg/dL. Liver enzymes were within normal limits. Initial lactate was 15.5 mmol/L. The VBG was remarkable for

References available online.

pH of 6.88, pCO2 of 21 mmHg, and HCO3 of 4 mEQ/L.

IV fluids, ondansetron IV, and multiple pushes of sodium bicarbonate were given. Nephrology and MICU were consulted, and the patient was started on a continuous bicarbonate infusion. The differential included sepsis, other causes of anion gap metabolic acidosis including toxic ingestion, mesenteric ischemia, and bowel obstruction, which are all ruled out. Therefore, given the history along with exam findings, the suspected diagnosis is metformin associated lactic acidosis (MALA). This is suspected to be precipitated by dehydration leading to acute kidney injury (AKI), causing metformin accumulation, and finally worsening the AKI.

DISCUSSION

Metformin is a commonly prescribed anti-diabetic agent and regularly places in the top 10 most prescribed medications in the United States each year.[1] Metformin is a biguanide that is recommended for prevention of progression in patients at high risk for developing Type 2 diabetes, for glycemic control in diabetes, and as first line therapy for elderly adults with diabetes. It is relatively well-tolerated, relatively low-cost, rarely causes hypoglycemia compared to insulin or sulfonylureas, and is recommended by the American Diabetes Association.[2,3,4] Though exceedingly rare, affecting approximately 1 in 30,000 patients,[5] metformin has an FDA black box warning due to lactic acidosis, which could cause multi-organ failure and death.[6] An estimation of MALA incidence is about 6.3 per 100,000 patient-years with 50% mortality.[7]

Lactate elevation and subsequent acidosis is a relatively non-specific finding seen in many pathologies and can be divided into two sub-categories. Type A is an elevation due to anaerobic metabolism from hypoxia or hypoperfusion. Type B is unrelated to hypoxia or hypoperfusion.[8] MALA pathology begins at the cellular level. Lactate elevation occurs due to inhibition of complex I of the electron transport chain, shifting ATP generation to anaerobic metabolism and lactate formation. Additionally, through metformin's inhibitory effects on gluconeogenesis, hepatic clearance of lactate is decreased, further potentiating the acidosis.[9,10] MALA typically occurs secondary to an underlying condition, in most cases renal injury or disease. Risk factors for MALA include sepsis, liver disease, heart or lung disease, alcohol use, and history of MALA.[2]

To date, there is no diagnostic test to confirm the specific diagnosis of MALA. Metformin levels can be obtained; however, they are of limited use in the ED as they require sending the sample to a reference laboratory and may take days to result. Additionally, metformin levels do not correlate with the severity of MALA.[2] While the diagnostic criteria are not well defined, literature suggests consideration of MALA with a pH <7.35 and a lactate of >5mmol/L in the context of known metformin exposure.[6] Additionally, several other causes of lactic acidosis, many reversible, must also be evaluated prior to making this diagnosis.

MANAGEMENT

Regarding predictions of outcomes for MALA, there is controversy regarding pH and lactate association with patient mortality. There have been case reports showing possible associations[2] however, there are larger studies that suggest outcome is relative to the complexity and reason for MALA.[11]

Recognizing MALA is key for quick initiation of treatment, made challenging by the fact that MALA is associated with nonspecific symptoms including altered mental status, vision changes, breathing problems, gastrointestinal symptoms, and dizziness.[2,12]

Supportive care is recommended, including airway management and blood pressure stabilization. Sodium bicarbonate is recommended for bicarbonate level <5mEQ/L. Hemodialysis is recommended in severe cases to correct the acidosis. It is encouraged if lactate is greater than 20 mmol/L, pH is less than or equal to 7, or if other therapies including sodium bicarbonate have been utilized without improvement in the patient's clinical condition.[2,4]

CASE RESOLUTION

The patient was admitted to the MICU for acute renal failure requiring emergent dialysis. An internal jugular dialysis catheter was placed for emergent dialysis in the ED, and intermittent hemodialysis was initiated in the ICU followed by continuous renal replacement therapy (CRRT). The bicarbonate drip was also continued to treat acidosis.

Additionally, given concern for prerenal contribution to renal injury given time spent outdoors in the heat, vomiting, and diarrhea, maintenance fluids were started. These treatments resulted in significant improvement in the patient's clinical status, with resolution of acidosis, improvement of Cr from 11 mg/dL to 1.8 mg/dL, lactate peaking at 29 mmol/L prior to dialysis, and down to within normal limits after hemodialysis and CRRT.

Soon after, the patient began tolerating oral intake with resolution of nausea, vomiting, diarrhea, and was deemed medically stable for transfer to the floor. The next day, he was discharged from the hospital with nephrology recommending stopping metformin. The patient followed up in the clinic 1 month later and reported feeling well, with full resolution of symptoms.

References available online.

UMMC Pediatric Emergency Department Response to the Rolling Fork Tornado

Heath Broussard II, DO
Andrew Buckelew, DO
Kerry Yancy, MD
University of Mississippi Medical Center

Editor's note: Thanks to Alix Tromblay, MD, Mohammad Sheikh, MD, Johnathan Princiotta, MSN, Hannah Dearman, RN, and John McCarter, MD, for their contributions.

On the night of March 24, 2023, central Mississippi was ravaged by an EF4 tornado that ultimately claimed the lives of 22 victims statewide and injured 143 others, many critically.[1] An EF4 storm has wind gusts of 166-200 mph,[2] typically creating forces that destroy well-built homes and can throw automobiles and even tractor-trailer trucks long distances. As the tornado stayed on the ground for 59 minutes, it caused damage in several Mississippi towns and surrounding rural areas, devastating the town of Rolling Fork.[3]

Rolling Fork is a town with a population of fewer than 2,000 — 40% under age 20 — and a 50% unemployment rate.[4] Many residents live in mobile homes, and few of the traditional homes and apartments were built to withstand a tornado of such magnitude. In Rolling Fork, 51.1% of the homes were lost, and 60.7% businesses were destroyed or sustained major damage.[5]

This tornadic devastation created a mass casualty event for the state of Mississippi. One of the greatest potential failures of a medical facility is a lack of preparation and proficiency required to care for such an event. As the only pediatric hospital and Level 1 trauma center in Mississippi, the University of Mississippi Medical Center (UMMC), located in Jackson, urgently began preparations for the inevitable surge of injured patients.

PEM-FOCUSED DISASTER RESPONSE

At approximately 9:30 pm, transfer requests began to come into Mississippi MedCom, the major emergency referral center for Mississippi which provides hospital transfer assistance and online medical control for about two dozen counties throughout Mississippi.[6] One was a call from an emergency physician at Baptist Memorial Hospital in Yazoo, near Rolling Fork, reporting that 3 children had arrived in critical condition, one receiving CPR. All would need to be transferred to UMMC, an approximately hour-long journey by ambulance. Shortly after this initial notification, MedCom staff received a call from EMS on scene that the number of children with serious

References available online.

injuries could be much greater and transfers might continue through the night. This uncertainty was worsened by the storm occurring at night.

UMMC's pediatric emergency department (PED) has 17 general use patient rooms, 2 trauma bays, and 7 "fast track" rooms. We also have the space to place several stretchers in the halls and tightly fit an extra stretcher into each trauma bay. At the time of the initial call from MedCom, a total of 20 patient rooms were filled. With less than an hour of warning, there was a clear need for available rooms to receive tornado victims and to prepare our staff. At 10 pm, our provider staff consisted of 2 PEM attending physicians, one of whom had recently completed a master's degree in emergency management, a pediatric and a family medicine intern, a second-year pediatric resident and third-year EM resident, a PEM fellow, and a nurse practitioner. We had a full complement of nurses and ED techs, as well as available X-ray and CT techs, and respiratory therapists covering the hospital. Led by the attendings, the physicians and nurse practitioner quickly met to discuss plans and prepare for the incoming patients.

