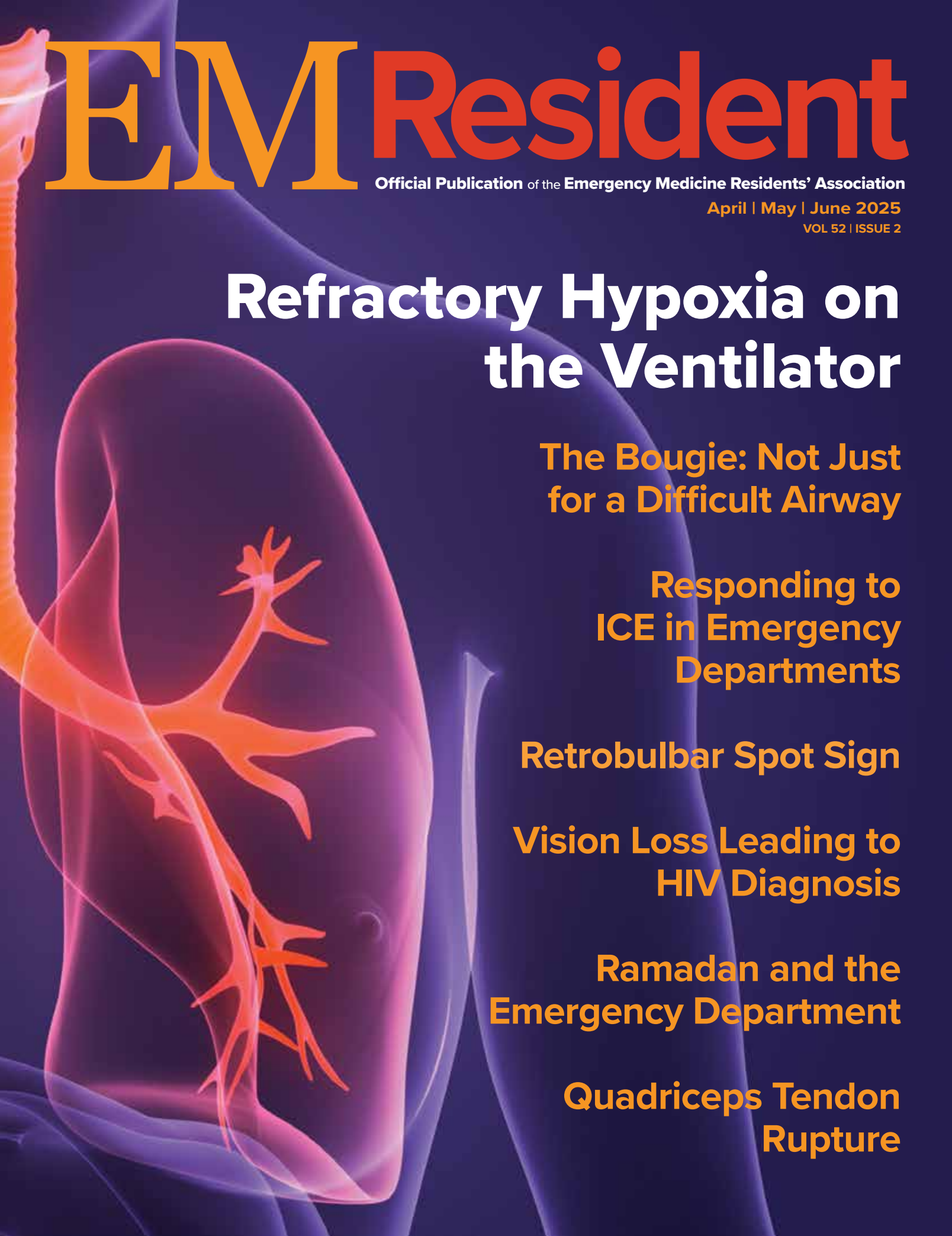

EM Resident

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Refractory Hypoxia on the Ventilator

The Bougie: Not Just for a Difficult Airway

Responding to ICE in Emergency Departments

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Ramadan and the Emergency Department

Quadriceps Tendon Rupture

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New Kid on the Track

Your skill alone is the only thing that should be necessary to convince anyone of anything. But in many situations, that's simply not the case. It can be hard to be the new kid on the track.

Through a muddy tear-off on the front of your helmet, under bright Saturday night lights on a remote race track in Florida, you see a petite blonde girl in her 20s hustle up to the window and peek her head in, asking questions about your pain and injuries after a crash.

"Who is this girl?" you might think to yourself, as racing safety crew are predominantly middle-aged males, often fire safety or EMS in their background.

Who is this new kid on the track?

I serve on weekends as part of the safety crew at a local dirt track, responding to wrecks and working to improve safety in motorsports. When I first began, there were occasionally questions about who I was, why I was there, and what my experience was with racing. When I would look at a driver sitting in a car on the track and tell them their axle was broken after a crash, they would occasionally look at the teenage male tow truck driver behind me for confirmation that I was correct. Over time, my attitude, demeanor, work ethic, and medical skill started to convince them that I knew a little bit about what I was talking about and could be trusted.

I probably don't look like what some may picture as the typical emergency medicine physician. I definitely don't look like what most race car drivers or pit crew may picture as a race track safety crew member or physician that they expect to come up to their car. I think that's totally OK.

Many of you may have had similar experiences, when you don't look like what others perceive you "should." They may have asked you inappropriate questions about your education, training, age, culture, or other aspects. It can be difficult in those instances to have to be questioned about these things because of your appearance.

There may not be any malice in the question, but it still impacts you. It can make you feel like you need to convince someone of your position in the department and your competence. My take on the situation would be this: Your skill alone is the only thing that should be necessary to convince anyone of anything. You don't need to look any specific way, certainly not to fit a cookie cutter image of an emergency medicine physician. **I look like an emergency medicine physician, because I am one.**

When I first began working as a tech in a small community emergency department, I learned quickly the camaraderie that exists between the team members in the emergency department. I often felt like I was the new kid at school early on. There was already a pre-determined culture between the nurses, techs, and physicians. Luckily, I happened to be in a department that was close-knit and tough but friendly. I felt similarly entering a new department for the first time as a resident. One of your main goals aside from patient care is to get the nurses and techs to like, trust, and enjoy working with you.

Whether you are the brand new medical student, first year intern, or newly minted attending entering a different ED for the first time this year, it can be intimidating to walk into the environment. It can be really difficult to infiltrate that environment, prove your skill, work ethic, and communication, and begin to be trusted and accepted by other staff.

It's not easy to be the new kid on the track. Stick to what you know, and be confident in your education. These attributes will shine through and carry through by the way you take care of your patients. You look like an emergency physician, and you are one.

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Residency Is Short—Make It Count

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

Ask any attending about their residency experience, and they will tell you the same thing: ***It was hard, intense, and flew by in an instant.***

In the midst of the constant grind of training, it may not feel like it's flying by. But as my PGY-3 year draws to a close, it feels like just yesterday, I was celebrating my own match day.

Residency is one of the most challenging yet rewarding periods of our careers. It demands our time, our energy, and our emotional resilience. It often feels like residency takes more than it gives. Many of us have heard, and perhaps even repeated, the sentiment that residency is something we just need to "get through." To some extent, that's true. If you simply strap yourself in for the ride, you'll emerge on the other side with the skills and knowledge to practice emergency medicine independently.

But should that be the goal?

There is so much more to this journey than survival. Residency is an opportunity — not just to learn medicine, but to shape the kind of physician, leader, and person you will be for the rest of your career. The decisions we make during this time — how we train, the people we surround ourselves with, and what values we uphold — are the building blocks of our future. Equally important, we must remember we are not *just* residents. We are people, with identities and passions that exist outside of the emergency department. Holding onto those things, nurturing them, and making intentional choices about how we spend our time can make the difference between looking back on residency as a period of exhaustion or one of growth.

PROTECT YOUR PASSION

Being an emergency physician is an incredible job, but even the most passionate can burn out. We see humanity at its most vulnerable, where trauma, pain, and suffering are part of our daily routine.

To protect your passion for EM, nurture the things that remind you why you chose this path in the first place. Maybe that means working with underserved populations, getting involved in advocacy, or teaching medical students. It could be taking time to celebrate the small wins — like the patient who thanks you for listening, the team that comes together for a flawless resuscitation, or the moment when a junior resident gains confidence under your guidance.

Just as important, invest in your passions *outside* of medicine. Residency may feel all-consuming, but it's essential to maintain a sense of identity beyond your job. Whether it's music, sports, art, travel, or simply spending time with family and friends — don't lose sight of what makes you, *you*.

SAY YES TO GROWTH

Residency is designed to challenge and push us outside our comfort zones. Growth comes from stepping outside our comfort zones. It requires us to say *yes* to trying something new, like the procedures that intimidate us or the leadership roles we're not sure we're ready for. Residency is the opportunity to do everything in a (somewhat) controlled setting.

Residency is already hard, so it's easy to avoid additional difficult opportunities. Once you are the attending, you may not have any backup. It is much easier to fall back to the level you trained than to hope you are ready to rise to the occasion. However, it requires additional discomfort during residency. The cases that scare you now will be the ones you handle effortlessly in the future — if you take the opportunity to learn from them today.

FIND YOUR PEOPLE

Residency is too hard to go through alone. No matter how strong or independent you are, you need a support system. Your co-residents will be the only people who truly understand what you're going through. The shared experience of long shifts, difficult cases, and late-night debriefs over PSB (post-shift beers — breakfast also acceptable) creates bonds that last a lifetime.

But beyond your residency class, seek out mentors who inspire you. Find attendings whose clinical practice, teaching style, or career path resonates with you, and learn from them. Medicine is an apprenticeship and the people we surround ourselves with influence the kind of doctors we become.

SHAPE THE FUTURE OF EMERGENCY MEDICINE

Residency isn't just about individual growth; it's about shaping the future of our specialty. The landscape of emergency medicine is shifting, and not always for the better. Job market concerns, workforce saturation, and the growing influence of corporate medicine — these are not distant problems that will be solved by someone else. They are our reality and our responsibility.

If you care about the future of EM, get involved. Advocate for fair working conditions. Mentor the next generation of medical students and residents. The future of our specialty is being written *now*, and we have the power to influence it.

DON'T JUST SURVIVE — THRIVE

It's easy to fall into the mindset of just making it through the next shift, the next week, the next in-service exam. But if we spend all of residency just trying to survive, we'll look back and realize we missed the moments that mattered.

Take a breath. Celebrate your wins. Buy champagne even though you had RBCs in your last LP. Learn from your losses, and know that you are doing your best. Invest in your growth, your relationships, and your well-being. Residency is short — *make it count*.

References available online.

EMRA CRITICAL CARE ROAD BLOCKS

Refractory Hypoxia on the Ventilator

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Special thanks to John Gaillard, MD, Associate Professor of Anesthesiology and Emergency Medicine at Wake Forest University School of Medicine, and Andrew Petrilli, MD, Assistant Professor of Anesthesiology at Wake Forest University School of Medicine and Assistant Program Director of the Wake Forest Critical Care Medicine Fellowship, who are both emergency medicine-trained intensivists practicing critical care in the Cardiothoracic ICU as well as a mixed Medical/Surgical ICU. Dr. Gaillard additionally works in the ED at Wake Forest Baptist Medical Center. Thanks also to EMRA Critical Care Committee Chair Dustin Slagle, MD, who reviewed this paper.

SOME PEOPLE USE MNEMONICS (LIKE DOPES) TO GUIDE THEIR INITIAL ASSESSMENT OF THE HYPOXIC PATIENT ON THE VENT. DO YOU USE A SPECIFIC STRUCTURE LIKE THIS OR WHAT IS YOUR INITIAL APPROACH IN EVALUATING SUCH PATIENTS?

Gaillard: I first evaluate O2 sat, is it a good signal, is the patient actually hypoxemic? Then I evaluate the patient, the endotracheal tube (ETT), and the ventilator. In terms of the patient, I listen for breath sounds, watch their breathing for signs of asymmetry (pneumothorax or severe atelectasis) or hyperexpansion (bronchospasm or breath stacking). I suction the ETT to ensure patency. I then look at the ventilator, focusing on things like ensuring everything is connected, my vent numbers (specifically I look at the mode and settings, the FiO2, the patient's peak and plateau pressures, and tidal volumes including comparison of exhaled vs. inhaled tidal volume (looking for air leak)).

Petrilli: I like to approach it by ruling out everything but the lungs first. You can use mnemonics like DOPES; I tend to start at the ventilator and work my way towards the patient, asking myself, "Is this a mechanical problem (tube dislodgement or obstruction, circuit kink, equipment problem, etc.), sedation issue, obstructive problem (bronchospasm, breath stacking, mucus plug), etc.? Once I've ruled out everything else, I focus on the lungs themselves and what I can do about sick lungs getting sicker.

WHAT DIAGNOSTIC TESTS DO YOU GET INITIALLY TO GUIDE MANAGEMENT?

Gaillard: I like to throw the ultrasound probe on the chest to look for things like pneumothorax, consolidations, right heart strain, and globally assess cardiac function. I will also get a chest X-ray to evaluate for lung pathology and check ETT position. If one lung is completely whited out, I consider mucus plug and that patient may need chest PT and a bronch. If the lungs look perfectly clear, I start to think about pulmonary embolism or other causes of shunt physiology. If the lungs show worsening pneumonia or ARDS, the problem may simply be that your patient's lungs are getting sicker.

Petrilli: I agree with the chest X-ray and ultrasound as well as a good physical exam. With regards to getting an ABG specifically, I think a baseline ABG can be helpful to establish that it is correlating well with the pulse ox, especially for patients in which pulse ox may be less reliable like those with darker skin tones. I do not believe in monitoring serial ABGs, however. It is painful and more time consuming in patients without an arterial line, and I think that monitoring VBGs and the pulse ox works just as well.

WHAT INFORMATION ARE YOU GETTING FROM THE VENTILATOR WHEN ASSESSING THE HYPOXIC PATIENT?

Gaillard: Of course I'm looking at the mode and settings first and any ventilator alarms. Then I'll look at peak and plateau pressures. If tidal volume (TV) is set, is the patient reaching that? What is the exhaled TV compared to inhaled TV (is there an air leak)?

Petrilli: I'm looking at the pressures needed to overcome airway resistance and oxygenate and ventilate lung

References available online.

units with respect to the patient's lung compliance. If peak pressures are >40 cm H2O, that indicates that the ventilator may be utilizing an excessive amount of pressure to overcome pathologic airway resistance or poor lung compliance. The inspiratory hold maneuver described below helps delineate problems with resistance versus compliance.

WHAT VENTILATOR MANEUVERS CAN YOU TRY TO EITHER TROUBLESHOOT OR CORRECT REFRACTORY HYPOXEMIA?

Gaillard: You should know how to do an inspiratory and expiratory hold. The inspiratory hold shows your plateau (alveolar) pressure (goal is <30 cm H2O to reduce risk of lung injury). High plateau pressures can indicate too much PEEP or tidal volume, air trapping, patient-ventilator dyssynchrony, or poor compliance. The expiratory hold will allow you to calculate intrinsic ("auto") PEEP. Auto PEEP indicates breath stacking which can occur with obstructive lung disease such as asthma or COPD, or other causes of air trapping like mucus plugging or inappropriate vent settings (too high TV or too short exhalation time).