The initial concern was opening as many beds as possible. We first decided that the admitting pediatric floor teams should be contacted quickly to hasten the disposition of current patients. The PED charge nurse called charge nurses on the floors and the PICU. These services were made aware of the impending mass casualty event and were asked to receive current PED patients who were already dispositioned for admission but were still awaiting lab and imaging results or who had not finished treatments that would usually be performed in the PED. The request was well-received and enacted. At the time of the arrival of the first victim, just before midnight, we had cut the PED census to 9, clearing 55% of the beds occupied at initial notification, and were ready to receive up to 20 patients with our current resources.

Next, we determined that all present and available staff and ancillary services, including environmental services, hospital police, and a variety of therapists and techs would be needed. This included 12 nurses in the PED, but 3 had shifts ending at midnight, which was now the expected time of arrival of the first patients, delayed by the limited transportation availability and the scope of the disaster. Two of these nurses volunteered to stay if needed. Additionally, 3 off-shift nurses volunteered to come in. A respiratory therapist who would normally "float" throughout the hospital was stationed in the PED, and we had 3 technicians for prepping, and keeping one-to-one observation on patients at risk for self-harm or elopement. The nursing supervisor agreed more staff from within the hospital could be moved to the PED if necessary. Our PED was staffed to handle more patients than we had available beds.

The physicians and nurse practitioner decided to divide into "medical" and "tornado" teams. The PEM fellow and the third-year EM resident would lead the care of the injured patients. The family medicine intern, the second-year pediatric resident, and the nurse practitioner would primarily care for the incoming medical patients and all the remaining current patients. The two attendings would primarily focus on the injured victims of the tornado and provide guidance and supervision of all other patients as needed. PED staff coordinated with the adjacent adult ED about suggested surge preparation, resources, coordination of care, and

References available online.

potentially sharing resident physicians. Ultimately, this team formation and division of responsibilities proved successful, as each team was able to better focus on their patients.

Two of the anticipated trauma victims soon arrived. The first victim we received had a severe head injury and was intubated, followed 10 minutes later by another child with a severe head injury who was still alert and speaking, accompanied by his mother. The next 2 patients arrived in a single ambulance an hour later, with no family members located. They also had severe injuries but were able to tell us enough demographic information for our social worker to begin to locate family members. Of course, this was greatly hampered by the devastating conditions in Rolling Fork. The fifth and final patient arrived a few minutes later, before 3 am. Despite the dire predictions, we received 5 injured victims that night. Sadly, the child who received CPR was pronounced dead at the Yazoo emergency department.

Having a surplus of available rooms, increased staffing, and the optimized division of responsibilities, we were able to provide for each pre-existing and trauma patient without sacrificing quality care. At no point did our PED feel overwhelmed. We did not receive the number of patients we had feared, so we had more than enough resources and contingency plans. However, as we worked, we did recognize other preparations and procedures that could have been beneficial.

LESSONS LEARNED

Streamline policy: Per hospital policy, any patient admitted to the pediatric intensive care unit must be accompanied by a resident or fellow to provide a detailed verbal report to the awaiting PICU care team and a smooth transition of care. In one instance that night, both the fellow and the EM resident were transporting victims to the PICU simultaneously. Within this short time frame, 3 additional patients arrived. This left the single attending and the "medical" team with trauma patients requiring high levels of care and potential resuscitation, while also caring for all of the non- tornado victim patients. In retrospect, the PICU could have suspended this acceptance policy by having a PICU physician or nurse practitioner come to the PED to assume care and receive the patient report. Perhaps a report over speakerphone could be given the entire PICU care team after the patient and provider arrived.

Sign-out: Once the initial victims arrived, the pediatric and family medicine resident and the nurse practitioner noticed a significant issue. As the residents and both attendings began treating the trauma patients, the "medical team" realized they had not received adequate information for the patients previously under the care of the "tornado team" residents. Some of these patients did not have their PED course planned to disposition. Ultimately, all patients were provided quality care, although discussing plans with the attending physician during the victims' critical periods was difficult. This could have been improved at the initial staff meeting by discussing more thorough plans of care for each patient with all providers. This would have allowed all members of both teams to be informed of each patient. Additionally, this would have allowed more efficient care for all present patients.

Visual through-put: A third improvement would be the use of a large dry-erase board. We would include a row for each patient, with columns for a brief list of active conditions, needed labs, X-rays, CTs, other studies, and consults from the trauma surgery, neurosurgery, and orthopedic surgery, with check boxes to indicate if these tests had been ordered and resulted and which consults had been requested and completed. We would also indicate which patient needed these services the most emergently. This would greatly increase efficiency and limit multiple phone calls to these services. The consultants and techs could serve multiple patients during a single trip to the PED instead of being requested for individual patients multiple times. The consulting surgical subspecialties have responsibilities in both the pediatric and adult hospitals at UMMC, so the "batching" technique would be especially helpful to all.

EM TAKE-AWAYS

- Despite receiving multiple trauma victims who required care simultaneously, we found that our preparation was highly successful. This provided an efficient and stable environment in what otherwise could have been both overwhelming and potentially unsafe.
- Mass casualty situations frequently overwhelm a health care system. Although there were certainly areas for improvement, prior expertise, quick thinking, and teamwork-oriented care were keys to our pediatric emergency department's successful response.
- Simply meeting as a provider team to discuss a plan, working closely with nursing and ancillary staff, and putting the plan into action, led to good and effective care.
- Our hope is that through our experience of the Rolling Fork tornado, others can learn ways to better prepare for the next mass casualty event.

References available online.

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Sherrill Mullenix Residency Coordinator of the Year, **Tim Fullen, BS, CAP** | University of Tennessee Health Science Center

Faculty Mentor of the Year, **Adedamola Ogunniyi, MD** | Harbor UCLA Medical Center

Faculty Teaching Excellence Award, **Eric Shappell, MD, MHPE**

EMRA Chief Resident of the Year, **Courtney Cox, DO** | OhioHealth/Doctors Hospital

EMRA Resident of the Year, **Brittany Garza, DO** | Central Michigan University

EMRA Fellow of the Year, **Alexis Kimmel, MD** | University of Cincinnati

Academic Excellence Award, **William Mundo, MD** | Denver Health Medical Center

Augustine D'Orta Humanism Award, **Kristen Panthagani, MD, PhD** | Yale New Haven Health

Stephen Tantama, MD, Military Excellence Award, **Matt Christensen, MD, LT, MC, USN** | Naval Medical Center San Diego

Rosh Review "One Step Further" Award, **Garrison Nord, MD** | Yale New Haven Medical Center

Jean Hollister Contribution to Pre-Hospital Care Award, **John Robert Douds Jr.** | Sidney Kimmel Medical College at Thomas Jefferson University

Dr. Alexandra Greene Medical Student of the Year, **Matthew Ben Piechnik** | Sidney Kimmel Medical College

ACEP/CORD Teaching Fellowship, **Nolan Broussard, MD** | University of Chicago (alumni)

LAC Travel Scholarship, **Brittany Garza, DO** | Central Michigan University College of Medicine

EMRA/ACEP EDDA Travel Scholarship, **DaShawn Hickman, MD, PhD** | Harbor UCLA Medical Center

EMRA EMBRS Scholarship, **William Mundo, MD** | Denver Health Medical Center

EMRA/ACEP Medical Student Elective in Health Policy, **Sonia Patel** | University of Miami Leonard M. Miller School of Medicine

EMRA/ACEP Resident-Fellow Health Policy Elective, **Alberto E. Perez-Sthory, MD** | University of Pennsylvania EM Residency

EMRA/EDPMA Travel Scholarship, **Johnothan Smileye Jr., MD** | Harvard Affiliated EM Residency (Administration Fellowship)

EMRA

#EMRAAwardWinners

EMRA Leaders: Recognition and Respect

As EMRA celebrated a momentous year in 2024 and builds on that energy in 2025, we want to shine a light on the individuals behind it.
Please help us recognize our 2024 leadership core and welcome our 2025 leaders. These are the people whose work, vision, and collaboration form the unshakeable foundation of EMRA.