Petrilli: The inspiratory hold can help you determine if there is a lung parenchyma problem or an airway resistance problem in the case of high pressure alarms. If the plateau pressure and the peak pressure are both high, this most often indicates a lung compliance issue (the sick lungs themselves are the problem). High intra-abdominal pressure can also cause an elevated plateau pressure via transmission of pressure via the diaphragm, so that is one confounder of which to be aware. If the plateau pressure is low or normal with high peak pressure, this indicates a resistance problem (e.g., mucus plug, patient biting tube or other tube issue, bad obstructive disease).

In the past there was a push towards recruitment maneuvers such as "40 for 40" (increasing PEEP to 40 mmHg for 40 seconds). More recent data have demonstrated that this actually leads to worse outcomes, particularly in moderate to severe ARDS, so I try to stay away from those. The current American Thoracic Society (ATS) guidelines on management of ARDS also recommend against recruitment maneuvers.

DO YOU TYPICALLY DISCONNECT THE VENTILATOR IN YOUR TROUBLESHOOTING PROCESS?

Gaillard: Some people do this to differentiate between a ventilator issue and an ETT or patient problem. I personally think you can get the same information from the ventilator graphics and numbers themselves. If the graphics are concerning for significant auto PEEP, I will consider disconnecting the patient from the vent.

DOES ANY VENTILATOR MODE ITSELF SHOW A BENEFIT OVER OTHERS IN REFRACTORY HYPOXEMIA?

Petrilli: There is no good evidence for this. A lot of it is institution-dependent or personal preference, but no mode has been shown to improve outcomes over others. Some individuals are big proponents of airway pressure release ventilation (APRV) and I think this can be a good mode if you know how to use it and titrate it (this is not a "set it and forget it" type of mode), but there isn't good evidence for true benefit over other modes at this point. Personally I feel that if you are going to use it, it should be started early on in the treatment course of severe disease and not relegated to a "rescue" mode.

ONCE YOU'VE RULED OUT OTHER CAUSES BESIDES SICK LUNGS, WHAT IS YOUR STEPWISE APPROACH TO REFRACTORY HYPOXIA IN ARDS?

Petrilli: My practice is generally consistent with the most recent ATS ARDS guidelines. A basic foundation to managing these patients is to ensure lung-protective ventilation with low tidal volumes (6-8 cc/kg or even 4-6 cc/kg ideal body weight) and low airway pressures (maintaining plateau pressure <30 cm H2O and driving pressure < 15 cm H2O). Systemic corticosteroids are also controversial but currently recommended for all patients with ARDS regardless of disease severity, with the DEXA-ARDS trial supporting early use.

The simplest way to start addressing hypoxia itself is to titrate PEEP and FiO2. The updated 2023 ATS ARDS guidelines now recommend a high PEEP strategy in patients with oxygenation and recruitment difficulty (using the high PEEP table from the ARDSNet trial). If you have esophageal manometry available, you can use this to estimate pleural pressure and thus calculate transpulmonary pressure and more accurately titrate PEEP. If you don't have this technology, you can sequentially increase the PEEP while checking plateau pressure via the inspiratory hold maneuver — if you're increasing PEEP and your plateau pressure is still <30 cm H2O and driving pressure < 15 cm H2O, this can indicate that you are recruiting more lung units to address hypoxemia. This just needs to be done with caution, as excess PEEP can cause hypotension due to increased intrathoracic pressure and right ventricular dysfunction due to increased pulmonary vascular resistance.

The next things I consider if the patient remains hypoxemic are patient positioning and synchrony. The PROSEVA trial showed that prone positioning provides a mortality benefit in patients with moderate to severe ARDS (PaO2/FiO2 [P/F] ratio <150) by improving V/Q matching. Prone positioning is especially beneficial in patients with dependent atelectasis and posterior disease when supine. I prone patients for 16 hours at a time when hypoxemia does not improve with titration of PEEP and FiO2 as above (i.e. PaO2/FiO2 remains below 150).

I will then trial temporary paralysis in patients with severe ARDS or those with dyssynchrony on the vent despite adjustments to sedation. The ACURASYS trial compared 48-hour paralysis with cisatracurium to heavy sedation and demonstrated a comparative mortality benefit with paralysis in patients with severe ARDS. The more recent Rose Trial, however, compared neuromuscular blockade to light sedation in severe ARDS patients and showed no benefit

References available online.

of paralysis. Because of the conflicting evidence this is a bit controversial, but I tend to trial paralysis in severe ARDS patients that remain dyssynchronous and have poor compliance despite heavy sedation. If their lung compliance improves with the paralytic trial, I will start them on a cisatracurium infusion for 48 hours or until their pulmonary mechanics and hypoxia improve.

Another option to consider is use of inhaled pulmonary vasodilators (e.g., epoprostenol [Flolan], nitric oxide, milrinone). Current evidence does not support any mortality benefit or reduction in number of days on the ventilator with these agents, but they do improve oxygenation by helping to reduce ventilation-perfusion mismatch and are low-risk medications. Given this, many clinicians will still try them in patients with refractory hypoxemia. I think the patient population that may most benefit from these agents are patients with pulmonary hypertension at baseline or right heart failure.

Gaillard: I typically walk through a similar stepwise approach of considering a change in ventilator mode, increasing FiO2 and PEEP while being mindful of peak and plateau pressures, proning, starting an inhaled pulmonary vasodilator (understanding that this improves oxygenation without improving mortality), trialing heavy sedation, and paralyzing if needed.

AT WHAT POINT ARE YOU CONSIDERING V-V ECMO AND IN WHICH PATIENTS?

Petrilli: Once I've tried all the strategies above, if the patient is still severely hypoxic, I consider V-V ECMO based on evidence (though mixed and difficult to interpret) from the EOLIA and CESAR trials. The Extracorporeal Life Support Organization (ELSO) guidelines recommend consideration of V-V ECMO in patients with potentially reversible respiratory failure refractory to conventional treatments who have: refractory hypoxemia (P/F <80) or severe hypercapnic respiratory failure (pH <7.25 with PaCO2 ≥60 mmHg). ELSO and ATS additionally recommend that patients should be early in the ARDS course (on ventilator for <7 days), which I generally agree with, though if the patient is young with reversible disease I will sometimes extend this window.

Gaillard: If all of the other strategies fail, I consider V-V ECMO in patients with reversible disease and without significant comorbidities who have been on the ventilator for <7 days. The RESP score can be used to predict survival after ECMO for patients with severe respiratory failure and can also be helpful in determining whom to cannulate.

Bursting at the Seams:
Blunt Trauma Causing Open Globe Rupture After Prior Ocular Surgery

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Central Michigan University

Sarah Harris-Gendron, MSW
Central Michigan University

Derek Schaller, MD, FACEP
Central Michigan University

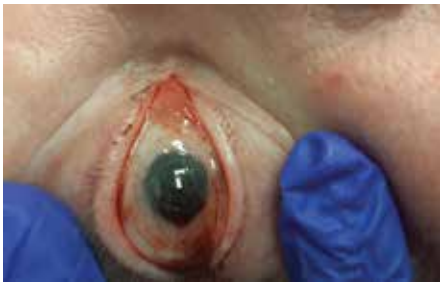

Image 1a. The patient's right eye, anterior view, upon arrival to the ED

Image 1b. The patient's right eye, lateral view, upon arrival to the ED

In the ED, there should be a high degree of suspicion for open globe injury in patients with significant blunt force trauma to the eye, especially in individuals with a history of prior ocular surgery.

CASE

A 79-year-old man with a history of prior bilateral corneal surgeries presented to the ED via EMS for evaluation of eye pain and vision loss. The patient reported he fell forward from a chair at home, striking the right side of his face against a coffee table, and subsequently experienced complete vision loss in his right eye.

On examination, there was a laceration on the right eye at the temporal edge of the limbus, anisocoria, and right-sided proptosis. Images of the patient at time of initial examination are represented in **Images 1a** and **1b**.

Patient factors including limited mobility prevented the use of a slit lamp to examine the eye. Instead, a Wood's lamp examination with fluorescein staining was obtained at bedside and a positive Seidel test was observed, as depicted in **Images 2a** and **2b**.

Tonometry was deferred due to our high degree of suspicion for open globe injury (OGI). Differential diagnoses included conjunctival laceration, scleral injury, corneal laceration, OGI, retrobulbar hematoma, and intraocular hematoma.

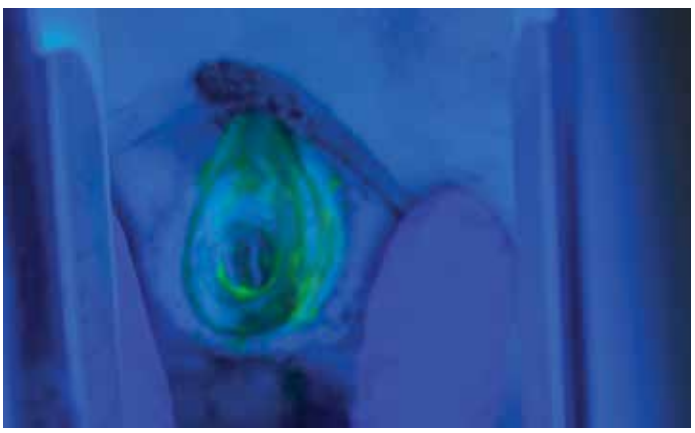

Image 2a. The patient's right eye, anterior view, under fluorescein staining through Wood's lamp in the ED

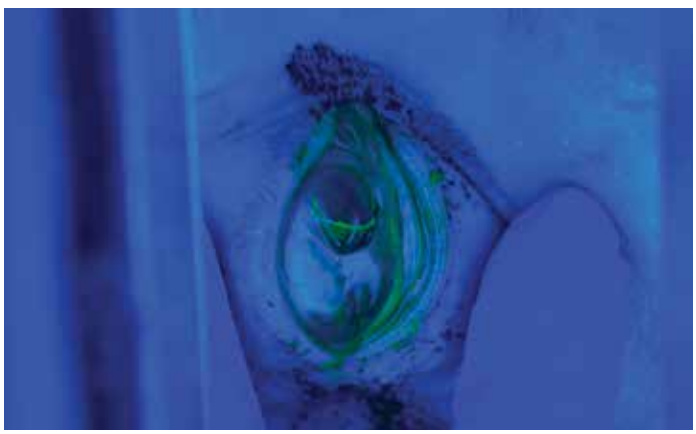

Image 2b. The patient's right eye, temporal view, under fluorescein staining through Wood's lamp in the ED

References available online.

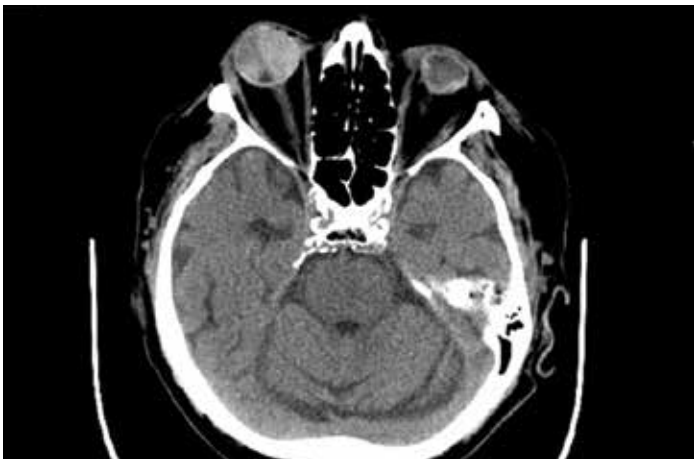

Image 3a. Transverse view of the CT non-contrast of the head demonstrating evidence of intraocular hemorrhage of the right eye

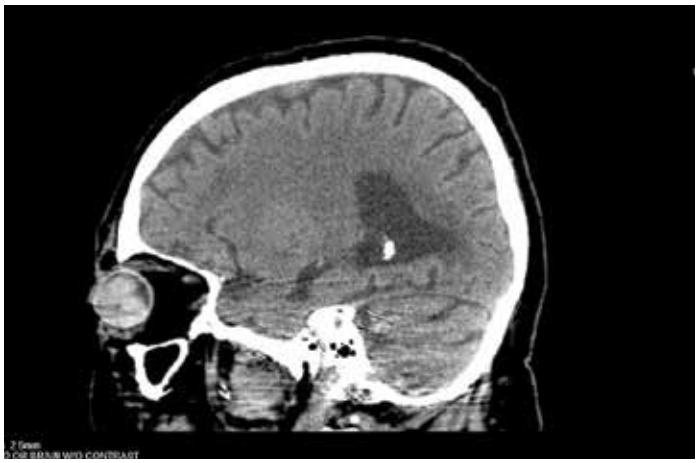

Image 3b. Sagittal view of the CT non-contrast of the head demonstrating evidence of intraocular hemorrhage of the right eye

Computed tomography (CT) of the head and neck was obtained, and key images are shown in **Images 3a** and **3b**. CT showed intraocular hematoma, but no retrobulbar hematoma was identified. No lateral canthotomy was indicated to relieve the proptosis. Additionally, no intracranial or bony calvarial injury was identified, and no other traumatic injuries requiring acute management were discovered. Empiric antibiotic therapy with IV cefazolin and Tdap was initiated. A makeshift eye shield was fashioned from a styrofoam cup, cut to fit the patient's facial contour, and placed over the affected eye.

Our facility did not have an on-call ophthalmologist available, and the patient was promptly transferred to a quaternary care center for further management.

DISCUSSION

OGI refers to a full thickness injury of the eye, involving the cornea, sclera, or both.[1] The Birmingham Eye Trauma Terminology (BETT) system is used to define types of eye injuries. OGIs are further categorized as a rupture, referring to a full-thickness injury of the eyewall by a blunt object, or a laceration, referring to a full-thickness injury of the eyewall caused by a sharp object due to penetration, perforation, or intraocular foreign body.[1]

OGIs have the highest incidence among young adult males, usually occurring in those 30-40 years of age.[2-7] However, there is also a smaller peak in incidence in elderly females, especially those 70-89 years of age.[2,6,7] The cause of open globe injury varies with age.[4-6] In younger age groups, globe laceration is more likely to result from assault, workplace injuries (e.g., working with metal), and motor vehicle accidents.[4-6] In older age groups, globe rupture resulting from falls accounted for the majority of injuries.[2-7] Of those with falls causing globe rupture, the majority of patients have a history of previous intraocular surgery.[2,4,6]

OGIs often result in loss of visual acuity. Studies have found globe ruptures to be associated with visual acuity <20/200 in up to 60% of patients.[7,8] Globe ruptures can also result in evisceration or enucleation.[7] Endophthalmitis is another complication of OGI, which occurs mostly from lacerations rather than ruptures.[2,7,9] Primary repair within 24 hours, intraocular tissues prolapse, and self-sealing of wounds were found to decrease the risk of development of endophthalmitis.[9] Intravitreal antibiotics and corticosteroids remain controversial for the prophylaxis of endophthalmitis, but vitrectomy is recommended.[9]

MANAGEMENT

In the ED, there should be a high degree of suspicion for OGI in patients with significant blunt force trauma to the eye, especially in individuals with a history of prior ocular surgery.[10] Patients with suspected OGI should have minimal manipulation of the eye during the ocular examination in the ED. Definitive management is surgical intervention. In preparation for surgery, a protective shield should be placed over the affected eye, tetanus status should be updated, and prophylactic antibiotics should be administered with coverage for *Staphylcoccus aureus*, *Bacillus* species, and *Pseudomonas* species. First-line antibiotics are cefazolin 1g IV or vancomycin, plus a 4th-generation fluoroquinolone.

CASE CONCLUSION

Review of external records revealed that after transfer to the receiving facility, the patient was evaluated by the ophthalmology team and promptly taken to the OR. Operative evaluation confirmed there was full thickness globe injury via dehiscence of a former keratoplasty, as well as lens dislocation. The wound was reapproximated, and subconjunctival injections of vancomycin, ceftazidime, and dexamethasone were administered.

The patient was admitted and treated for elevated intraocular pressures with acetazolamide as well as timolol and atropine drops. The patient's course was complicated by choroidal hemorrhage, for which he underwent a vitrectomy, and then was discharged. At a 2-month follow-up visit, visual acuity in the affected eye without correction was noted to be hand motions.

References available online.

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Differential Beyond Deep Vein Thrombosis for Unilateral Leg Swelling:

A Case of May-Thurner Syndrome in the Emergency Department

When patients present with unilateral leg swelling, a few top differential diagnoses come to mind. Most of these patients, at least at our shop, obtain ultrasound imaging during the triage process. Since almost 1 million cases of deep vein thrombosis (DVTs) are diagnosed each year in the United States, it can be an easy diagnosis to make at the door.[3] However, when the ultrasound is negative, how does your differential diagnosis evolve? We present a case of an elderly female with unilateral leg swelling that was diagnosed with symptomatic May-Thurner syndrome.

CASE REPORT

A 93-year-old female with a medical history of dementia, aortic stenosis status post transcatheter aortic valve replacement (TAVR), hypertension, hyperlipidemia, and a known pancreatic mass of unknown etiology presented to the emergency department with 10 days of left leg swelling. She reported pain only with palpation. She had been able to ambulate without difficulty. She denied any trauma, recent falls, recent travel, shortness of breath, or chest pain. She reported mild relief with elevation of her extremity. She had been compliant with her medications – one of which was a diuretic – and had no recent changes in her medications. She denied any personal or family history of clotting or bleeding disorders.

Her initial vital signs revealed a temperature of 97.8°F, HR 66 bpm, BP 143/62, RR 18 breaths per minute, and 97% saturation on room air. She was well-appearing on exam, in no acute distress, with pertinent positives of chronic venous insufficiency changes to her bilateral lower extremities, more pronounced on the left along with asymmetric pitting edema, increased on

References available online.

the left. She had no cardiac murmurs and her breath sounds were clear without rhonchi or wheezing. Her abdomen was soft, non-tender, and non-distended.

She was found to have quite unremarkable complete blood count and basic metabolic panel except for a bicarbonate of 33 mmol/mL. Other notable findings were hemoglobin of 13.4 g/dL, sodium of 142 mmol/mL, creatinine of 0.88 mg/dL, NT-proBNP 10,707 pg/mL (normal range of those greater than 75 years old of 0-1,800 pg/mL), and d-dimer of 631 ng/mL DDU (d-dimer unit) (normal age adjusted range less than 243 ng/mL DDU).

Imaging was obtained and a left lower extremity ultrasound revealed no evidence of DVT, a left-sided Baker's cyst, and lower extremity edema. A computed tomography (CT) of the abdomen/pelvis with intravenous contrast revealed extrinsic compression upon the left common iliac vein by the right common iliac artery compatible with May-Thurner syndrome without visible thrombus of the left iliac or common femoral vein, severe atherosclerosis of the abdominal aorta and iliac arteries, along with associated asymmetric left lower extremity edema. Incidental findings on her CT imaging revealed trace bilateral pleural effusions and a 2-centimeter probable cystic pancreatic mass.

Vascular surgery was consulted given symptomatic findings of classic May-Thurner syndrome. After discussion of management options and consideration of her age, history of dementia, and relatively mild symptoms, her family opted for more conservative management including lower extremity elevation and compression stockings. She was able to ambulate in the ED without difficulty, given strict return precautions, and ultimately was discharged home.

DISCUSSION

May-Thurner syndrome, also known as iliac compression syndrome or Cockett syndrome, involves extrinsic compression of the left common iliac vein by the right common iliac artery against the lower lumbar spine.[2] It is typically diagnosed in female patients aged 25-50 years old. It is a diagnosis to consider in those with recurrent DVTs or those with asymmetric leg swelling without signs of DVTs on ultrasound as it has a prevalence of 20%.

Although initially thought to be genetic, it is thought to occur secondary to chronic irritation of the pulsatile iliac artery against the common iliac vein resulting in venous spurs.[3] These spurs increase the risk of blood clots. Risk factors for this syndrome include female sex, severe dehydration, and scoliosis. They also include risk factors for hypercoagulability such as prolonged travel, recent surgery, immobilization, oral contraceptive use, and pregnancy. It is important to note that May-Thurner syndrome may or may not have blood clots at the time of diagnosis.[1]

May-Thurner syndrome is typically diagnosed on CT venography. As clots more commonly occur in the iliofemoral junction, ultrasound of the legs can be negative and ultimately miss the diagnosis. The gold standard diagnosis is contrast venography with transvenous pressure measurements.[1] However, this typically only occurs pre-operative prior to vascular therapy.

Management includes several different modalities. Typical management is with full dose anticoagulation. Thereafter, consideration for thrombolysis or thrombectomy of the clot can be considered. Stent placement may occur with this based upon evaluation by interventional radiology or vascular surgery. Conservative management includes elevation of the extremity and compression stockings.[5]

DIFFERENTIAL

When considering unilateral leg swelling, it is important to not anchor on a diagnosis of DVT. DVTs are common, but it is also important to assess the patient and look for skin changes that may clue the clinician into diagnoses such as phlegmasia alba dolens or phlegmasia cerulea dolens, as these increase the acuity of the condition. These syndromes are caused by a large clot burden that can lead to an ischemic limb. Skin changes are also seen in cellulitis; however, this usually includes systemic symptoms as well. Cellulitis of the lower extremity will be associated with a source such as a wound of the feet or legs. In middle-aged to elderly patients, consider osteoarthritis or gout if the swelling overlies a joint. Leg swelling may be from a more systemic condition as well, such as heart failure. Although heart failure typically is symmetrical and bilateral, some patients may have unilateral, asymmetric leg swelling. Finally, as in our patient, we were most concerned for abdominal cancer/metastases. Diseases within the pelvis that can compress blood flow to the legs are important to consider as it may be missed on initial examination and work-up.

TAKE-HOME POINTS

Although May-Thurner should be included in your differential diagnosis of unilateral leg swelling, be sure to keep in mind other causes as well, as the case is not always a DVT.

Our case was unique in several aspects.

First, she is the oldest patient per literature review with a diagnosis of May-Thurner syndrome. The second oldest was diagnosed at the age of 91 years old.[2]

Second, visualization of her May-Thurner syndrome was seen on CT scans since 2009 but was not interpreted as such by radiology, and she remained asymptomatic during that time.

Third, her daughter had been diagnosed with May-Thurner syndrome while she was in her 50-60s. This is not thought to be a hereditary disease.

Our patient had an exceptional case of May-Thurner syndrome given her unique aspects and few risk factors for predisposition.

References available online.

Nausea, Vomiting, and Tachycardia. Oh My!

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Nausea and vomiting are major chief complaints in the emergency department, accounting for around 2.5 million annual ED visits.[1] While the majority of these cases are due to innocuous etiologies such as gastritis, GERD, etc., EM physicians are trained to rule out the potential life-threatening causes such as acute coronary syndrome (ACS), aortic dissection, diabetic ketoacidosis (DKA), increased intracranial pressure, esophageal ruptures, small bowel obstruction, appendicitis, torsion, and ectopic/ruptured ectopic pregnancies. In the pediatric population, the differential also includes things such as pyloric stenosis, intussusception, and non-accidental trauma (NAT). When considering cardiac causes of nausea and vomiting in young adults, ACS would be quite uncommon, and most would not routinely include congestive heart failure (CHF) high on the differential, making it an important talking point.

CASE SUMMARY

An 18-year-old otherwise healthy female presented with a chief complaint of nausea and vomiting. These symptoms had been ongoing for the past 2-3 weeks, along with intermittent abdominal pain. Additionally, she reported multiple episodes of emesis per day and could barely keep down water at that time. When she presented to the ED, she mentioned that she lost 30 pounds over the past 2-3 weeks. She denied any chest pain, shortness of breath, fevers, or chills. Her last menstrual period was 3 weeks prior. She was sent in by her primary care physician for further evaluation of her vomiting and weight loss due to the duration of her symptoms.

Initial vital signs noted tachycardia of 130 bpm, blood pressure (BP) of 85/33, pulse ox of 100% on room air, respiratory rate of 18, and she was afebrile at 98.1°F. Initial EKG showed sinus tachycardia of 116 bpm. On the initial physical exam, the patient was noted to be tachycardic. She also had bilateral 1+ edema of the legs to the knees. Her lungs were clear, and her heart was tachycardic but regular. Her abdomen was soft, non-distended, and non-tender.

Notably, during the patient's ED stay she never complained of chest pain or shortness of breath. She attributed her tachycardia, nausea, and vomiting to being anxious at the time.

Resuscitation was started with 2L of 0.9% NS and anti-emetics as noted below. The patient was placed on a cardiac monitor. IV access was obtained, along with labs including a septic workup. Blood work revealed a CBC with normal hemoglobin and a normal white count. CMP was notable for a glucose of 68, magnesium of 1.2, direct bilirubin of 0.4, and a lactate of 2.1. Lipase was normal and a beta hCG quant was negative. Urinalysis was negative for UTI but did show 50 ketones. Magnesium was replenished in the emergency department.

Our differential included PE, appendicitis, colitis, and dehydration. The patient was unresponsive to these fluids and remained hypotensive and tachycardic, so we ordered a CT angiogram for PE and CT abdomen and pelvis with contrast.

In the department, the patient was given diphenhydramine, metoclopramide, ondansetron, and magnesium for nausea and electrolyte abnormalities. CT imaging revealed severe cardiomegaly and right heart strain.

On reassessment, the patient's vitals were 97/72, pulse of 104, and respiratory rate of 15 breaths/minute. Upon obtaining CT results, a bedside limited echo was performed. This revealed significantly reduced EF as well as grossly dilated heart. The global left ventricular function was significantly reduced. Cardiology was called to the bedside, and a formal stat echo was consistent with the bedside echo. This revealed EF <10% and possible left ventricular non-compaction. Given echo findings and a new diagnosis of cardiomyopathy and persistent hypotension, there was a concern for cardiogenic shock, and milrinone was initiated. The patient was subsequently admitted to Cardiac ICU.

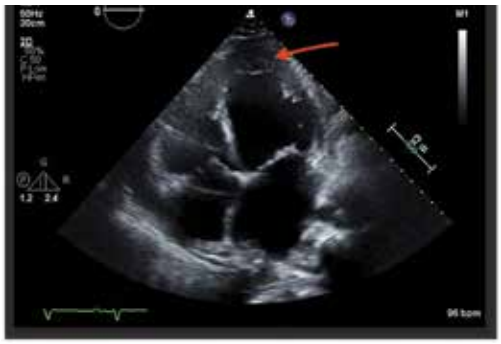

Image 1. Non-compaction in the left ventricle. In this image, you can see the non-compacted trabeculae in the apex of the left ventricle. This non-compaction was the cause of our patient's acute congestive heart failure.

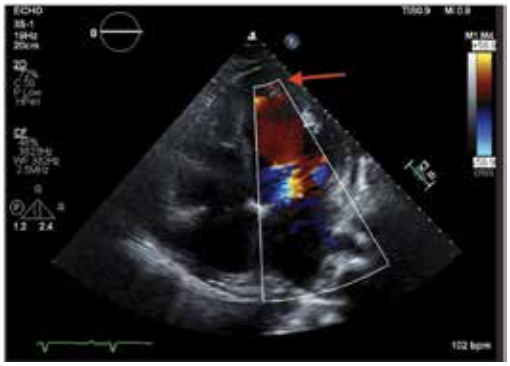

Image 2. This color Doppler ultrasound image shows those same trabeculae at the apex.

References available online.

Our patient spent 10 days in the hospital for new onset decompensated heart failure with a reduced ejection fraction (HFrEF). Cardiology, Rheumatology, and Infectious Disease teams were consulted on her case. Additional testing/workup included a respiratory viral panel, which was negative, and an autoimmune workup for possible hypothyroid-induced dilated cardiomyopathy and viral myocarditis – both of which were also negative. Gastroenterology stated the nausea and vomiting were secondary to volume overload and a pericardial effusion. A cardiac MRI showed non-compaction cardiomyopathy involving the left and right ventricles with global enlarged chambers and hypokinesis. Mitral and tricuspid regurgitation and a moderate-sized pericardial and small pleural effusion were also noted. Prior to discharge, the patient was started on guideline-directed medical therapy (empagliflozin), furosemide, losartan, spironolactone, metoprolol, and a Life Vest. She was referred to the heart failure clinic for follow-up at an outside hospital.

CASE CONCLUSION

One month after her initial presentation, the patient was placed on the transplant list, as her cardiac function had not recovered with medical management. She successfully underwent a cardiac transplant 3 months later and has been doing well since then.