2024-2025 Committees

ADMINISTRATION & OPERATIONS COMMITTEE

2024:
Chair **Andrew Luo, MD, MBA**;
Chair-elect **Savanah Harshbarger, MD**;
Vice Chairs **Karan Bains**; **Puja Singh, MD**; **Neha Gupta, MD**;
Assistant Vice Chairs **Richard Ngo**; **Josue Minaya, MD**

2025:
Chair **Josue Minaya, MD**;
Chair-elect **Chad Nix, MD**;
Vice chairs **Jack Buckanavage, MD**; **William Boyer, MD**

CRITICAL CARE COMMITTEE

2024:
Chair **Dustin Slagle, MD**;
Chair-elect **Alexandra Hallagan, MD**;
Vice Chairs **Alex Lucas, MD**; **Gabriel Colon, MD**; **Josh Wild, DO**; **Luxi Qiao, MD**;
Assistant Vice Chairs **Marcus Gresham, MD**; **Kenneth Felsenstein, MD**; **Szymon Kutyla, MD**; **Dominique Gelmann, MD**

2025:
Chair **Alexandra Hallagan, MD**;
Chair-elect **Alex Lucas, MD**;
Vice Chairs **Josh Wild, DO**; **Alex Cavert, MD**; **Dominique Gelmann, MD**; **Luxi Qiao, MD**;
Assistant Vice Chairs **Yi Qin Zhu, MD**; **Pooja Dave, MD**; **Aarif Motorwala, MD**; **Jonathan Hanan, MD**

DIVERSITY & INCLUSION COMMITTEE

2024:
Chair **Daniel Ayorinde, MD**;
Chair-elect **Jessica Andre, MD**;
Vice Chairs **Katie Rodriguez, MD**; **Dante Robinson, MD**; **Janee Murray, MD**

2025:
Chair **Jessica Andre, MD**;
Chair-elect **Cathryn Phouybanhdyt, MD**;
Vice Chairs **Avery Ford, MD**; **Kelsey Newbold, DO**;
Assistant Vice Chair **Ravi Sandhu, MD**

EDUCATION COMMITTEE

2024:
Chair **Hannah Seyller, MD**;
Chair-elect **Kayla Basedow, MD**;
Vice Chairs **Katelyn Hagstrom, MD**; **Revelle Gappy, MD**; **Kendrick Lim, DO**; **Andres Lopez, MD**;
Assistant Vice Chairs **Dakota Burke, MD**; **Denise Manfrini, MD**; **Jasmeen Randhawa, MD**

2025:
Chair **Kayla Basedow, MD**;
Chair-elect **Allison Gasnick, MD**;
Vice Chairs **Kendrick Lim, DO**; **Christopher Thompson, DO**; **Andres Lopez, MD**;
Assistant Vice Chair **Ellis Jang, MD**

GOVERNMENT SERVICES COMMITTEE

2024:
Chair **Eric Kretz, MD**;
Chair-elect **Kyler Osborne, MD**;
Vice Chair **Michael Shulby, DO**;
Assistant Vice Chairs **Morgan Denecke, MD**; **Ryan Leone, MD**

2025:
Chair **Kyler Osborne, MD**;
Chair-elect **Evan Miller, DO**;
Vice Chairs **Ryan Leone, MD**; **Tyler McGuire, MD**;
Assistant Vice Chair **Christopher Heins, MD**

HEALTH POLICY COMMITTEE

2024:
Chair **Melanie Yates, MD**;
Chair-elect **Nicholas Melucci, DO**;
Vice Chairs **Stephanie Berg, MD**; **Phillip Groden, MD**; **Katarina Jones, MD, MPH**; **Alberto Perez, MD**;
Assistant Vice Chairs **Nicholas Nasser, MD**; **Angelica Rego, MD**

2025:
Chair **Nicholas Melucci, DO**;
Chair-elect **Alberto Perez, MD**;
Vice Chairs **Srikar Adibhatla, MD**; **April Huckleberry, MD**; **Parth Patel, MD**

INTERNATIONAL COMMITTEE

2024:
Chair **Sara Teising, MD**;
Chair-elect **Ekiomoado Olumese, MD**;
Vice Chairs **Vanya Zvonar, MD**; **Evan Miller, DO**;
Assistant Vice Chair **Kelly Mackenzie, MD**

2025:
Chair **Ekiomoado Olumese, MD**;
Chair-elect **Aditya Lal Vallath, IMG**;
Vice Chairs **Amera Hassan, MD**; **Gabriela Hernandez, DO**

PEDIATRIC EM COMMITTEE

2024: Chair **Rebecca Leff, MD**;
Chair-elect **Lauren Altomare, DO**;
Vice Chair **Ashley Sholmire**;
Assistant Vice Chair **David Surace (Iskandar), DO**

2025: Chair **Lauren Altomare, DO**;
Chair-elect **Hannah Miller, MD**;
Vice Chair **Alisha Agarwal, MD**

PREHOSPITAL & DISASTER MEDICINE COMMITTEE

2024:
Chair **Luke Wohlford, MD, MPH**;
Chair-elect **Shilpi Ganguly, MD**;
Vice Chairs **Matthew Lyons, DO**; **Elizabeth Mangin, MD**; **Sriram Venkatesan, MD**;
Assistant Vice Chairs **Kyle Essex, DO**; **James Nardini, MD**; **Alicia Stoebenau, DO**

References available online.

2024-2025 Committees

2025:
Chair **Shilpi Ganguly, MD**;
Chair-elect **Matthew Lyons, DO**;
Vice Chairs **Mohammad Abuakar, DO**; **Kyle Essex, DO**; **Elizabeth Mangin, MD**; **Sriram Venkatesan, MD**;
Assistant Vice Chair **Alicia Stoebenau, DO**

RESEARCH COMMITTEE

2024:
Chair **Charles Sanky, MD, MPH**;
Chair-elect **Anna Heffron, MD**;
Vice Chair **Michael Makutonin, MD**;
Assistant Vice Chairs **Laura Harding-Jackson, DO**; **Katrina Muraglia, MD**; **Jose Reyes, MD**

2025:
Chair **Michael Makutonin, MD**;
Chair-elect **David Gordon, MD**;
Vice Chairs **Emilia Clementi, MD**; **Laura Harding-Jackson, DO**;
Assistant Vice Chair **Lauren Eisner, MD**

SIMULATION COMMITTEE

2024:
Chair **Reed Colling, MD**;
Chair-elect **Elizabeth Yim, MD**;
Vice Chairs **Kyle Cohen, DO**; **Danier Ong, MD**;
Assistant Vice Chairs **Christian Casteel, DO**; **Salil Phadnis, MD**

2025:
Chair **Elizabeth Yim, MD**;
Chair-elect **Kyle Cohen, DO, NRP**;
Vice Chairs **Danier Ong, MD**; **Allison Smith, MD**

SOCIAL EM COMMITTEE

2024:
Chair **Molly Curtiss, DO**;
Chair-elect **Alessandra Della Porta, MD**;
Vice Chairs **Darrow Traylor, MD**; **Margaret Hayslip, MD**; **Phuong Vo, DO**;
Assistant Vice Chairs **Haley Furman, MD**; **Natalie McConnell, MD**

2025:
Chair **Alessandra Della Porta, MD**;
Chair-elect **Margaret Hayslip, MD**;
Vice Chairs **David Bronstein, MD**; **Mitchell Love, MD**; **Clara Keum, MD**

SPORTS MEDICINE COMMITTEE

2024:
Chair **Joshua Rayburn, MD**;
Chair-elect **Christian Holland, MD**;
Vice Chairs **Eric Swanson, MD**; **Teresa Crow, MD**;
Assistant Vice Chairs **Lauren Brady, MD**; **Benjamin Gold, MD**

2025:
Chair **Christian Holland, MD**;
Chair-elect **Jacquelyn Pearlmutter, DO**;
Vice Chairs **Ricky Gable, MD**; **Raymond Zhou, MD**