DISCUSSION

Noncompaction cardiomyopathy (NCCM) is a type of cardiac disorder that is believed to occur during embryogenesis. The thought is that this arises from a failure of compaction of the ventricle during ventricular formation. During development, there is a 2 layered ventricular wall: one layer has a thin epicardial surface and a second inner layer has trabeculations that are not compacted. The trabeculae are associated with recesses that communicate with the ventricular cavity but not the circulation of the coronaries.[2]

NCCM was recognized as a distinct cardiomyopathy in 2006 and has an incidence of 0.12/100k patients or 12 in 10 million patients.[3] There are two distinct age ranges of presentation: those in the first few years of life and those in adulthood.[4] It is the third most common cardiomyopathy in children (after dilated and hypertrophic cardiomyopathy), with most children being diagnosed before 1 year of age. Diagnosis in adults is most often triggered by an abnormal EKG or echo. It is also believed there is a significant familial component to these patients and that inheritance can be either autosomal dominant or X linked, which has been seen in a small group of patients.[4]

Complications can include tachydysrhythmias that can worsen heart failure symptoms in pediatric patients. In adults, NCCM often causes syncope. Additionally, there is a risk of thromboembolic events as clots can be formed in the honeycomb structure, similar to clots from stasis of atrial fibrillation. Treatment is aimed at tachydysrhythmias and anticoagulation or antiplatelet therapy. The first-line treatments are beta-blockers or ACE inhibitors. As with many cardiomyopathies, once a patient reaches end-stage heart failure, a transplant is the best option for resolution. In North America, 4% of children on the heart transplant list carry a diagnosis of NCCM.[5] Presenting symptoms in adults include dyspnea, palpitations, chest pain, syncope or presyncope, and prior stroke.[6]

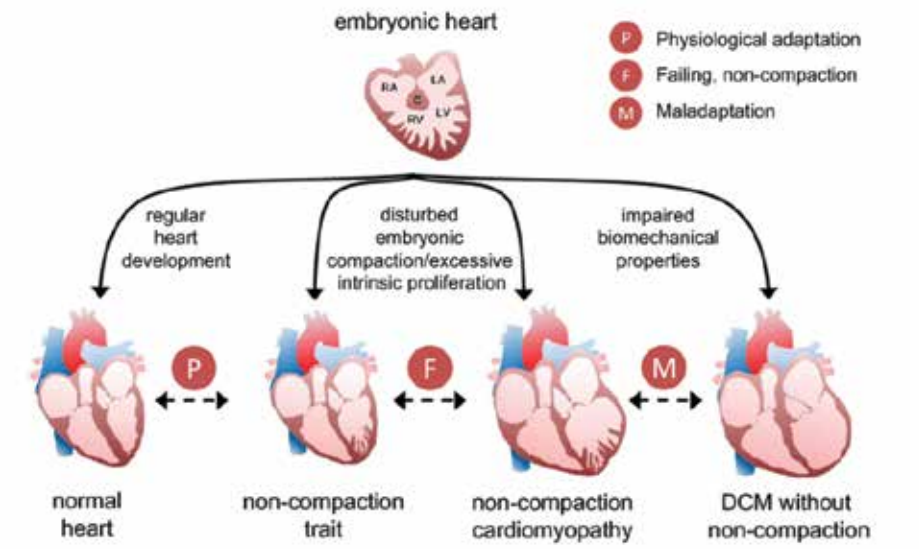

Image 3. Embryonic development of the heart and the trabeculae seen with noncompaction cardiomyopathy (NCCM).[8]

LITERATURE REVIEW

In a small retrospective study performed at a children's hospital in Turkey involving 29 patients, presenting symptoms seen in children include heart failure symptoms (69%), murmur (10%), chest pain (7%), and asymptomatic (7%).[7]

In the case report by Capin et al., a 7-year-old male presented several times over a 3-week period to the ED. This patient's symptoms were noted to be fatigue and suicidal ideation, which progressed to diffuse abdominal pain. He was placed in the ICU and started on a BiVAD due to the severity of the disease. The treatment of this patient was also a heart transplant.[8]

Through our literature research, we saw small sample sizes, with presentations happening mostly in the first years of life or later in life in adulthood. Many of the patients who present with this cardiomyopathy present in the early stages of life, rarely in early adulthood. Additionally, when an otherwise healthy 18-year-old woman presents to the ED with nausea and vomiting, congestive heart failure is not immediately on the differential but should be considered, and POCUS can help evaluate for this as well as other causes of hypotension.

References available online.

EMRA ULTRASOUND POCUS For The Win!

Retrobulbar Spot Sign

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Central retinal artery occlusion (CRAO) is an ocular emergency that commonly presents as sudden, painless, monocular vision loss. It can be a harbinger of serious comorbidities, making diagnosis important. POCUS has shown to be a quick and easy way to diagnose CRAO in the emergency department.

CASE

A 70-year-old female with no known past medical history presented to the ED for evaluation of sudden-onset left eye vision loss. She reported that she went to bed around 8:30 pm the night before and woke up at 9:30 am with a severe left-sided headache that was followed by complete, unilateral loss of vision in her left eye. She denied any other associated symptoms, including numbness, paresthesias, or weakness.

PHYSICAL EXAM

Vital signs revealed a blood pressure of 155/64 mmHg, heart rate of 96 beats per minute, temperature of 98.7° F, respiratory rate of 23 breaths per minute, and SpO2 95% on room air.

On physical exam, bilateral conjunctivae were clear without any injection, discharge, or scleral icterus. Pupillary exam was notable for a relative afferent pupillary defect on the left eye. Her right pupil was reactive to light and accommodation. Her visual acuity evaluation revealed complete left eye visual field vision loss with intact light perception. The vision in her right eye remained at baseline. Her extraocular movements and the remainder of her cranial nerves remained intact.

She exhibited no dysarthria or facial droop and had full strength and sensation in bilateral upper and lower extremities. There was no tenderness or fullness to her temporal regions.

LABS AND IMAGING

The patient was evaluated for acute stroke and received a non-contrast CT head that was negative for acute abnormality. She also had a CT angiography of the head and neck that showed atherosclerotic disease of the carotid arteries without acute occlusion. Notable labs included ESR at 64 and CRP at 3.4, but were otherwise unremarkable.

POCUS OCULAR EXAM

An ocular point-of-care ultrasound was performed on the patient's left eye while in the ED. A linear probe was utilized on the ocular setting. The operator fanned through the eyeball in both the sagittal and transverse axes to visualize the structures of the eye. The

References available online.

relevant anatomy of the eye can be seen in Image 1, with a video clip of normal anatomy seen in Video 1.

On this patient's ultrasound, a hyperechoic density was seen in the optic nerve sheath just distal to the retina (Video 2, Video 3). This was recognized as a "retrobulbar spot sign," which is concerning for a central retinal artery occlusion (CRAO) (Image 2, Image 3).

DISCUSSION

CRAO is an ocular emergency that commonly presents as sudden, painless, monocular vision loss. It is caused by either partial or complete blockage of the retinal artery. The incidence is thought to be approximately 1-2 in 100,000.[1] The mean age of those affected is 60-65 years, and it affects males more often than females.[1] The most common etiology overall is carotid artery atherosclerosis.[2] Other causes include embolism from the heart or carotids, as well as hypercoagulable states, hematologic conditions, giant cell arteritis, and other vasculitides.[1] Risk factors for CRAO are identical to those of other vascular diseases like strokes or coronary artery disease, and may include hypertension, hyperlipidemia, diabetes, and smoking.[2]

POCUS has shown to be a quick and easy way to diagnose CRAO in the ED. When evaluating the optic nerve, a hyperechoic "spot" can be seen, thought to be a calcification or clot in the retinal artery.[3] This is referred to as the spot sign. One study found that the spot sign on ultrasound had a sensitivity of 83% and a specificity of 100% for detection of an embolic CRAO.[4] Another study found that identification of the spot sign was found to have high interobserver agreement, which suggests that CRAO could be easily detected among ED physicians.[5]

Identification of the spot sign may even provide insight into the cause of the CRAO.[6] In a prospective study of patients with CRAO, 46 patients were classified based on the cause of their occlusion. The causes included cardioembolism, large artery atherosclerosis, vasculitis,

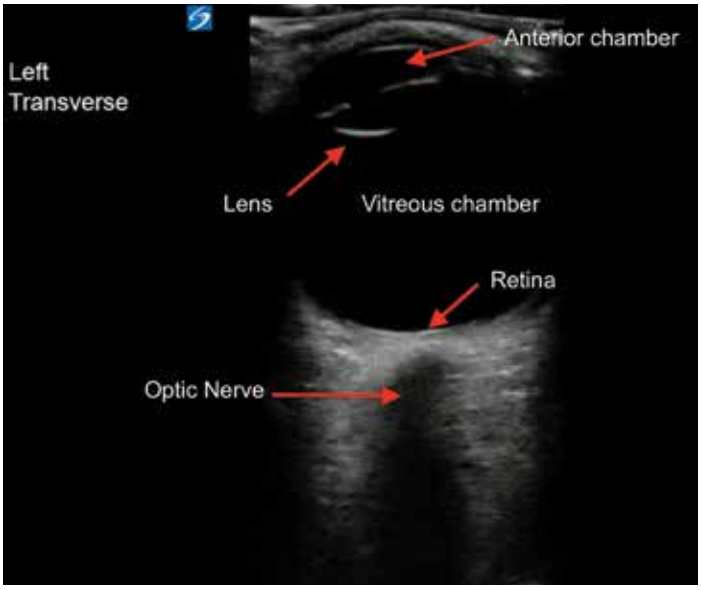


Image 1. Relevant anatomy of the eye

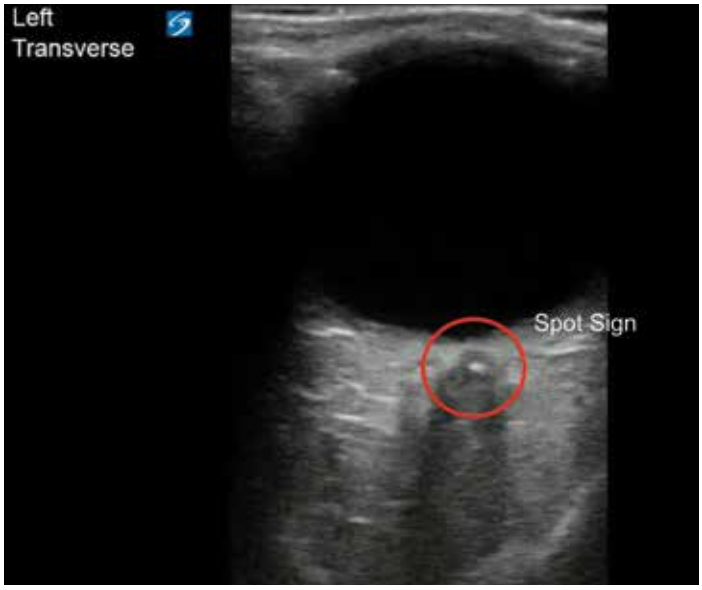
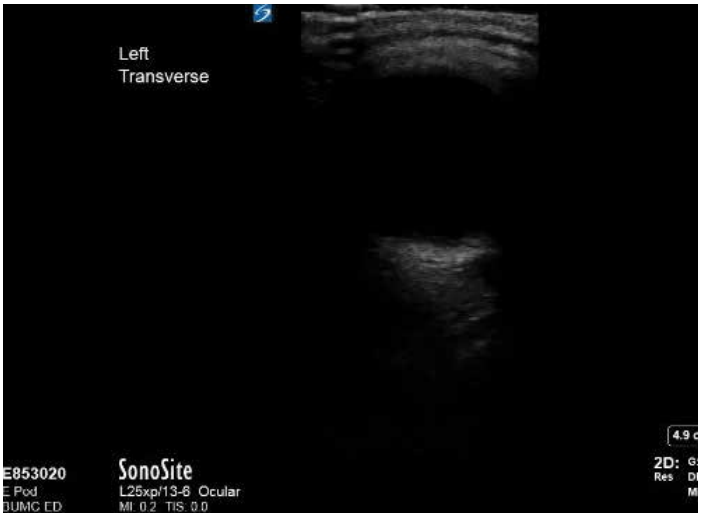
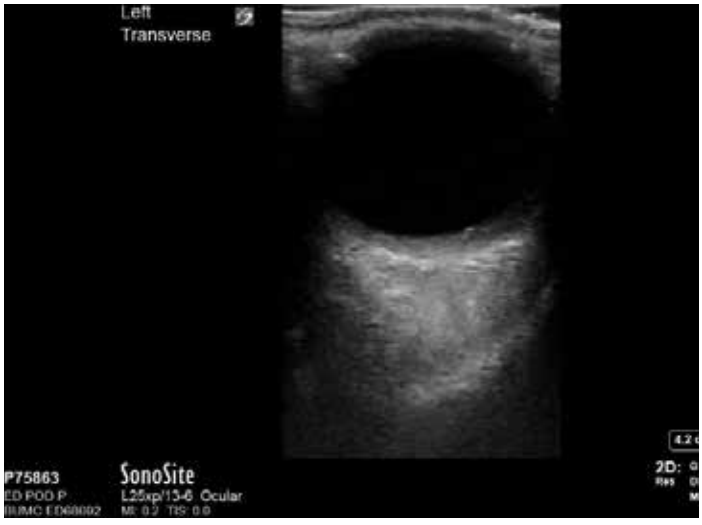


Image 2. Retrobulbar spot sign, transverse view



Video 1. Normal eye, transverse view



Video 2. Retrobulbar spot sign, transverse view

References available online.

and undetermined cause. Interestingly, 59% of the patients with large artery atherosclerosis displayed a spot sign on ultrasound, compared to only 20% of the patients with cardioembolism, and 0% of the patients with vasculitides.[6]

Lastly, a spot sign may help predict a patient's response to therapy.[6] In a small prospective study of 11 patients with CRAO who received tPA, 4 were spot sign negative and 7 were spot sign positive. All of the spot sign-negative patients had improvement in their visual acuity after thrombolytics, while the spot sign-positive patients did not.[6]

Ultimately, additional research is needed to further support the spot sign as a diagnostic and prognostic modality for CRAOs. However, the current data highlights the potential utility and ease of POCUS as a diagnostic modality for identifying CRAO. This is especially true when compared to traditional fundoscopy, which is likely utilized far less by emergency physicians.[8]

MANAGEMENT OF CRAO

Patients who present within 4.5 hours of symptom onset may receive tPA as treatment for CRAO.[2] For those outside this window, there are a variety of other treatment options. For example, reduction of intraocular pressure can be attempted, either through medications like acetazolamide or mannitol, or through anterior chamber paracentesis.[1,2] Vasodilatory medications like pentoxifylline or nitroglycerin can be used to improve ocular blood flow.[1,2] Ocular massage may be used to improve retinal perfusion and attempt to dislodge the embolism.[2] Lastly, hyperbaric oxygen therapy can be used as a temporizing measure while waiting for definitive reperfusion.[2] Additional treatment options such as intra-arterial thrombolytics and surgical revascularization are still being studied.[2]

Both Ophthalmology and Neurology are typically involved in management of these patients. At this time, there are no definitive evidence-based treatment guidelines for CRAO.[9] Some patients may experience spontaneous recovery of vision, however the overall prognosis is poor.[2]

HOSPITAL COURSE AND RESOLUTION

This patient was evaluated by Neurology and did not receive tPA, as she was outside the 4.5-hour window. They agreed with the concern for CRAO; however, they recommended treatment for giant cell arteritis as well, given the patient's elevated inflammatory markers. She received a 5-day course of IV methylpredisolone, and underwent biopsies of the temporal arteries (performed by Vascular Surgery). Biopsies were subsequently negative for temporal arteritis.

Hyperbaric oxygen therapy was recommended by Ophthalmology, for which she completed 5 treatments while inpatient. She had an MRI of the brain and orbits that revealed acute supratentorial and infratentorial infarcts in multiple vascular territories, concerning central embolic etiology per Radiology. Her formal echocardiogram showed a dilated right atrium, raising the question of atrial fibrillation as a cause, despite being in normal sinus rhythm throughout her hospital stay. Her echo also revealed severe mitral annular stenosis, which was thought to be another potential source of embolism. She was started on aspirin, atorvastatin, and received an insertable loop recorder with Cardiology.

The patient was discharged home to follow up with outpatient Cardiology and was ultimately prescribed apixaban. Unfortunately, she had persistent vision impairments in her left eye.

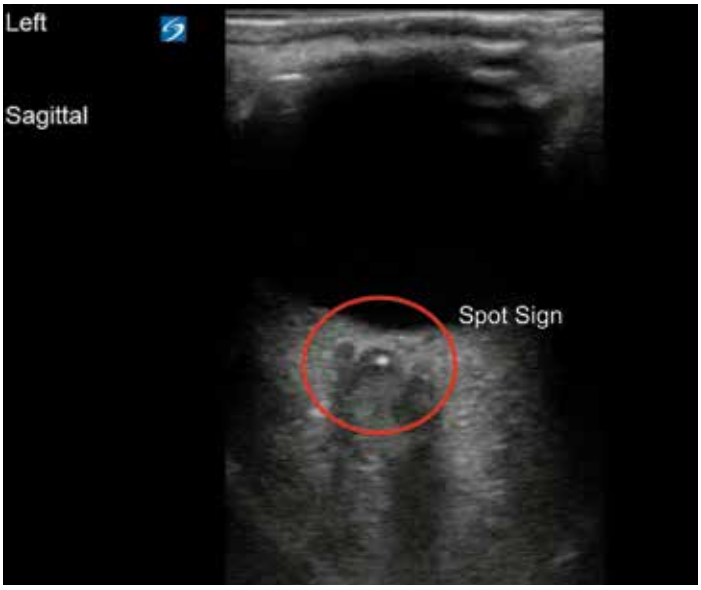


Image 3. Retrobulbar spot sign, sagittal view

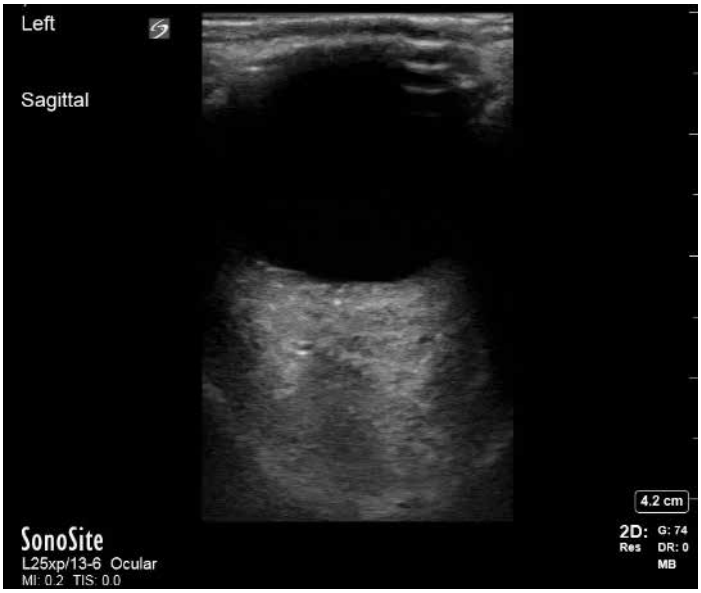


Image 3. Retrobulbar spot sign, sagittal view

References available online.

The Bougie: Not Just for a Difficult Airway

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Up to 200,000 patients per year in the U.S. are given percutaneous endoscopic gastrostomy (PEG) tubes. The PEG tube is indicated in many critically ill patients, including those with a limited ability to tolerate oral intake due to trauma or obstruction; PEG tubes may also be used for gastric decompression. However, a sizeable portion of those who receive PEG tubes struggle with dislodged tubes.

CASE PRESENTATION

A 72-year-old male with a history of left side deficit secondary to a previous stroke presented to the ED after pulling his trach and G-tube. The patient was oriented to self and place, and physical exam showed no bleeding or signs of infection around the site. However, the G-tube was pulled at least 3 hours prior to arrival. After 3 hours there's concern for closure of the tract, even for an established G-tube.

Instead of attempting to open the tract with a Foley catheter, a bougie was used, as it is more rigid than the urinary drainage catheter. It was applied with gentle pressure to re-open the lumen, then replaced with a smaller G-tube. The follow-up X-ray with diatrizoate showed the G-tube in place within the body of the stomach and without contrast extravasation. There were no complications from the procedure.

DISCUSSION

Accidental PEG tube dislodgement rates have been reported between 1% and 13.4% when followed longitudinally for the lifetime of the PEG, with some reports showing displacement in up to 20% of patients.[1,2] A report in the Pennsylvania Patient Safety Authority described G-tube dislodgments as the most frequently reported complicated of G-tubes; one study revealed a rate of 78.3% displacement over 5 years. The report included potential causes of G-tube dislodgment, including the patient pulling on the tube and tube movement during patient care.[3]

As nursing home populations rise while a nursing shortage persists, the chance of a G-tube dislodgment occurring and going unnoticed in that setting increases. However, replacement of the tube is time-sensitive to prevent the tract from closing; some reports encourage replacement within a 2-hr window to prevent complications.[3]

TROUBLESHOOTING DISLODGED TUBES

Historically, we have placed a Foley catheter in the stoma to keep it open until time allows for replacement of the tube. After determining the length of time the tube has been in place, the length of time it's been dislodged, and performing an abdominal exam, replacement of the tube can take place.

One other piece of equipment can be considered in managing dislodged PEG tubes: the bougie. There are no formal studies concerning the efficacy or complication rate of using a bougie to keep the PEG stoma open. In some cases, the Foley catheter may not access the stoma once it's started closing. EMDocs recommends attempting access with red rubber catheters, curved forceps, or an irrigation syringe to widen the stoma. However, it's never been recommended to use a bougie.[4] The bougie provides enough flexibility to receive positive feedback from surrounding structures while simultaneously incorporating enough rigidity to prevent rolling on itself.[5] In contrast, the Foley catheter method frequently doesn't work, may cause trauma to the site, or coil on itself.

CONCLUSION

Due to the rigidity, availability, and size of the bougie, it makes an excellent option for G-tube replacement and tract dilation in the setting of delayed G-tube replacement requiring a downsized tube.

In recent experience it has had a 100% success rate in a small sample size in various community emergency departments within the Texas area, including several nursing home patients who may not have the knowledge or equipment readily available to keep the tract patent in a timely manner.

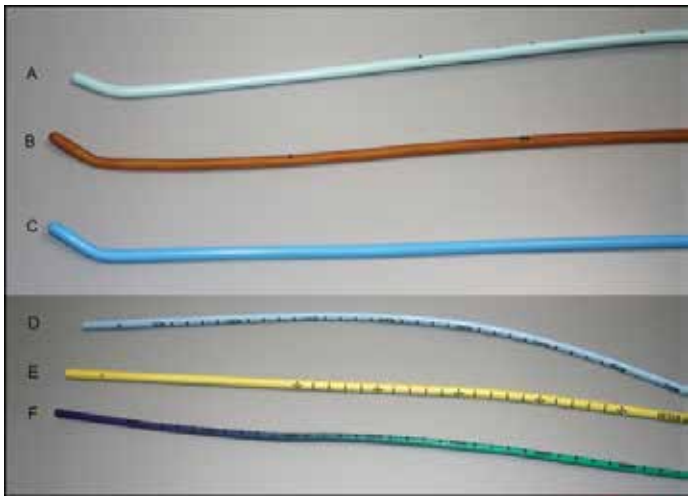

Image 1. Endotracheal tube introducers and airway exchange catheters
(A) Portex single-use introducer (Smiths Medical); (B) the reusable Eschmann tracheal tube introducer (Portex Venn, Smiths Medical); (C) gum-elastic bougie (SunMed); (D) Cook Medical Aintree intubation catheter; (E) Cook airway exchange catheter; (F) Cook airway exchange catheter (Image courtesy of Anesthesia News)

References available online.

Responding to ICE in Emergency Departments:

Protecting Patients and Navigating Legal Obligations

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Hospitals and emergency departments have long been considered places of refuge, where individuals can access medical care without fear of discrimination or interference, regardless of their immigration status. However, recent changes in immigration enforcement policies, including the recission of 2011 Sensitive Locations Memorandum, have heightened concerns about how health care providers should respond to Immigration and Customs Enforcement (ICE) agents visiting hospitals. This shift has significant implications for patients, clinicians, and hospital operations, especially in emergency settings where patient trust and safety are paramount.

Historically, ICE was discouraged from conducting enforcement actions — such as arrests or interrogations — in certain locations, including hospitals, schools, and places of worship. The intent of this policy was to ensure individuals could access essential services without fear of deportation. However, recent policy changes have diminished these protections, permitting ICE to conduct enforcement actions in hospitals under certain conditions.[1] This policy shift has raised alarm within the medical community, particularly in emergency departments where vulnerable populations frequently seek care.

This shift could deter undocumented individuals from seeking medical care, placing healthcare providers in the challenging position of balancing legal compliance with their ethical duty to care for all patients. EDs must adopt detailed protocols and training to ensure patient privacy and safety while adhering to federal laws such as the Health Insurance Portability and Accountability Act (HIPAA).[2,3]

To navigate these changes, ED staff must be prepared with clear protocols and thorough training to manage ICE interactions. This article combines a comprehensive hospital policy with step-by-step instructions to equip ED staff with the knowledge and tools necessary to handle ICE interactions effectively and ethically.

THE IMPACT ON PATIENTS AND EMERGENCY CARE:

1. Patient Fear and Avoidance of Care

Undocumented individuals may delay or avoid seeking medical care due to fear of deportation or detention. Studies show that increased ICE activity near sensitive locations correlates with decreased healthcare utilization among immigrant communities.[4] This avoidance can lead to worsening health conditions, delays in treatment, increased emergency department visits for preventable issues, and public health risks.

2. Disruption of Emergency Services

ICE activity in emergency settings can create chaos, disrupt patient care, and increase stress for both patients and staff. The presence of law enforcement agents may escalate tensions, particularly for vulnerable populations already experiencing trauma.[3]

3. Ethical and Legal Considerations

Health care teams have an ethical obligation to treat all patients equitably, regardless of their immigration status.[5] Simultaneously, HIPAA mandates the protection of patient information and limits when and how it can be disclosed, even to law enforcement.[2]

POLICY: MANAGING ICE INTERACTIONS IN EMERGENCY DEPARTMENTS

Purpose: To establish clear procedures for responding to ICE agents in a manner that prioritizes patient safety, respects legal obligations, and maintains the hospital's commitment to equitable care.

Scope: This policy applies to all employees, medical staff, contractors, and volunteers at the hospital.

Policy Statement

- The hospital is a safe space for all patients seeking medical care, regardless of immigration status.
- ICE agents are not allowed access to patient areas or information without a valid judicial warrant.
- All staff are expected to prioritize patient care and privacy in every situation.

PROCEDURES

A. ICE Arrival at the Hospital

- **Initial Interaction**
  - ICE agents must identify themselves and state the purpose of their visit.[1]
  - Staff should request identification and any supporting documentation, such as a warrant.

- **Notification**
  - Immediately notify the designated Point of Contact (POC), such as the hospital administrator or legal counsel.[6]
  - Security personnel should be informed and positioned to monitor the situation without escalating tensions.
- **Verification of Warrant**
  - Determine if the warrant is judicial (signed by a judge) or administrative (issued by ICE). Judicial warrants allow access under specific conditions, while administrative warrants do not grant access to private areas.[3]

B. Patient Privacy and Safety

- **Access to Patients**
  - ICE agents may not interview, detain, or remove patients without a valid judicial warrant.
  - Do not allow ICE agents to access restricted areas such as patient rooms or operating areas.
- **HIPAA Compliance**
  - Never disclose patient information without a valid judicial warrant or explicit patient consent.[2]
  - Ensure that medical records do not document immigration status unless medically relevant.[3]

C. Informing Patients

- If ICE agents request to interview or detain a patient, inform the patient (if medically appropriate) and explain their rights:
  - The right to remain silent.
  - The option to decline speaking to ICE agents without an attorney present.[5]
  - Offer the patient contact information for legal aid organizations specializing in immigration law.[7]

D. Training and Education

- Provide comprehensive training for staff on the hospital's policy, including:
  - Recognizing valid warrants.
  - HIPAA compliance.
  - De-escalation techniques when interacting with law enforcement.[6]
- Conduct annual policy refreshers and simulate scenarios to reinforce procedures.

E. Documentation

- Record the details of any ICE interaction, including:
  - Names and badge numbers of agents.
  - Date, time, and reason for the visit.
  - Actions taken by staff and outcomes.[6]
- Submit the documentation to the POC and legal counsel for review.

DETAILED STEP-BY-STEP GUIDE FOR ED STAFF

1. Understand Legal Protections and Responsibilities

- All staff must understand that patient information, including immigration status, is protected under HIPAA. Information can only be disclosed with patient consent or a valid court order.
- Train staff to differentiate between a judicial warrant (signed by a judge and legally binding) and an administrative warrant (issued by ICE and not sufficient for access).[2]
- Train staff on how to handle ICE interactions respectfully and lawfully.[3]
- Patients have the right to decline speaking with ICE agents and may request legal representation before interacting with them.

2. Prepare a Response Plan

- Assign a Point of Contact (POC) to manage ICE inquiries and coordinate staff responses.[6]
- Develop clear protocols for documenting ICE interactions and preserving patient confidentiality.

3. Address ICE Agents Professionally

- Greet ICE agents professionally and ask for identification and the purpose of their visit.
- Request a judicial warrant to confirm the legal basis for their actions. If ICE agents present an administrative warrant, explain that it does not authorize access.[3]
- Direct ICE agents to a non-patient area and ensure they are accompanied by authorized personnel at all times.
- Avoid confrontations or escalating tensions.

4. Focus on Patient Safety

- Shield patients from ICE activity as much as possible.
- Ensure patient care continues uninterrupted.
- Do not disclose any patient information, including location or medical records, without a valid judicial warrant.[2]
- Focus medical records on clinical care only. Avoid noting immigration status unless it is medically relevant.

5. Advocate for Patients

- If ICE requests to detain or speak with a patient, inform the patient of their rights to refuse to speak with ICE agents and provide support if they wish to contact legal counsel.[7]
- If the patient is critically ill or in an unstable condition, explain that medical care takes precedence over enforcement actions.
- Offer contact information for local immigration legal aid organizations or advocacy groups.
- Reassure patients and families that the hospital is committed to providing care regardless of immigration status.

6. Document the Incident

- Record key details, including: names and badge numbers of ICE agents; time, date, and purpose of the visit; copies or descriptions of any warrants or documents presented; actions taken by staff.
- Submit an internal incident report to hospital administration and legal counsel.

7. Conduct Post-Incident Review

- Debrief staff and review actions taken during the incident.
- Update policies and training materials as needed to address any identified gaps.

WHY THIS MATTERS

The presence of ICE agents in emergency departments can erode trust in healthcare systems, especially among immigrant populations. Patients may avoid seeking care, leading to worsened health outcomes and further disparities. By implementing clear protocols and maintaining a commitment to equitable care, healthcare providers can protect vulnerable patients while navigating this complex legal landscape.

References available online.

RESPONDING TO ICE
IN THE EMERGENCY DEPARTMENT

Protect Patient Privacy. Verify Documentation. Contact Admin.

Know the Rules

- **Private Areas:** Know which areas of the hospital are public and which are closed to the public.
- **JUDICIAL WARRANT REQUIRED:** Must be signed by judge or magistrate. ***Administrative warrants*** (from Department of Homeland Security [DHS] / Immigration and Customs Enforcement [ICE] / Homeland Security Investigations [HSI]) **do not** authorize access to private areas.
- **"Plain View" Surveillance:** Even without a warrant, ICE can examine anything in plain view, including conversation in private areas that can be heard from public areas.
- HIPAA Compliance: **All patient information — including immigration status — is protected under federal law.** Never disclose it without the patient's consent or a valid judicial warrant.

Step-By-Step Guide

- **Stay Calm:** Do not panic. Do not confront ICE agents directly.
- **Notify Hospital Designated Point of Contact**. Call _____
- **Request Documentation:** Politely ask for identification and a judicial warrant signed by a judge or magistrate. Review the warrant. Administrative warrants or subpoenas issued by DHS/ICE/HSI do not automatically grant permission, but the hospital may choose to comply.
- **Defer to Designated Representative:** Calmly tell agents that you are not authorized to provide information or access to private areas, but that, following protocol, a designated representative has been alerted.
- **Protect Patient Care:** Ensure medical care is not interrupted or delayed.
- **Advocate for Patients:** Inform patients of their rights, including the right to legal representation and the option to decline speaking to ICE. Advise patients not to run from or confront agents. You may offer contact information for local immigration legal aid organizations, if available.
- **Request Location of Detainees:** If agents remove any patients or employees, ask the agents where they are being taken.
- **Document the Incident:** Record agent names, departments, badge numbers, times, visit purpose, warrant details, how agents were dressed, actions taken and/or outcomes, and any deviations from the scope of the warrant or other possible misconduct by agents (e.g., leading employees or patients to believe they could not move or leave) for hospital records.