TECHNOLOGY, TELEMEDICINE, AND INFORMATICS COMMITTEE

2024:
Chair **Mitchell Blenden, MD**;
Chair-elect **Sidra Speaker, MD**;
Vice Chair **Ryan Korn, MD**;
Assistant Vice Chair **Austin Schoeffler, MD**

2025:
Chair **Sidra Speaker, MD**;
Chair-elect **Monique Arnold, MD**;
Vice Chairs **Avi Patel, MD**; **Alec Labadie, MD**

TOXICOLOGY COMMITTEE

2024:
Chair **Emanual (Mason) Jackson, MD**;
Chair-elect **Laura Szczesniak, MD**;
Vice Chair **Brittany Garza, DO**;
Assistant Vice Chairs **Katherine Markin, MD**; **John Michael Sherman, MD**; **Caroline Wright, MD**

2025:
Chair **Laura Szczesniak, MD**;
Chair-elect **Claire Seo, MD**;
Vice Chairs **Katherine Markin, MD**; **Hayn Jackson, MD**

ULTRASOUND COMMITTEE

2024:
Chair **Louisa Weindruch, DO**;
Chair-elect **Olivia Williams, DO**;
Vice Chairs **Sean Beckman, DO**; **Justine Milligan, MD**; **Ochan Kwon, MD**;
Assistant Vice Chairs **Ryan Bellinger, MD**; **Daniel Castro, MD**

2025:
Chair **Olivia Williams, DO**;
Chair-elect **Sean Beckman, DO**;
Vice Chairs **Ochan Kwon, MD**; **Daniel Castro, MD**; **Miranda Teixeira, MD**;
Assistant Vice Chairs **Ryan Bellinger, MD**; **Madeleine Silverstein, MD**; **Ayo Isola, MD**

WELLNESS COMMITTEE

2024:
Chair **Jessie Cable, DO**;
Chair-elect **Diana Gregoriou, MD**;
Vice Chairs **Melina Kanji, MD**; **Danielle Andrews, MD**;
Assistant Vice Chair **Kadeen Forrest, MD**

2025:
Chair **Diana Gregoriou, MD**;
Chair-elect **Coral Byrns, DO**;
Vice Chairs **Melina Kanji, MD**; **Sarah Beth Hanna, DO**

WILDERNESS COMMITTEE

2024:
Chair **Joseph Mueller, MD**;
Chair-elect **Jackson Anderson, MD**;
Vice Chairs **Jacques Lowe, MD**; **Mariko Ching, MD**; **Carlos Enciso Lopez, MD**;
Assistant Vice Chairs **Danusha Sanchez, IMG**; **Brian Legvold, MD**; **Erin Kinney, MD**; **Megan Barthels, MD**; **Ian Brodka, MD**

2025:
Chair **Jackson Anderson, MD**;
Chair-elect **Mariko Ching, MD**;
Vice Chairs **Kelly Dinh, DO**; **Olivia Chase, DO**; **Megan Barthels, MD**;
Assistant Vice Chairs **Hunter Stutz, DO**; **Erin Kinney, MD**

References available online.

2024-2025 Medical Student Council

EMRA MEDICAL STUDENT COUNCIL

2024:
Chair **Jinger Sanders**;
Vice Chair **David Gordon**;
Editor **Allison Gasnick**;
Legislative Coordinator **Kyle Avery**;
Mentorship Coordinator **Atira Shenoy**;
Osteopathic Coordinator **Kelly Dinh**;
Student Advising Coordinator **Zoee Castro**;
Web Technology Coordinator **Katie Tucker**;
AMA MSS Representative **Kailey Jacobson**;
AMA MSS Alternate **Danielle Sychowski**;
West Coordinator **Colton Crawford**;
Pacific Regional Representative **Ellis Jang**;
Southwest Regional Representative **Tushank Chadha**;
Midwest Coordinator **Mohammad Abuakar**;
Great Plains Regional Representatives **Thomas Johnston** and **Payton Wolbert**;
Northeast Coordinator **David Bronstein**;
Northeast Regional Representatives **Derick Goff** and **Breanna Sharp**;
Mid-Atlantic Regional Representative **Eli Brennan**;
Southeast Coordinator **Kristina Pagano**;
Southeast Regional Representatives **Babayode Bakare** and **Cristina Sanchez**;
South Central Regional Representative **Samantha Jacobson**;
International Representative **Nicole Ho-Sang**

2025:
Chair **Zoee Castro**;
Vice Chair **Atira Shenoy**;
Editor **Olivia Voltaggio**;
Legislative Coordinator **Seamus Vahey**;
Mentorship Coordinator **Linley Faith Dunn**;
Osteopathic Coordinator **Kentlee (Brogan) Crockett**;
Student Advising Coordinator **Kiersten Diercks**;
Content Coordinator **Benjamin Jacob DeYoung**;
Midwest Coordinator **Alexander Luke Alley**;
Northeast Coordinator **Derick Goff**;
Southeast Coordinator **Babayode Bakare**;
West Coordinator **Mary Pendleton**;
AMA-MSS Representative (2026) **Danielle Sychowski**;
AMA-MSS Representative (2027) **Matthew Nguyen**;
Pacific Representative **Jose Barrera**;
Great Plains Representatives **Peyton Victoria Lehman** and **Grey Braybrooks**;
Northeast Representatives **Ivana Re Baldie** and **Eli Brennan**;
Mid-Atlantic Representative **Catherin Allen**;
South Central Representative **Jeanette Hoang**;
Southwest Representative **Nada Khaled Mohamed**;
Southeast Representatives **Hannah Greene Hart** and **Kelsey Clabby**;
International Representative **Kwame Bell**

References available online.

2024-2025 EMRA Representatives to ACEP Sections & Committees

EMRA REPRESENTATIVES TO ACEP SECTIONS

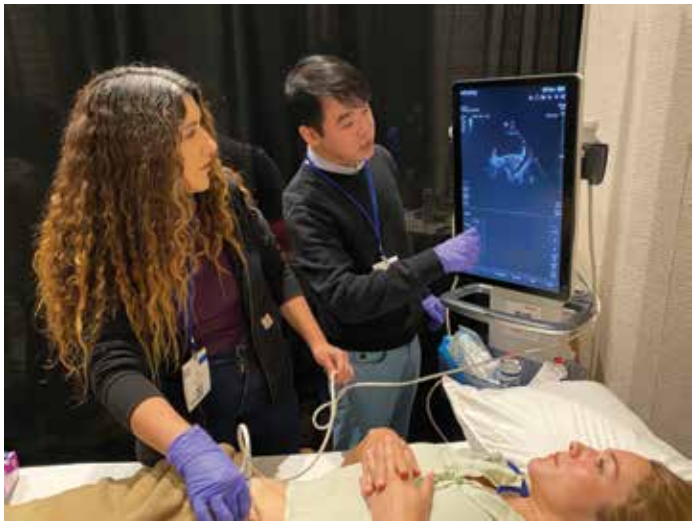
Air Medical Transport: **Kyle Essex, DO**
American Association of Women Eps: **Haely Studebaker, MD**
Careers in EM: **Derek Martinez, DO**
Critical Care Medicine: **Dustin Slagle, MD**
Cruise Ship Medicine: **Zachery Campbell, DO**
Democratic Group Practice: **Zachary He, DO**
Disaster Medicine: **Matthew Lyons, DO**
Diversity, Inclusion, and Health Equity: **Daniel Ayorinde, MD**
Emergency Medicine Informatics: **Michael Makutonin, MD**
EM Workforce: **Stephanie Berg, MD**
Emergency Telehealth: **Mitchell Blenden, MD**
Emergency Ultrasound: **Louisa Weindruch, DO**
EMS-Prehospital Care: **Kyle Essex, DO**
EM Practice Management and Health Policy: **Michael Dorritie, DO**
Event Medicine: **James Nardini, MD**
Freestanding Emergency Centers: **Catherine Scocca, DO**
Forensic Medicine: **Alicia Stoebenau, MECM, NREMT-P**
Geriatric EM: **Ryan Leone, MD**
International EM: **Sara Teising, MD**
Locum Tenens: **Taylor Sheppard, OMS-II**
Medical Directors: **Haely Studebaker, MD**
Medical Humanities: **Melina Kanji, MD**
Observation Medicine: **Brittany Garza, DO**
Pain Management and Addiction Medicine: **Juliet Corcillo, MD**
Palliative Medicine: **Nicholas Nasser, MD**
Pediatric EM: **Rebecca Leff, MD**
Research, Scholarly Activity, and Innovation: **Charles Sanky, MD, MPH**
Rural EM: **Kevin Molinar**
Sports Medicine: **Josh Rayburn, MD**
Social EM: **Alessandra Della Porta, MD**
Tactical EM: **Sriram Venkatesan, MD**
Trauma & Injury Prevention: **Janee Murray, MD**
Toxicology: **Mason Jackson, MD**
Quality Improvement and Patient Safety: **Katarina Jones, MD, MPH**
Undersea and Hyperbaric Medicine: **Emma Rogers, MD**
Wellness: **Jessie Cable, DO**
Wilderness Medicine: **Joseph Mueller, MD**
Young Physicians: **Jess Adkins-Murphy, MD**