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Always prioritize patient safety and care in compliance with hospital policies and laws.

A Curious Case of Cardiogenic Syncope with a Neurologic Source

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Syncope can have many causes that are not always evident at the initial presentation.

CASE PRESENTATION

An 80-year-old male presented to the ED following a syncopal episode with a ground-level fall. According to the patient's family, the patient was his usual self before he lost consciousness while walking within their home. The patient reportedly remained unconscious for an unknown length of time with no jerking movements, loss of bladder function, or post-awakening confusion. Upon arrival to the ED, the patient was found to have intermittent heart rates in the 15-20s with rebound to the 50s-60s. During the bradycardic episodes, he was symptomatic with dizziness. The patient was otherwise hemodynamically stable. He denied the use of cardiovascular medications but reported an increase in pyridostigmine dosage for his myasthenia gravis approximately 2 months ago. He was admitted to Cardiology with plans for urgent pacemaker implantation.

DISCUSSION

When considering the etiology of syncope, the differential can be categorized into reflex, orthostatic, and cardiac causes. The origin of a reflex-based syncope is most commonly vasovagal secondary to a trigger, such as stress or prolonged standing. It can also be situational, such as due to carotid sinus hypersensitivity.[1] Orthostatic causes can be another origin of syncope and may be the result of hypovolemia, autonomic dysfunction, or the use of certain medications such as vasodilators or inotropic/chronotropic blockers. Finally, cardiac syncope is syncope secondary to malfunction of the cardiovascular system and can include bradycardia, tachycardia, and structural diseases such as hypertrophic cardiomyopathy, ischemia, or valvular dysfunction.[2]

In this case, the patient's syncope was most likely cardiac in nature given his symptomatic bradycardia upon presentation to the ED. Initial management of symptomatic bradycardia is dependent on the patient's hemodynamic stability. If vital organ function is acutely impaired, then atropine is the initial treatment of choice. If bradycardia persists with the use of atropine, then sympathetic beta-1 stimulating medications, such as epinephrine or dopamine, may be indicated with the initiation of transcutaneous pacing if symptoms continue.[3]

In a hemodynamically stable patient, management becomes directed toward determining and treating the underlying cause of the bradycardia. In many cases it is due to sinus node dysfunction, acute myocardial ischemia, or medication toxicity; however, it is also important to consider non-cardiac causes of bradycardia in the differential.[4]

Herpes simplex virus (HSV) encephalitis is an acute central nervous system infection associated with significant morbidity and mortality. It often presents with a prodromal phase of fever, malaise, headache, and nausea followed by more severe neurologic symptoms such as meningismus and seizures.[5] In rare cases, HSV encephalitis has also been shown to cause sinus node dysfunction and arrest.[6]

Diagnosis of HSV encephalitis can be through lumbar puncture, which most often shows pleocytosis of cerebral spinal fluid with lymphocytic predominance and elevated protein. Brain CT (**Image 1**) or MRI can also be used as a diagnostic tool displaying temporal lobe abnormalities.[7]

References available online.

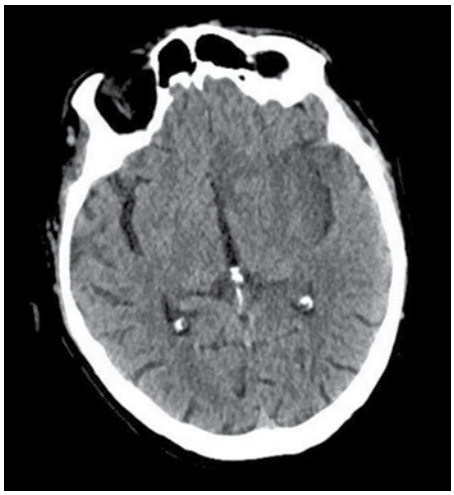

Image 1. CT revealing signs of CNS dysfunction

Early identification and treatment with intravenous acyclovir is crucial, as the disease carries significant rates of morbidity and mortality. Mortality is predicted to be 20-30% with prompt diagnosis and treatment but can be up to 70% for untreated infection.[8] Long-term morbidity may include significant cognitive and behavioral disability including anterograde amnesia, Klüver Bucy syndrome, and seizures. When considering cardiac impairment from HSV, a previous case suggests prompt treatment may be sufficient to reverse disease-associated sinus node dysfunction without the need for permanent pacemaker implantation.[9]

HOSPITAL COURSE

Several hours after admission, the patient underwent uncomplicated dual chamber pacemaker placement with plans to be discharged in the morning. However, upon examination the following day, the patient was displaying both expressive and receptive aphasia with right leg weakness. Workup for a possible ischemic event, seizures, or delirium revealed a negative non-contrast head CT, CTA head and neck, and cerebral perfusion imaging. Meanwhile, the patient began experiencing respiratory distress with SpO2 in the high 80s. A chest X-ray showed pulmonary vascular congestion.

With rapidly deteriorating neurological and respiratory status, the patient was transferred to the ICU and intubated. Given the deterioration of neurological status, a lumbar puncture revealed xanthochromic cerebral spinal fluid with elevated protein, elevated RBCs, and normal WBCs with elevated neutrophil fraction. HSV encephalitis was suspected at this point, and the patient was started on acyclovir. PCR results later confirmed HSV.

CONCLUSION

Syncope can have many causes that are not always evident at the initial presentation. As evidenced by this case, HSV encephalitis can have a variety of presenting signs and symptoms that may not appear to be of neurological origin. Clinicians should be cognizant of this variability and consider that acute CNS infections, such as HSV, may be an etiology for sinus node dysfunction in cardiac syncope patients.

Osteomyelitis and Auditory Hallucinations Associated with Long-Term Inhalant Use

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A 54-year-old male presented to the emergency department with a chief complaint of hallucinations and recent use of inhalants.

He reported a history of intermittent inhalant use over the past 15 years, primarily using electronic duster cans. He reported inhaling 10-15 cans daily over the past week, after a period of abstinence. The patient stated that over the past few days, he had been hearing voices commanding him to remove his firearm out of his safe. The patient also reported that he had been seeing moving shadows and people in his home that he believed were trying to rob him. On the morning of presentation to the ED, the patient removed his firearm from the safe without remembering, and expressed fear that he might accidentally harm a family member with his firearm.

Initial vital signs in the ED included BP 125/84, HR 87, T 36.7 C, RR 20, SpO2 97% on room air. On examination, the patient appeared tremulous with pressured speech but was oriented to person, place, time, and event. Skin exam was notable for a chronic appearing wound present on the right thumb (**Image 1**) with tissue loss and probe-to-bone noted. The right forearm also demonstrated signs of thermal injury (**Image 2**). On further questioning, the patient could not report the duration of his thumb or arm injuries.

Cardiovascular, respiratory, and abdominal exams were otherwise unremarkable. Labs were notable for a white blood cell count of 12,200/uL, ESR 43 mm/h, and CRP 62.6 mg/L. Comprehensive metabolic panel and venous blood gas were normal. EKG showed normal sinus rhythm and normal intervals. Radiographs of the hand demonstrated small cortical erosion at the volar thumb distal phalanx, consistent with chronic osteomyelitis.

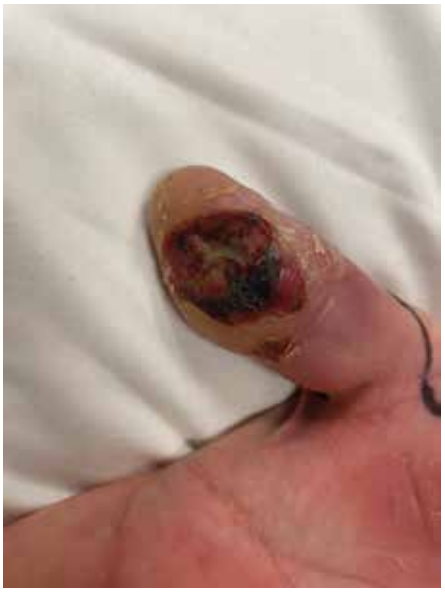


Image 1

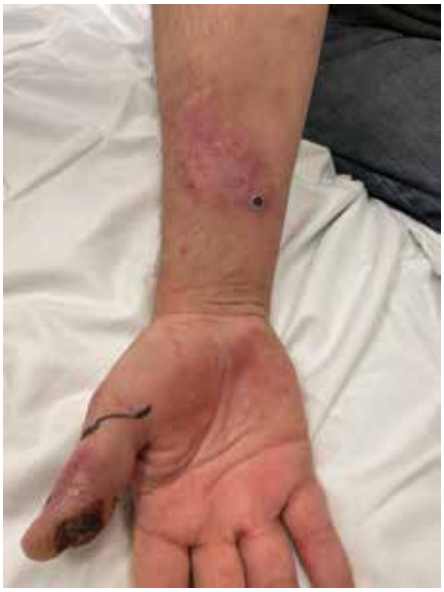


Image 2

References available online.

DISCUSSION

Inhalant use, otherwise known as huffing, sniffing, bagging, or dusting, typically involves the inhalation of volatile or aerosolized hydrocarbon-based products leading to a euphoric effect or otherwise altered mental state. More commonly used inhalants include chlorofluorocarbons (e.g., difluoroethane, tetrafluoroethane) found in computer keyboard spray or "duster," aromatic hydrocarbons (e.g., toluene, xylene) found in adhesives and paint thinners, aliphatic hydrocarbons (e.g., butane, gasoline) found in petroleum distillates, or nitrites (e.g., "poppers"), commonly sold as club drugs. Additionally, non hydrocarbon inhalants such as nitrous oxide (whippets) also have been used as inhalants. The pharmacologic effects and clinical presentations can vary significantly from one substance to another.[1,2] Further, there is little literature characterizing the pharmacologic properties of inhalants, as many are not marketed for human consumption and otherwise do not serve a medical or therapeutic purpose.

Inhalant use is more common in younger individuals, with most reported users aged 25 and younger. Inhalant use is also more prevalent in lower socioeconomic classes and rural settings, which is likely due to the relative ease of access and inexpensive nature of most inhalants.[2]

Inhalants have high lipophilicity, leading to most striking CNS effects. Initial CNS effects include euphoria and hallucinations, both auditory and visual hallucinations. Toxicity can lead to CNS depression, slurred speech, confusion, tremor, weakness. Ataxia, lethargy, seizures, coma, respiratory depression can occur as well.[2]

Psychiatric effects from inhalants may include disorientation, agitation, psychosis, or psychomotor slowing. In the absence of laboratory abnormalities, treatment is largely supportive, and benzodiazepines are recommended.[2] Ongoing psychiatric disturbances may require consultation with Psychiatry and potential hospitalization. Patients may experience withdrawal from long-term inhalant use, which often mirrors alcohol or benzodiazepine withdrawal symptoms including tachycardia, tremors, diaphoresis, vomiting, and seizures.[1] Medical and behavioral management may be required to treat inhalant use disorder, which is a distinct diagnosis according to DSM-5.[3]

Complications of inhalant misuse include hypoxic brain injury and sudden death from hypoxia, which may occur in as little as a single ingestion. White matter degeneration can occur after chronic use.[1,4] Any inhalant can potentially cause "sudden sniffing death" via myocardial sensitization, often seen after increased physical activity (e.g., running away from parents or authorities after being caught using inhalants). The mechanism behind myocardial sensitization is thought to be due to the blocking of potassium current, leading to prolonged repolarization. This produces potential for dysrhythmia propagation during increased activity or stress that leads to catecholamine surge. More commonly, cardiotoxicity presents with palpitations, shortness of breath, syncope, and ECG abnormalities including atrial fibrillation, premature ventricular contractions, QT prolongation, and U waves.[2]

The most significant respiratory complication is hypoxia, which is either caused by displacement of inspired oxygen with inhalant, reducing FiO2, or by rebreathing exhaled air with bagging.[2]

Chemical pneumonitis is seen frequently with aliphatic hydrocarbons due to their toxic effect on pulmonary tissues with aspiration.[5] Intraoral thermal/chemical injuries in these patients can also lead to airway compromise.[6] Inhalant use is also associated with apnea, methemoglobinemia, renal tubular acidosis, hepatotoxicity, and acute myelocytic leukemia.[1,4,7] Appropriate resuscitative measures should be considered in ill-appearing patients, including a low threshold for intubation. Continuous cardiac monitoring, monitoring of pulse oximetry, and serial echocardiograms may also be necessary.

Dermatologic findings associated with inhalant use include cold thermal injuries, or "frostbite" secondary to holding a can while huffing, or perioral dermatitis secondary to irritant properties of the inhaled substances ("glue sniffer's rash").[8] Patients may also show larger rashes after prolonged exposure to cans or accidental discharge of contents onto the skin while altered.[9]

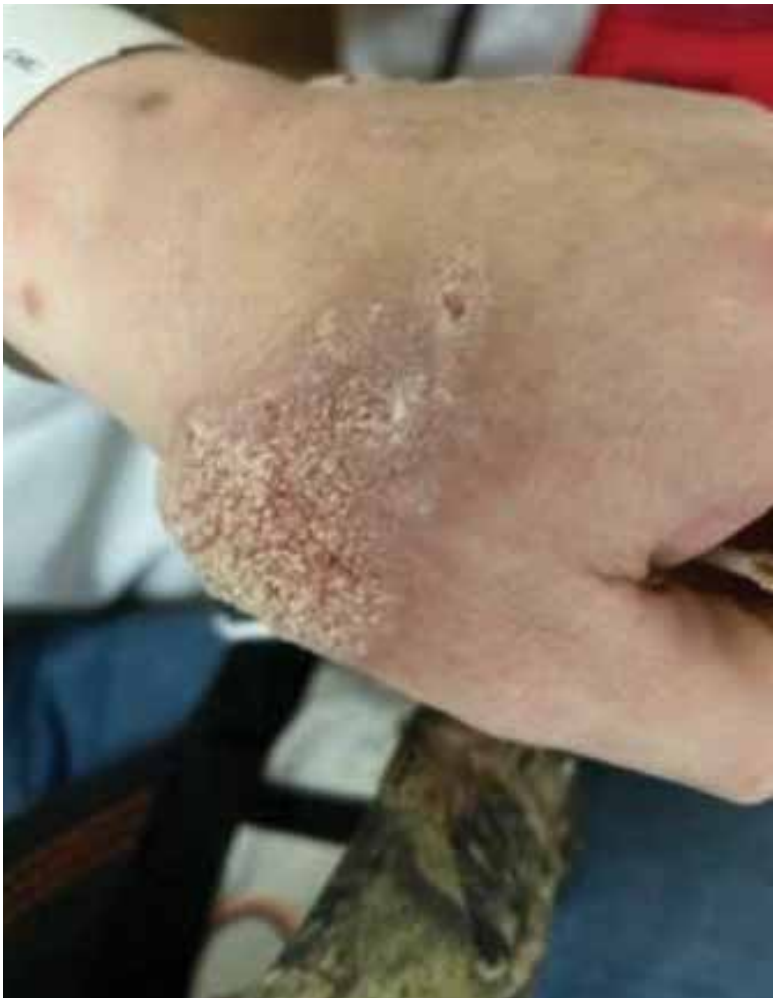
In addition to appropriate resuscitative measures, it is important to perform a thorough skin examination in patients suspected of inhalant use; consider consultation with burn surgery or orthopedics as necessary. In the patient described above, chronic inhalant use and repeated thermal injury to the thumb from the spray can is suspected to have led to gradual tissue destruction to the bone.

CASE CONCLUSION

The patient was admitted voluntarily to the mental health unit for psychiatric stabilization and treatment of substance use disorder with an orthopedic consult. The orthopedic management of the patient's osteomyelitis during his hospitalization included ceftriaxone IV in the ED and oral amoxicillin-clavulanic acid, as admission to the inpatient mental health unit precluded ongoing IV drug administration.

References available online.

Vision Loss Leading to HIV Diagnosis



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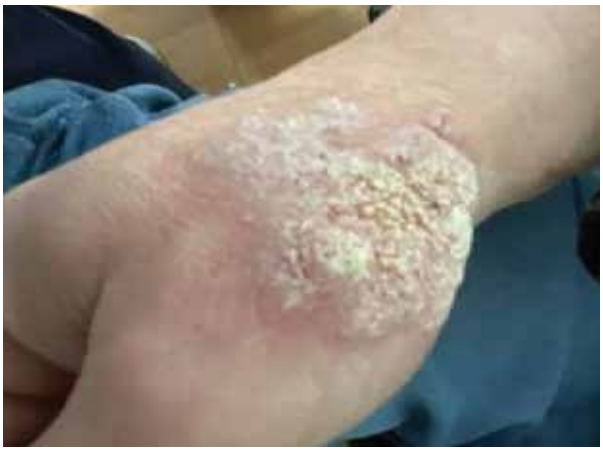

We present a case of a 41-year-old male with a history of IV drug use and MRSA bacteremia who presented to the ED with a rash and bilateral vision loss.

The patient endorsed a rash in the upper and lower extremities, which worsened over the last several months. In addition, he endorsed several weeks of a non-painful rash on his scalp with associated hair loss. Regarding his visual complaints, his symptoms began several weeks ago as night vision impairment. Over time, his vision continuously worsened, and upon presentation, he had developed significant "cloudiness" at the center of his visual fields. He reported only seeing "outlines" of objects or people, but his vision overall was very hazy. A brief review of systems was significant for bilateral ocular discharge, headache, as well as a genital sore which resolved several weeks ago. No fevers were reported.

Past medical history included acute septic pulmonary embolism, allergic rhinitis, anxiety, and GERD. He reported using albuterol and eucerin, and listed allergies to cephalosporins, penicillin, and pollen.

The patient endorsed a long history of IV drug use (methamphetamine), most recently within the past month. The patient also states his last sexual encounter was over a year ago. He was unemployed and living with his father. He denied ethanol use but reported smoking 2-4 cigarettes monthly. He denied recent travel.

Upon physical exam, the patient appeared well-developed, pleasant, in no apparent distress, with no focal deficits neurologically. Vital signs were BP 138/84, pulse 109, RR 20, temp 99.7, oxygen 97 % on room air.

The patient's eyes showed traces of edema on both lids/lashes, with chemosis/injection with moderate clear/yellow discharge. His corneas were mildly hazy, without defects or edema. Anterior chamber was 4+ flare, cells suspended; irises showed synechiae

References available online.

with prominent vessels. The patient's lenses showed pigment on the capsule, with hazy anterior vitreous. Extraocular movements were intact. Acuity in the right was sensitive to hand motion, and in the left, to light perception.

The patient's skin showed multiple irregular rough, scaly plaques over bilateral upper extremities, and irregular erythematous macules over the anterior scalp with overlying fine scale. Bilateral lower extremities presented with multiple irregular erythematous macules with areas of excoriation.

The differential diagnosis included Lupus, Syphilis, HIV, HPV, Gonorrhea/ Chlamydia, HLA-B27 related condition, hypertensive retinopathy, diabetic retinopathy, bacterial/viral conjunctivitis, uveitis, endophthalmitis, verruca dermatitis, cutaneous syphilis, and other etiologies.

INITIAL EVALUATION

Initial workup from the emergency department revealed elevated CRP (12), ESR (109), and an unremarkable CBC and BMP. Multiple laboratory tests were subsequently added on including blood cultures, ANA , quantiferon, Toxoplasma Gondii IgG/IgM Antibody, and Histoplasma, all of which were negative. Given the severity of the vision loss and the extensive rash, consultations were requested from Dermatology and Ophthalmology.

The patient was evaluated by Dermatology, who believed the scalp rash was likely consistent with seborrheic dermatitis, but that the bilateral upper and lower extremity rash was felt to be consistent with very large verrucae. Given the unusual size and persistence of verrucae, they suspected the patient was likely immunosuppressed, and additional labs were ordered including HIV (HIV-1 positive, RPR (reactive, >1:1024), FTA ABS (reactive), eye cultures (negative).

The patient was also seen initially in the ED by Ophthalmology, who then brought the patient over to their clinic for a more detailed eye examination outlined above. Their findings were concerning for endophthalmitis, and the patient was returned to the ED for admission to the Hospitalist Service with Dermatology, Ophthalmology, and Infectious Disease consulting.

CONFIRMATORY EVALUATION/ DIAGNOSIS

At the Ophthalmology clinic, where their examination was concerning for bilateral endogenous endophthalmitis, an ocular paracentesis was performed and the patient was instructed to return to the ED for admission for IV antibiotics, ID consultation, and an echocardiogram to rule out endocarditis. During his

admission, labs were significant for reactive RPR/FTA-ABS, HIV-1 antibody, and CMV IgG. Blood cultures showed no growth and fungal culture was negative. Hepatitis B, Hepatitis C, ANA, and ANCA were negative. His toxicology screen was positive for methamphetamine. CSF following a lumbar puncture demonstrated elevated protein and reactive VDRL. Echocardiogram was negative for endocarditis. Eye culture demonstrated rare PMNs. A left forearm skin biopsy was consistent with secondary syphilis.

DIAGNOSIS AND OUTCOME

Based on the physical exam and laboratory findings, the patient was diagnosed with syphilis meningitis, ocular syphilis (endophthalmitis), and syphilitic dermatitis, in the setting of new HIV infection. The patient was admitted to the hospital for 2 weeks of IV penicillin. He received a dose of intravitreal ceftazidime as well as voriconazole. Around the time of discharge, the patient was endorsing a persistent but improved visual deficit. Repeat visual acuity was significant for the following:

VISUAL ACUITY:
- Right: 20/200
- Left: HM/inconsistent CF

ADNEXA/LIDS:
- Right: Mild crusting
- Left: Mild crusting

CONJUNCTIVA/SCLERA:
- Right: White and quiet
- Left: Trace injection

CORNEA:
- Right: Clear
- Left: Clear

ANTERIOR CHAMBER:
- Right: Grossly quiet
- Left: Improving clot of fibrin/debris in front of the lens, tr hyphemia in area of clot

IRIS:
- Right: Dilated, all synechiae broken
- Left: Irregular, breaking synechiae but still attached to fibrin clot, yellowed iris

The patient was subsequently discharged with ophthalmology and addiction medicine referrals. He was provided prescriptions for steroid eye drops and started on bictegravir/emtricitabine/tenofovir alafenamide.

MANAGEMENT

Syphilis is known to progress through 4 different stages, each with unique signs and symptoms. During primary, secondary, or early latent syphilis the patient can often be cured with a single injection of long-acting benzathine penicillin-G. Late latent syphilis is treated with 2.4 million units of penicillin G once a week for three weeks. Once a patient develops syphilitic meningitis or ophthalmic syphilis, the treatment includes IV penicillin for 10-14 days.[1]

The hallmark of HIV treatment involves antiretroviral therapy. There are several classes of antiretroviral medications and regimens including nucleoside reverse transcriptase inhibitors, integrase strand transfer inhibitor-based regimens, non-nucleoside reverse transcriptase-based regimens, and protease inhibitor-based regimens. While these medications cannot cure HIV or AIDS, they are successful in attaining treatment goals which include maximally and durably suppressing plasma HIV RNA, the restoration and preservation of immunologic function, and the reduction of HIV-associated morbidity. This therapy also serves to prolong the duration and quality of survival, and finally to prevent HIV transmission.

DISCUSSION

Human immunodeficiency virus is generally acquired through sexual intercourse but may also be passed perinatally or through exposure to infected blood (IVDU, health care-related exposure). Acute HIV may be associated with vague symptoms such as fever, rash, diarrhea, and headache; however, 60% of individuals will be asymptomatic. The HIV attaches to the CD4 molecule and CCR5, which allows the virus to fuse and spill contents into T-cells. The virus integrates into the host genome, which eventually leads to viral budding and the release of more viruses.

Over time, a patient's T-cell count will drop, leaving them susceptible to successive AIDS-defining illnesses. The hallmark of treatment for HIV is known as highly active antiretroviral therapy (HAART). HAART often consists of drugs from at least two different classes of antiretroviral therapy such as nucleoside analog reverse transcriptase inhibitors, non-nucleoside analog reverse transcriptase inhibitors, and protease inhibitors.[2] Current recommendations call for HAART as quickly as possible after the diagnosis is made. For those who are diagnosed in the setting of an opportunistic infection, HAART should be initiated shortly after the infection has been adequately treated.[2]

Syphilis has been found to enhance the transmission of HIV, likely secondary to increased incidence of genital ulcerations, which promote spread. HIV patients are more likely to present initially with secondary syphilis as well as more rapid progression to neurosyphilis.[3] An LP is required for the diagnosis and patients found to be positive require an LP every 6 months until negative.[4] Early treatment of these patients is imperative, and the treatment of neurosyphilis requires nearly 2 weeks of IV penicillin.

References available online.

Ramadan and the Emergency Department:
Compassionate Care for Fasting Patients

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The holy month of Ramadan is a sacred time during which millions of Muslims worldwide embark on a profound spiritual journey marked by fasting, prayer, and reflection. In the emergency department, this period presents unique challenges and opportunities for health care providers. Fasting can impact the health of patients with chronic or acute conditions, making it essential for emergency clinicians to navigate both the medical and cultural dimensions of care. By understanding the practices and significance of Ramadan, clinicians can ensure compassionate, respectful, and effective treatment for Muslim patients, even in critical moments.

ISLAM AND THE HOLY MONTH OF RAMADAN

Ramadan, the ninth month of the Islamic lunar calendar, is a sacred time of spiritual devotion, worship, discipline, and self-restraint from worldly activities.[1] It marks the period when the Prophet Muhammad is believed to have received the first revelations of the Quran, Islam's holy scripture.[1] Fasting during the month of Ramadan is one of the Five Pillars of Islam, which are the foundation of a Muslim's faith and practice. The other pillars include the declaration of faith (*Shahada*), performing five daily prayers (*Salah*), giving to the poor (*Zakat*), and undertaking the pilgrimage to Mecca (*Hajj*) once in a lifetime, provided one is physically and financially able.[2]

The observance of Ramadan begins and ends with the sighting of the crescent moon, symbolizing the start and conclusion of the month.[3] This year, Ramadan is projected to start on Feb. 28, 2025. Because the Islamic lunar calendar is shorter than the solar calendar by approximately 10–12 days, Ramadan shifts earlier each year, cycling through all seasons over a 33-year period.[2,3] This progression introduces unique challenges, as fasting during the summer months can be particularly difficult due to longer daylight hours, whereas fasting in winter is often more manageable.[4]

During Ramadan, Muslims refrain from eating, drinking, smoking, and sexual activity from sunrise to sunset; this often includes refraining from taking medications.[2] A typical day in Ramadan is centered around fasting, prayer, reflection, and community. It begins with *Suhoor*, a pre-dawn meal, followed by the morning prayer, *Fajr*. Muslims then fast from dawn to sunset, abstaining from food, drink, and other physical needs, while focusing on spiritual growth, patience, and charity. During the day, prayer, Quran recitation, and acts of kindness are emphasized.

The fast is broken at sunset with *Iftar*, often starting with dates and water, followed by a communal meal with family and friends. The evening concludes with the *Tarawih* prayers, special nightly prayers performed in local congregation, often at the local mosque.

References available online.

Ramadan concludes with the holiday of Eid al-Fitr, during which Muslims gather for a morning prayer, food, and gifts. However, emergency clinicians should be aware that some Muslims may continue fasting for six additional days after Ramadan.[5] This is because, according to Islamic tradition, these six days, combined with the Ramadan fast, are considered equivalent to fasting the entire year.[5] Additionally, many Muslims adhere to the tradition (Sunna) of the Prophet Muhammad by fasting on Mondays and Thursdays throughout the year.[1] Consequently, emergency clinicians may encounter fasting patients even if it is not Ramadan.[1]

EXEMPTIONS TO FASTING

In the event of a health emergency, Muslims are permitted to break their fast, as fasting is intended to be a spiritual challenge rather than a risk to one's health; fasting that poses a danger to health is not in accordance with Islamic teachings.[1,6] However, it is important to recognize that although Islam is a flexible religion, some Muslims may still choose to fast despite being ill.[2,7] Those who are pre-pubescent, elderly, pregnant, breastfeeding, ill, traveling, menstruating, or have medical conditions that make fasting harmful are exempt.[2,8,9] Under these conditions, one can either make up the fast later or feed the needy as a charitable expiation.[6]

CONSIDERATIONS FOR THE EMERGENCY CLINICIAN

When providing care to a fasting patient, emergency physicians should carefully consider several key factors including dehydration, diabetes, pregnancy or breastfeeding, and medication management.

DEHYDRATION

Fasting patients visiting the ED during Ramadan may present with dehydration, especially when fasting during longer, hotter summer days. Dehydration can manifest as headaches, fatigue, or hypotension. In such cases, emergency physicians should recognize that interventions like intravenous fluid hydration and enteric medications (e.g., oral acetaminophen) will invalidate the fast, which may be a concern for the patient (medications are covered in "Medications in the Emergency Department").

DIABETES

Recent studies indicate that individuals with chronic conditions (such as diabetes, asthma, hypertension, and renal failure) often adjust or discontinue their treatment regimens during Ramadan without consulting their healthcare providers.[10,11] A recent study found that Muslim patients with diabetes are hesitant to report self-directed changes in medication due to feelings of vulnerability and being misunderstood by their providers.[12] As a result, patients may present to the ED with serious conditions, such as hypoglycemia, hyperglycemia, or electrolyte derangements. In these cases, it is essential for ED clinicians to engage in empathetic discussions with patients and their families about the risks and benefits of interventions that may provide relief but invalidate the fast.