EMRA REPRESENTATIVES TO ACEP COMMITTEES

Academic Affairs: **Nastassjia deBourbon, MD**
Bylaws: **Anna Heffron, MD, PhD**
Clinical Emergency Data Registry/EMDI: **Michael Dorritie, DO**
Clinical Policies: **John Thompson, MD**
Coding and Nomenclature Advisory: **Alexa Golden, MD**
Communications: **Jess Adkins-Murphy, MD**
Disaster Preparedness and Response: **Lindsay Davis, DO**
Diversity, Equity, Inclusion: **Tanesha Beckford, MD**
Education: **Abagayle Bierowski, MD**
Ethics: **Derek Martinez, DO**
EM Practice: **Andy Hung-Yi Lee, MD, MBA**
EMS: **Kelly Tillotson, MD**
Federal Government Affairs: **Kenneth Kim, MD**
Finance: **Jessica Adkins-Murphy, MD**
Health Innovation Technology: **Ryan Korn, MD**
Medical-Legal: **Lauren Rosenfeld, MD**
Membership: **David Wilson, MD**
Pediatric EM: **Alexandria Georgadarellis, MD**
Public Health & Injury Prevention: **Nia Rush, MD**
Quality and Patient Safety: **Joseph Brooks, MD**
Reimbursement: **Carly Rosen, MD**
Research: **Monique Arnold, MD**
State Legislative/Regulatory: **Hannah Gordon, MD, MPH**
Wellbeing: **Leyanet Gonzalez, MD**

EMRA REPRESENTATIVES TO ACEP TASK FORCES

ACEP ED Accreditation Task Force: **Blake Denley, MD**
Urgent Care: **Stephanie Strong, MD**
Artificial Intelligence: **Michael Makutonin, MD**; **Sidra Speaker, MD**; **Mitchell Blenden, MD**



References available online.

50 years EMRA's

"It's so important for residents to have their own organization. We really believed that and worked for it. To see it all 50 years later is really something."

EMRA's most well-known founding father, Joe Waeckerle, MD, shared perspective and wisdom at the 50th Anniversary Gala in Las Vegas in October, topping off a celebration of 5 decades of supporting trainees.

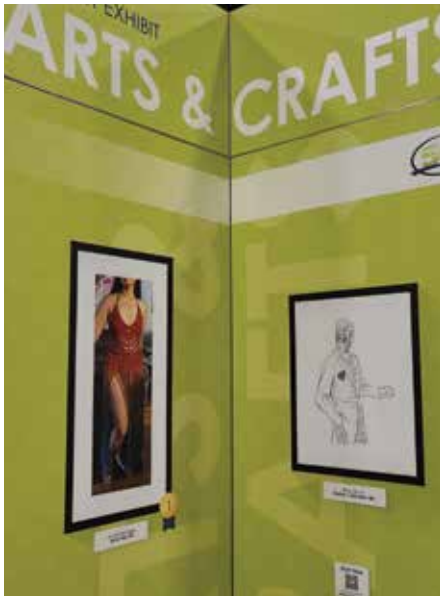
In 1974, Dr. Waeckerle was among a small group of young doctors dedicated to a fledgling specialty who, over drinks at a bar in Dallas, sketched out the framework of an organization for residents – run by residents.

"EMRA was founded to give residents their own voice," Dr. Waeckerle said. "We didn't know what would happen, but we knew we needed to have a say in things."

The 50th anniversary celebrations in Las Vegas featured everything from a star-studded gala to a special Hall of Honor, EMRA Art Competition, EMRA Store, and all of EMRA's leadership, education, and networking events.

References available online.

50th Anniversary



Welcome, EMRA Board!

The Representative Council elected new EMRA Board members in October, and the outgoing leaders met up with the new class to pass the baton. Please thank and welcome (from left) incoming AMA Representative **Anna Heffron**, MD, PhD; outgoing Medical Student Council Chair **Jinger Sanders**; outgoing Director of Health Policy **Kenneth Kim**, MD; Secretary of the Board/EM Resident Editor **Morgan Sweere**, MD, MPH; Speaker of the Council **Jacob Altholz**, MD; Director of Education **Joe-Ann Moser**, MD, MS; outgoing Speaker **Michaela Banks**, MD, MBA; President-Elect **Polly Wiltz**, DO; EMRA President **David Wilson**, MD; Immediate Past-President **Blake Denley**, MD; Director of Leadership Development **Grace Bunemann**, DO; outgoing Director of Leadership Development **Derek Martinez**, DO; Director of Health Policy **Katarina Jones**, MD, MPH; Vice Speaker of the Council **Ian Brodka**, MD; EMRA Representative to ACEP **Kelsey Morgan**, MD; outgoing EMRA Representative to ACEP **Aaron Kuzel**, DO, MBA.

References available online.

50 years EMRA's

Scenes & Spotlights

We hosted a packed week, and EMRA's best showed up. Huge congratulations and thanks to everyone who took part in our feature events in Las Vegas.

Help us recognize:

Case-con Student Category

Winner: David Gordon - A Silent Threat: Basilar Artery Occlusion Presenting as Prodromal Hemisensory Deficit
Runner-up: Payton Wolbert - An Electrifying Connection: A Case of Atrio-Esophageal Fistula

Case-con Resident Category

Winner: Mumin Mushtaq Ahmed Hakim, MD - Sore throat after a generalized tonic clonic seizure: Level 5 to a level 1 scenario!
Runner-up: Thomas Rauser, MD - Blue Baby Blues: Post-Circumcision Acquired

References available online.

Scenes & Spotlights

Methemoglobinemia

SimWars Medical Mystery Sleuths

University of Florida-Jacksonville (2024 champions)
Einstein Medical Center Philadelphia
HCA Aventura Hospital
Rutgers NJMS
Massachusetts General/Brigham
University of Central Florida/HCA Osceola
UCSF Fresno
Texas Tech University HSC El Paso
Thanks to our sponsor, Laerdal.

References available online.

50 years EMRA's

Scenes & Spotlights

EMRA Quiz Show: Board Prep Shenanigans

Albert Einstein Medical Center (2024 champions)
Central Michigan University
George Washington University
Staten Island University Hospital
Stanford Health Care

References available online.

50th Anniversary

Scenes & Spotlights

EMRA hosted a juried Art Gallery. Congratulations to People's Choice Winner Rafael J. Carrillo Torres, MD, University of Puerto Rico.

Visit www.emra.org/art-gallery to see all submissions.
Visit https://emra.smugmug.com/EMRA-at-ACEP24 for more photos from our 50th anniversary.

References available online.

Scenes & Spotlights

References available online.

I Was the Attending, but My Residents Were More Experienced

Masood Mohammed, DO, MS
Abrazo Health

Being a new attending in a department with senior residents who have practiced the same amount of time or even longer than yourself is a unique experience. Growing into this role is not without its challenges — or its rewards.

Emergency Medicine residency teaches us to be a jack of all trades. From managing critical patients to leading traumas and running codes, we enter residency prepared for nothing and leave ready to manage almost anything.

Or so I thought.

Although I felt prepared to become an attending and supervise residents, one thing I never learned in my three-year EM residency was how to manage fourth- and fifth-year residents. Yet this was the situation I found myself in at my new program which included an EM categorical residency as well as EM/IM and EM/FM combined residencies. Mere weeks after being the confident, well-versed third year resident in my previous department, I again felt like a very small fish in a large, unfamiliar pond.

There was plenty of discomfort, awkwardness, and uncertainty at the outset. Although it took practice and some inevitable mistakes, over time I learned, I grew, and things improved.

Along the way I garnered many pieces of wisdom that made these circumstances not only better, but enjoyable. For the new attending in the new department who finds themselves in a similar situation, here are some of the tips that helped me:

1. TRUST YOUR TRAINING.

You may not have learned how to be *this* attending in residency, but you did learn how to be *the* attending. You have already spent thousands of hours learning how to navigate difficult and strenuous situations, so the tools are already in your kit. One of the glories of Emergency Medicine is that sometimes even if the destination is unknown, the path is always discoverable. Lean on all the hard work you have already put in...that diligence is what enabled you to matriculate into this position in the first place.

2. BE TEAM-ORIENTED.

No one succeeds in the emergency department alone. Residents, regardless of their years of experience, will still look to their attending in tenuous moments, and we should be able to look to them as well. The location of certain equipment or the steps of a protocol may be foreign to you, but the resident who has worked in the department for four or even five years is a treasure trove of information. In addition to this, relying on your residents conveys a willingness to trust them and can deepen the all-important attending-resident relationship. You can be successful together, not despite one another.

3. CONNECT THROUGH YOUR CONFUSION.

Expertise within a busy and ever-changing ED does not come overnight. This is a notion that should be highlighted, not feared. Use these gray areas as an opportunity to introduce yourself to nurses, techs, clerks, and other staff and inform them that you are new to your position! The number of times someone stopped what they were doing to assist and orient me was refreshing and appreciated. More importantly, it allowed me to learn names and form connections with the people I would be working with and relying on day in and day out.

4. BEFRIEND YOUR RESIDENTS.

The best attending-resident relationship is one built on trust. While a lot of trust can be built within the department, this should not be the only place it is fostered. Attending resident conferences, journal club, and other social outings can break down many of the unnecessary hierarchical ideals that residents may sometimes experience. These events can create interconnectedness between two *people*, not just two *doctors*. The best interactions I have had with residents came after investing time getting to know them so that they felt more comfortable coming to me for help managing a patient or for general life advice.

5. THERE IS ALWAYS A TEACHABLE MOMENT.

Even residents who have more years of experience than you have not experienced everything that Emergency Medicine has to offer. Perhaps during residency you did a unique rotation at a critical access center, or you have used different equipment than what is offered at this institution, or you have simply employed alternative treatments for a condition than what the resident is proposing. All of these are teachable moments that help the resident grow and expand their knowledge while giving you reassurance in your teaching skills.

Being a new attending in a department with senior residents who have practiced the same amount of time or even longer than yourself is a unique experience. Growing into this role is not without its challenges, and there will undoubtedly be bumps along the way. Nonetheless, opening oneself up to this new experience and being eager to grow alongside these learners can make for happiness, healthiness, and the utmost job satisfaction.

References available online.

A Deep Dive with Scott Weingart, MD, FCCM

Morgan Sweere, MD, MPH
EMRA Board Secretary
EM Resident Editor-in-Chief
University of Florida — Jacksonville

empower
Sharing Our Stories

This edition of EMPower interviews ED critical care guru, Dr. Scott Weingart!

Scott Weingart, MD, FCCM

Dr. Weingart received his medical degree and completed a residency in Emergency Medicine at the Mount Sinai School of Medicine. He then went on to fellowships in Trauma, Surgical Critical Care, and ECMO at the Shock Trauma Center in Baltimore. He is a professor of emergency medicine at Nassau University Medical Center and an adjunct professor at the Icahn School of Medicine at Mount Sinai. In addition, Dr. Weingart is a physician coach concentrating on the promotion of eudaimonia and optimal performance. He is best known for his podcast on Resuscitation and ED Critical Care called the EMCrit Podcast, which has been downloaded > 100 million times.

What is your favorite thing about emergency medicine?

There aren't too many professions where just by showing up to work, you're doing good for the world. Our job has intrinsic good. We are providing a service that allows us to make the world a better place.

References available online.

empower
Sharing Our Stories

What is your favorite thing about emergency medicine?

There aren't too many professions where just by showing up to work, you're doing good for the world. Our job has intrinsic good. We are providing a service that allows us to make the world a better place.

If you were restarting residency, what advice would you give yourself?

If anything was going on in the department, no matter where I was assigned, I was in the mix. Getting involved makes a huge difference, but not everyone in the department can do this, or the department will come to a halt. Find the balance of being involved in the critical cases while not abandoning your assignments and responsibilities

What is the best career insight that you want to pass along?

One of the key factors to career longevity that often falls off as we progress is to have fun. With coaching, I get to take people who are already astounding in their career and make them better. Usually, the better is in terms of their enjoyment and appreciation of our amazing job. Emergency medicine has the most potential to have the 8-12 hours we are on shift be just pure joy. While we think the barriers to that ideal are systemic, what I find in coaching is that they are actually self-imposed. Go to the bathroom, drink and eat, pop outside for 60 seconds while calling a consult – keep it fun! You have far more agency in that regard than you think.

What is the most important trait for a leader to have?

The kind of leader I always wanted to be was "the innovator." Figuring out new and better ways to do things. However, this must be balanced with real care and compassion for the team members. Otherwise, it is brutal and relentless.

What is one thing you have learned from failure?

You can't take it too seriously. Mistakes are the best teacher. You can either get it right, or you can learn, and those are great options.

What is your best time management tip?

I've kept with a key precept from *Getting Things Done* by David Allen, which is that your mind should be for creating and making connections, not remembering things. There are external processes that can keep up with those things, like schedules and to-do lists. But there is complexity in how to manage these systems and the investment of 8-10 hours learning an excellent management style will pay off for decades.

Favorite chief complaint:

Altered mental status/unresponsive

What is the most significant risk you have taken and the outcome

Career-wise, doing critical care when there was no certification and no guarantee that there would be any jobs. Thanks to those who have fought for the certification pathway, it is a totally accepted pathway now. Either way, I'm so glad I made that choice.

Share 2 things that are on your desk right now.

- A skull, because of the stoic practice of memento mori (remember death). We are all mortal. Consider that each day could be our last, so live it. Let silly things go. Don't waste time.
- A really nice fountain pen. I believe in this time of insanse technology, we must harken back to the history of innovation as well!

What is the best on-shift snack?

- Free pizza or doughnuts, but these days I try not to indulge, but gosh they taste better for being grabbed on-shift.
- Something fatty and high protein. I'm a big fan of having mindful meals or snacks on shift. Take time to eat for 5 minutes.

What is the most recent book you read?

Beautyland by Marie-Helene Bertino. It's about a girl who is convinced she is an alien and follows her throughout her life.

What are you learning about right now?

Learning is a full-time job. What I'm currently exploring is whether vasopressin could be a push or bolus dose.

What is your favorite song to hype you up before a shift?

"Paint It, Black" by the Rolling Stones

What message would you pass along to the readers of *EM Resident*?

Remember how you were as a trainee when you become an attending. Trainees are not supposed to know better. They don't have the same understanding and knowledge as you do now, on day one.

Remember that even when it gets dark, this is the best job, and we are so lucky to be doing it. If you start having a more high-level view that most of the world would trade places with you in a heartbeat, that will give you a better perspective.

References available online.

Pregnant in Residency: An Act in Three Parts

Ellen Shank, MD
Emergency Medicine Chief Resident, PGY-3
University of California Davis Emergency Medicine

Despite my protruding second-trimester bump, I had never exited a patient's room so quickly.

As a second-year EM resident working in the pediatric ED, I had assigned myself to a 4-year-old with a chief complaint of dysuria, as he had a history of a vesicorectal fistula leading to frequent urinary tract infections in his short life. Walking into the room, I found an uncomfortable-appearing child in a brown Winnie the Pooh T-shirt, with a concerned mother at bedside. We chatted about his presentation (2 days of fever and general discomfort), and then I tugged off his shirt to reveal vesicles in various stages spreading across his chest.

My heart quickened as I peered over him and noticed the rash popping up on his hands, arms, legs. I quickly excused myself from the room, giving thanks under my breath that I was still wearing an N95 despite the institutional expiration of the mask mandate. After relaying this story and my concern to my co-resident, they agreed without hesitation to take over the care of this likely varicella case, and I spent my post-shift hours that day reading up on vertical transmission.

It is a tricky thing to balance an unborn child's wellness with the needs of the patients walking into the ED. Throughout those 9 months, the fear of nosocomial infection and uncertainty in navigating choices surrounding maternity leave, counterbalanced with the warmth and kindness of colleagues, attendings and patients made my first pregnancy a profoundly confusing and heartwarming journey.

I remember sitting in the ergonomic spinning chair at my program director's office for my maternity leave planning meeting, wondering if I was asking too much. While most PDs make informal adjustments as needed, most EM programs do not have formalized scheduling policies for their pregnant residents.[1] I was 8 weeks into this journey – exhilarated, terrified, nauseous. Out of the 20 annual templated schedules, there was exactly 1 that would allow for the flexibility of 2 months that did not require coverage during the dates of my predicted due date, ensuring — to the extent that I could control it — that none of my co-residents would have to step in to cover my absence. My program director responded, to my immense relief, "Choose whatever schedule you want, and we'll make it happen."

Several months later, another memorable interaction — chief complaint: testicular pain. He was in his mid-50s, with a neat plaid shirt and a backwards-facing trucker hat. As I entered the room, he beamed. "You're having a boy! I can tell, just by the way you're carrying." Turns out he had 10 children of his own, with 5 grandchildren and counting. He was here for STI screening after an episode of unprotected sex. The trick: to balance charitable feelings toward this man and listen humbly to his parenting advice while performing a genital exam, without

References available online.

tipping the scales toward condescension. With gusto, pantsless, he narrated the early stories of his children: the thrill of a swelling belly, the sacred act of cutting the umbilical cord, the early, milky, sleepless nights. Bashfully yet steadily, my excitement for parenthood continued to grow with each memory he recounted.

Fast-forward to my 35th week of pregnancy, as I waited to board a flight back home after a medical conference. Before leaving on the trip, I made sure to check with my OB/Gyn — an intern whom I worked with as a senior in the ED — in order to obtain the required letter confirming my ability to fly until week 36. The prior night, I could not sleep after a full day of business meetings, subsequent socials, and 6 miles of walking through the city. A deep, persistent pain in my lower abdomen spurred my first visit to emergency OB triage, halfway across the country from my husband and family. As I boarded an Uber at 0300 from the conference hotel to a nearby hospital, the gentleman driving gave his standard introduction but quickly became silent. After a long 5 minutes, he blurted out, "Ma'am, I don't mean to be rude, but are you about to have a baby?!" I chuckled softly, "I really hope not." For the next few hours, I laid in OB triage hooked up to a tocometer and Doppler, trying and miserably failing to get comfortable. The contractions mercifully petered off after some fluids and rest. The nurses were incredibly kind, and cautioned me about the risks of dehydration and overexertion. I bid them goodbye and caught a ride back to the hotel to finish out the conference in the morning, taking with me a lesson in the physiologic changes of pregnancy learned the hard way.

It is no small wonder that studies show pregnant residents working long hours and nights may be at higher risk for depression,[2] hypertension,[3] spontaneous abortion,[4] and preterm birth.[5] **After years of pushing the limits, accustomed to our ability to cram in more than we believe is possible, the very real physical constraints of pregnancy are shocking.** How do we hold these two realities in tandem – the jealous demands of a residency schedule, with the flourishing of new life and a new role as a mother? Hospital versus home? Patients versus family?

All told, I had a relatively easy pregnancy journey. My co-resident husband and I benefited from a short commute, financial stability, and grandparents nearby to ease our childcare burden. Thanks to the work of our resident union, I also had the option to take a full 2 months of maternity leave prior to returning to work, delaying graduation by just 2 weeks in favor of more time with the little one in early postpartum days. These privileges challenge my preconception of the mutual exclusivity of the roles of resident and mother, pushing role balances toward equilibrium, allowing space for patients *and* family, hospital *and* home.

I pondered all these things as a summer rain was falling lightly outside O'Hare; it looked like a christening of the tarmac and of this beautiful city. I breathe in, I breathe out. I am grateful for my calling to medicine. I am grateful for my husband, family, and friends. I am grateful for frequent fetal kicks. And, more than ever before, I am grateful to be headed home.

References available online.

Viewpoint: A Preferential Option for the Poor in the Emergency Department

Phillip Jordan, MD
Eastern Virginia Medical School Emergency Medicine

To be a border physician is to be confronted daily with the human consequences of a failed immigration policy. Among the patients we treat are migrants who have fallen from the border wall, migrant victims of assault — by both security forces and the cartel, often indistinguishable — and migrant patients hit by vehicles while running the gauntlet of highways on the other side of the border wall.

Our migrant patients suffer from the same exacerbations of chronic diseases characteristic of any emergency department (diabetic ketoacidosis, hypertensive urgency, and more) but also from tropical diseases such as malaria and dengue. Sometimes, they are accompanied by Border Patrol or other law enforcement officers. Other times, they have found their way to our emergency department, seeking help. What unites all of these patients is their preventable suffering. The wall fall patient is the most emblematic of these injuries, but all of the conditions for which they seek help have been caused or exacerbated by restrictive immigration policies that raise the cost for seeking a safe future for yourself and your children.

These patients we treat in El Paso are perhaps the most vulnerable in the country. Often they lack any sort of access to health care. Sometimes, they present to our emergency department without any legal documentation. And when they do, they are often accompanied by law enforcement; the staff of our emergency department may be the first non-law enforcement Americans they encounter.

As a volunteer at many local migrant shelters, I often find myself in the position of referring patients to my own emergency department. The emergency department is unique in that it is perhaps the only health care location where a version of "universal care" in the United States can be guaranteed. The idea that we treat all comers equally, regardless of ability to pay, was something that drew me to this specialty.

What do we owe these patients? The answer is simple: We owe them everything. Dr. Paul Farmer, founder of Partners in Health, had a profound insight:

"Diseases…make a preferential option for the poor. That is, the poor are sicker than the non-poor. They are at heightened risk of dying prematurely, whether from increased exposure to pathogens or from decreased access to services…"

Partners in Health was founded on this insight: If diseases make a preferential option for the poor, then health care must too. For the migrant patients I send to the ED, this is perhaps the only chance they will get to receive quality health care. In the emergency department, I can order any lab that I want and consult any specialist I want. If there is any patient who deserves a million-dollar workup, it is the migrant patient.

Your patients in the ED — the unhoused, the destitute sick — also deserve this sort of work-up.

If you or I were treated in the department — resident physicians with ample access to health care — we would have countless other opportunities to seek follow-up care if our issues weren't resolved. Our destitute patients do not have that luxury.

As emergency medicine residents, we are often driven to "move the meat," to dispose of and discharge patients as quickly as possible. But "efficiency cannot trump equity in the field of health and human rights" (Paul Farmer). So many of our patients are victims of political pathologies. The first step to undoing the damage is treating these patients with dignity and the highest standard of care.

This paradigm will undoubtedly be controversial to cash-strapped hospitals and EDs. Our specialty has long operated as a band-aid for the policy-driven effects we see daily. Instead of being the societal band-aid, we must reframe our role to truly encompass that of the healers we swore to be. That starts by reframing our understanding of these patients not as "meat to move," but as dignified human lives who deserve nothing less than the best of care, regardless of citizenship status or ability to pay.

References available online.

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EMRA ECG Challenge

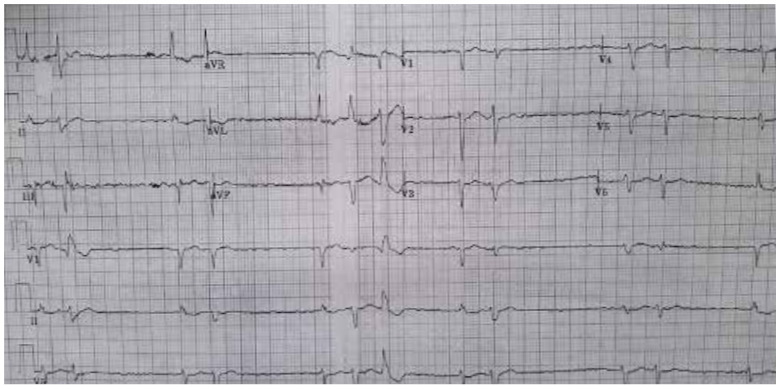
Matthew Hysell, MD
Medical Student Program Director, Research Director
Corewell Health Lakeland Emergency Medicine Residency

Elizabeth Nisper, MS4
Central Michigan University College of Medicine

Jeremy Berberian, MD
Associate Director of Emergency Medicine Resident Education, Department of Emergency Medicine, ChristianaCare

CASE

A 65-YEAR-OLD FEMALE WITH A HISTORY OF ATRIAL FIBRILLATION PRESENTS TO THE EMERGENCY DEPARTMENT WITH ALTERED MENTAL STATUS, WEAKNESS, AND DECREASED ORAL INTAKE.



WHAT IS YOUR INTERPRETATION OF HER ECG?

ANSWER ON PAGE 59

References available online.

ECG Challenge

ANSWER:

This ECG shows an irregular rhythm with an average ventricular rate of 70 bpm, no obvious p-waves or flutter waves, left axis deviation, and variable QRS complex duration. There are 2 and 3-beat groups of QRS complexes with the QRS complex duration increasing between beats except for the 5th group which does the opposite. The RR intervals between the 1st, 3rd, 5th, and 8th QRS complexes are somewhat consistent but then vary for the remainder of the ECG.

The presence of repeating groups of beats should prompt consideration of a 2nd degree AV block, but this is ruled out by the absence of p-waves. Other possibilities include a junctional rhythm with PJCs or PVCs, atrial fibrillation with PJCs or PVCs, and hyperkalemia.

The presence of a slow rhythm suggests decreased or blocked AV node conduction, and the frequent premature beats suggests increased automaticity. Given the patient's history of atrial fibrillation, these findings are concerning for digoxin toxicity.

In general, atrial fibrillation is not an intrinsically slow rhythm in the absence of a diseased or poisoned AV node. Digoxin works as a negative chronotrope by increasing the vagal effects at the AV node to slow conduction. Too much digoxin can lead to severe bradydysrhythmias and AV blocks. The initial portion of this ECG looks like atrial fibrillation with an underlying regular junctional rhythm suggestive of a 3rd degree AVB, which is one of the "classic" dysrhythmias seen with digoxin toxicity. Digoxin also works as an inotrope by increasing intracellular calcium, which can lead to increased automaticity and the presence of frequent premature beats as seen in the case ECG.

Digoxin toxicity can present with a range of bradycardias, tachycardias, and various blocks. Common dysrhythmias include paroxysmal atrial tachycardia with variable block and bradycardic rate, accelerated junctional rhythms, bidirectional ventricular tachycardia, and atrial fibrillation with slow ventricular response or a regular bradycardic rate due to the presence of a 3rd degree AV block.

Common causes of chronic digoxin toxicity include renal disease, electrolyte abnormalities, dehydration, and drug interactions. Common symptoms seen with chronic toxicity include anorexia, abdominal pain, nausea, and CNS symptoms (e.g., AMS).

Digoxin effect, also called the "Salvador Dali" sign, describes the ECG features seen with digoxin use and include scooped ST-segments, prolonged PR intervals, and shortened QT intervals. Note that these ECG changes are seen with digoxin use and are not a marker of toxicity. It is also important to note that patients can have digoxin toxicity clinically with normal serum concentrations, especially in the presence of hypomagnesemia, hypokalemia, and/or hypercalcemia.

CASE CONCLUSION

The patient's labs were notable for an acute kidney injury and elevated digoxin level. Treatment with hemodialysis was initiated due to the severity of the patient's renal dysfunction and concurrent digoxin toxicity. Typical first-line treatment for symptomatic dysrhythmias is digoxin immune Fab (eg, Digibind or DigiFab). This will cause potassium to shift into the cells and can lead to clinically significant hypokalemia, so potassium levels should be monitored closely after administration. It will also cause serum digoxin levels to rise, making repeat levels unreliable.

DIGOXIN LEARNING POINTS

General Features

- Includes cardiac glycosides (e.g., digoxin and digitalis derivatives, foxglove, oleander, lily of the valley, and secretions from *Bufo* toad spp.)

EKG Features

- Digoxin effect seen with therapeutic levels:
  - Scooped ST-segment most pronounced in leads with tall R-wave (digoxin effect)
  - PR lengthening
  - QT shortening
- Toxicity can cause almost any dysrhythmia, including:
  - Atrial fibrillation with slow ventricular response or a regular bradycardic rate
  - Paroxysmal atrial tachycardia with variable block and bradycardic rate (uncommon)
  - Bidirectional ventricular tachycardia (very rare)

Clinical Significance

- Acute toxicity may present with hyperkalemia
- Treatments include:
  - Digoxin-specific antibody
  - Atropine for AV blocks
  - Phenytoin or lidocaine for ventricular dysrhythmias

References available online.

Board Review Questions

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1. A 78-year-old patient presents with abdominal pain radiating to the back, bloody stool, and hematemesis with near-syncope. Medical history includes hypertension and surgical grafting for abdominal aortic aneurysm repair. Their vital signs include BP 71/53, P 115, R 16, and T 37.4°C (99.3°F). On examination, a palpable abdominal mass and central abdominal tenderness are noted. What is the most appropriate next step?

A. Administer a proton pump inhibitor and a somatostatin analogue
B. Request a gastroenterology consultation for urgent endoscopy
C. Request a surgery consultation for an emergent surgical procedure
D. Transfuse type-specific blood products when available and then reassess

2. An 86-year-old woman presents with a 6-month history of fatigue, lethargy, increasing confusion, constipation, poor appetite, slowed speech, and dyspnea. Her vital signs include BP 105/60, P 55, and T 33.5°C (92.3°F); SpO2 is 91% on room air. She has bilateral pleural effusions and nonpitting peripheral edema. What is the most likely drug-induced etiology of this presentation?

A. Amiodarone
B. Diltiazem
C. Flecainide
D. Procainamide

3. Which laboratory test result is the gold standard for diagnosing gout?

A. Absence of bacteria in aspirate
B. High uric acid levels in serum
C. Negatively birefringent crystals on aspirate
D. WBCs less than 2,000 and PMNs less than 25% on aspirate

4. Which finding from the history and physical examination raises the most concern that a patient has an alcohol use disorder?

A. Horizontal gaze nystagmus on examination
B. Inappropriate sexual behavior after two drinks
C. Multiple alcohol-related injuries in the past year
D. Slurred speech and an unsteady gait at triage

5. A 74-year-old woman presents with an anterior shoulder dislocation after a fall. Her vital signs include BP 86/40 and P 110. Intravenous access is established, and a fluid bolus results in mild improvement in her blood pressure. She has no allergies. Which medication is preferred for procedural sedation to reduce the dislocation?

A. Fentanyl
B. Ketamine
C. Midazolam
D. Propofol

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ANSWERS
1) C. 2) A. 3) C. 4) C. 5) B

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