PREGNANCY AND BREASTFEEDING

Research shows that most pregnant Muslim women do not consult with their physicians before deciding to fast during pregnancy.[13] Approximately 88% of pregnant Muslim women choose to fast during Ramadan, with a higher incidence among immigrant women when compared to U.S.-born.[14] Although pregnant and breastfeeding women are exempt, many still choose to fast to meet their spiritual needs. However, many remain flexible and may discontinue fasting if health issues arise, aligning with Islamic principles of avoiding harm.[14] Breastfeeding is another exemption to fasting, as it may affect milk supply and micronutrients, which can also lead to unwanted stress and in some cases decrease milk supply.[8,9]

MEDICATIONS IN THE EMERGENCY DEPARTMENT

Several medications could invalidate a fast, including any enteric medication, IV fluids, nebulized medications, and blood transfusion. Generally, medications administered intravenously (IV), intramuscularly (IM), or subcutaneously (SubQ) are permissible and do not invalidate the fast (see Table 1 for exemptions), thus medication routes should be adjusted when possible.[1]

Although challenging in the emergency department, patients on chronic medications could benefit from adjusting their schedules to coincide with non-fasting hours.[16] Consideration of long-acting formulations (e.g., sustained-release) or altering dosing regimens to

Table 1. Acceptable medications during Ramadan fasting

• **Sublingual medications/lozenges** ◦ Not permitted: nicotine	• **Topical medications/patches**	• **Creams, ointments, lotions**
• **Vaccinations** ◦ Not permitted: PO vaccines	• **Metered-dose inhalers** ◦ Not permitted: nebulizers	• **Injected medications via IM, IV, SQ** ◦ Not permitted: fluids, glucose, electrolytes
• **Insulin therapy**		

Table 2. Acceptable procedures relative to Ramadan observance

• **Oxygen supplementation**	• **Contrast dye for imaging studies** • **Not permitted: oral contrast**	• **Nasal packing**
• **Pelvic exam**	• **Foley catheter insertion**	• **Endoscopy**

Adapted from ALiEM's *Emergency Medications and Procedures That Are Permitted while Fasting*.

References available online.

once or twice daily may help optimize medication management during fasting periods.[16] It is important to advise patients to consult their primary care doctors, as they may need guidance not only on modifying their medications but also on making lifestyle adjustments to mitigate potential health risks during fasting.

PRACTICAL ADVICE TO THE EMERGENCY CLINICIAN

Many Muslims, even when faced with medical conditions that may endanger their health, may still choose to fast.[2,7] Barriers to discussing fasting include negative past experiences, fear of disrespect, or blanket prohibitions without culturally sensitive explanations.[15] In these situations, it is essential for emergency clinicians to approach patients with humility, empathy, and compassion, fostering trust and enhancing the patient-physician relationship.[17]

Interventions such as enteric medications, IV fluids, or blood transfusions invalidate fasting, but may be medically necessary and should be approached through shared decision-making and empathy.[1] A gentle reminder that breaking the fast for health reasons is permissible and can be compensated later can help build trust and support the delivery of optimal care.[1] Furthermore, encouraging adequate nutrient intake and hydration during the pre-dawn and evening meals (*Suhoor* and *Iftar,* respectively) may help prevent myriad conditions including dehydration, lightheadedness, syncope, electrolyte derangement, and hypoglycemia.

By understanding Islamic beliefs and recognizing how religious practices shape the lives and health behaviors of Muslim patients, clinicians can foster trust, improve communication, and provide culturally sensitive and medically effective care.[17] This approach enhances the patient-physician relationship and ensures that patients feel respected and supported in their health care decisions, ultimately contributing to better health outcomes and a more inclusive environment.

TAKE-HOME POINTS

- During the month of Ramadan, many Muslims fast from sunrise to sunset, which may include altering or discontinuing medication plans.
- Muslim patients experienced higher rates of ED visits during Ramadan.
- Fasting that poses a danger to health is not in accordance with Islamic jurisprudence, and those with chronic illness are exempt from fasting. However, many may still choose to fast.
- Patients are more likely to follow the advice from providers who are understanding and knowledgeable about their religious practices.
- Compassion, respect, and understanding of Ramadan can foster trust and contribute to better health outcomes.

Fire in the Canyons:
EMRA MedWAR 2024 at Red Rock Canyon National Recreation Area

EMRA's MedWAR returned to its roots in Red Rock Canyon in 2024, celebrating a milestone as EMRA marked 50 years of serving residents.

PRELUDE

The sun rose over the eastern horizon at Red Rock Canyon. Deep canyons and towering cliffs, recently shadowed, lit on fire in a mixture of orange and red. The cool air began to give way, hinting at the coming heat of the day. Past the starting line, a trail wound its way into the steep washes and canyons adjacent to the Calico Hills. It was Oct. 2, 2024, and a challenging course with tests of medical knowledge lay ahead for the 11 teams competing in EMRA MedWAR 2024.

A BRIEF HISTORY OF MEDWAR

EMRA MedWAR is an annual event held yearly as part of EMRA's programming during ACEP Scientific Assembly. MedWAR was founded in 2000 by Michael Caudell, MD, FAWM, DiMM, and David Ledrick, MD, at the Medical College of Georgia. Teams navigate a wilderness course, completing medical scenarios and answering questions along the way. Since its inception, MedWAR has expanded to include events throughout the country, including the first EMRA MedWAR in 2016 at Red Rock Canyon.

For each EMRA MedWAR since, the location uniquely represents the landscape and history of the host city, whether Wissahickon Valley Park in Philadelphia, Lake Chabot Regional Park in San Francisco, Blue Hills Reservation State Park in Boston, a virtual edition in 2020, Cuyamaca Rancho State Park in San Diego, or Seneca Creek State Park in Washington, D.C. The 2024 return to the Mojave Desert was a reminder of the stark and unforgiving landscape surrounding Las Vegas.

PREPARATION

Preparation for MedWAR began months in advance through the efforts of the EMRA Wilderness Medicine Committee, EMRA staff, and faculty advisors Hillary Irons, MD, PhD; Mike Caudell, MD, FAWM, DiMM; and Taylor Haston, DO, DiMM, MPH, MS. A site was selected, a course was charted, scenarios and questions were written, logos and swag were designed, sponsors were brought on board, and permits were acquired. Special thanks should be extended to the wonderful rangers at Red Rock Canyon NRA, who helped with course design and event planning. Teams signed up the summer before the race, preparing physically for the demands of the course and mentally for the array of wilderness medicine topics they might encounter.

THE DAY OF THE RACE

Racers arrived at 7:30 am for check in, greeted by the expanse of the harsh Mojave landscape at Red Rock Canyon. This year's EMRA MedWAR became the hottest MedWAR on record, with temps over 103°F for much of the race. While racers fueled with coffee and food, volunteers, including members of the Las Vegas Metropolitan Police Department Search and Rescue team, ventured into the desert to set up scenarios. At the same time, race directors set up emergency cooling and hydration stations and trained volunteers on heat stroke and hyperthermia treatment. Fortunately these efforts paid off, as no racers or volunteers suffered significant heat illness throughout the day.

The first challenge racers met was a mass triage incident that required correct disposition and transport decisions for victims of a lightning strike. After this, they embarked towards the valleys south of the Calico Hills. Scenario 1, set in

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References available online.


Bring it.
EMRA MedWAR 2025
Compete Sept. 10!
Team sign-up due July 10
EMRA
MEDWAR

the barren, exposed desert, presented an altered heat stroke patient with a cactus spine protruding from their eye. Scenario 2 added a toxicologic flavor as teams identified deadly plants and decided which one a pediatric patient had snacked on. The third and fourth scenarios, situated along the rolling sandstone hills, consisted of a rattlesnake bite and a testicular torsion. Traumatic injuries awaited teams at the far end of the course. Participants encountered a fallen climber with a spinal cord injury and a mountain biker with a pneumothorax and SVT.

Despite the heat and exposure, all teams returned within the 6-hour time limit, exhausted and a bit dehydrated but with shade and cool drinks awaiting at the finish line. The champion of the day was "Dora the Rxplorers" from Yale Emergency Medicine, followed by UCSF Fresno in second place and Ohio State in third.

Thank you again to all the racers and volunteers who braved the heat to participate; we look forward to having you back next year!

SUMMARY

MedWAR is a time-honored tradition that brings together residents and medical students from around the country, no matter the setting. This year, racers from far flung locales including Florida, Connecticut, Ohio, and California, among many others, came together to share in their passion for emergency medicine and wilderness. The 11 teams raced through the desert during the hottest EMRA MedWAR yet. They displayed resilience, sportsmanship, and a sense of adventure, embodying the spirit of the great explorers and physicians who came before them. These racers, many of whom will become attending physicians next year, leave no doubt of the passion and joy they will bring to their work and the field of emergency medicine.

For those interested in helping out with next year's MedWAR, find details here: https://www.emra.org/be-involved/events--activities/emra-medwar or email emrawildernessctte@emra.org.

There are also several regional MedWAR events throughout the country. Check out www.medwar.org and @MedWAR17 on Twitter/X for dates and locations.

For those interested in contributing to the conservation efforts at Red Rock Canyon National Recreation Area, further information can be found here: https://www.friendsredrock.org/

For those interested in contributing to the SAR efforts of the LVMPD, further information can be found here: https://lvmpdsar.org/

References available online.

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5-P021

Delayed Presentation of Gluteal Compartment Syndrome Complicated by Sciatic Neuropathy Following Prolonged Immobilization Due to Opioid Overdose: A Case Report

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Compartment syndrome is an orthopedic emergency that can be difficult to identify but can lead to significant morbidity and mortality if unidentified or left untreated.

It results from high intra-compartmental pressures which can cause ischemia of the affected tissue, necrosis, and nerve damage.[1] A major risk factor for developing compartment syndrome is prolonged immobilization, either secondary to recent surgery, trauma (especially pelvic trauma or crush injury), or in the setting of alcohol/substance use.[2]

Gluteal compartment syndrome is a rare type of compartment syndrome that is often difficult to identify and/or diagnose. There are three gluteal compartments, and any of the three may be affected by gluteal compartment syndrome. These include the anterior tensor fascia lata compartment, the gluteus medius/minimus compartment, and the gluteus maximus compartment.[3]

Inadequate perfusion may lead to irreversible damage; thus, early recognition of compartment syndrome is imperative as immediate fasciotomy is required to improve patient outcomes.[4] Tissue ischemia from compartment syndrome may lead to rhabdomyolysis. The muscle

References available online.

ischemia and fluid sequestration from compartment syndrome can also lead to hypovolemia, further perpetuating acidemia and acute kidney injury. The sequelae of rhabdomyolysis may include hyperkalemia resulting in life-threatening dysrhythmia, and myoglobinuria resulting in deposition in the distal renal tubules causing tubular cast formation causing acute renal failure.[4] Ultimately, this may lead to organ failure or possibly death if not addressed and treated early.

Other potential complications of gluteal compartment syndrome include peripheral neuropathy, complex regional pain syndrome, and sciatic nerve palsy. The sciatic nerve is particularly vulnerable as it may be compressed by compartment syndrome or expanding hematoma leading to potentially irreversible damage or lasting neuropathy/nerve palsy.[5] Early diagnosis and fasciotomy are necessary to improve functional prognosis in addition to mitigating morbidity/mortality risks.[6]

Gluteal compartment syndrome is complicated to diagnose as no definite diagnostic guidelines exist, and it has limited documentation in the literature. As it is largely due to prolonged immobilization, it may be difficult to identify if patients are in a state of altered consciousness or unable to provide history or subjective complaints. One of the preeminent symptoms of compartment syndrome is pain out of proportion to physical examination findings;[7] however, patients who cannot provide immediate history are unable to verbalize that they are experiencing unilateral gluteal pain, and thus identifying a tense compartment may be easily missed on initial physical examination. Gluteal compartment syndrome may also be misdiagnosed as a buttock abscess, hematoma, or deep venous thrombosis, which may delay definitive management.[8]

A thorough secondary examination is imperative in identifying any concerning unilateral extremity findings concerning for compartment syndrome, especially if history is noncontributory as noted above. There are other clues we may use; however, that may prompt re-evaluation and consideration of compartment syndrome. This includes the development of rhabdomyolysis as indicated by renal dysfunction, acute renal failure, electrolyte derangements, myoglobinuria, rising lactate, decreased sodium bicarbonate levels, leukocytosis, or inflammatory markers. Although many of these serum biomarkers may be within normal ranges, the combination of lab derangements may help prompt thinking about possible compartment syndrome, especially in the setting of a rising CK level as it is a significant indicator of myocyte damage.[7]

In the following case, we will discuss a 24-year-old male patient who presented to the emergency department after being found unresponsive and in cardiac arrest by EMS, who had return of spontaneous circulation after EMS initiated ACLS protocol.

CASE REPORT

A 24-year-old male with a past medical history of asthma and substance use disorder presented to the emergency department after return of spontaneous circulation was achieved by EMS. Per EMS, the patient was last seen well by his grandmother in the house the night prior and was found unresponsive the following morning. On EMS arrival at the home, the patient was unresponsive and pulseless. He was noted to have an open needle next to him, vomitus present on his person, and noted to have pinpoint pupils on examination. He was found to be in asystole on paramedic arrival and ACLS was initiated. He was given 16 mg total of intranasal naloxone and received 3 doses of epinephrine with ongoing CPR. The patient at one point was noted to be in PEA arrest, and subsequent return of spontaneous circulation was achieved. Afterward, the patient was intubated in the field by EMS.

On EMS arrival to the emergency department the patient's heart rate was 131, blood pressure 162/121, intubated and receiving manual respirations, oxygen saturation of 100%. The patient was transferred to a ventilator, respiratory therapy provided suctioning, and the patient was noted to have spontaneous respirations with rhonchorous breath sounds auscultated bilaterally. The patient was noted to appear tremulous, possibly with subtle tonic-clonic movements. He also had pinpoint pupils. As there was concern for possible seizure activity and ongoing overdose, the patient was given an additional 0.4 mg IV naloxone push as well as two 2 mg midazolam pushes, both for possible seizure activity and for additional sedation as the patient was breathing over the ventilator. Given that the patient was hypertensive and there was possible seizure-like activity, a propofol drip was started at 20 mcg/kg/min. The patient was initially not febrile on axillary temperature. An i-STAT showed the patient was hyperkalemic, with a potassium of 6.7 and an EKG was immediately obtained which revealed slightly peaked T-waves. The initial pH on i-STAT was 7.0, PCO2 was elevated at 88. POC glucose of 77. Given the hyperkalemia and acidosis, the patient received 1 amp of bicarbonate, 2 g of calcium gluconate, 1 amp of dextrose, and 10 units of insulin. The patient was started on a precedex drip for more adequate sedation. On secondary examination of the patient, no obvious traumatic injuries were noted. CT scan of the head without contrast showed no acute intracranial hemorrhage or mass effect or CT evidence for acute anoxic injury.

Initial labs were significant for leukocytosis with a white count of 29.8 and neutrophilic predominance, hemoglobin 12.5, potassium of 7, anion gap of 25, BUN 26, creatinine 3.1, AST 164, ALT 111, CK 27,000, high-sensitivity troponin 121. The urine toxicology screen was positive for cannabinoids, fentanyl, and cocaine. UA showed 2+ albumin, 4+ glucose, 3+ hemoglobin, 10 WBCs, 8 RBCs, and slight bacteria. One gram of ceftriaxone was given for concern of pneumonia. The patient was then admitted to the medical ICU for further management. Targeted temperature management was initiated with a target temperature of 36C. He was noted to have a tense right lateral gluteal compartment on examination, raising

References available online.

concern for compartment syndrome, particularly as there was evidence of rhabdomyolysis and acute renal failure in his labs. Trauma surgery was consulted due to suspicion of compartment syndrome. The patient was noted to have intermittent generalized tonic-clonic movements, particularly of his bilateral upper extremities, initially thought to be shivering due to targeted temperature management. He was given aspirin and buspirone without effect. The patient continued to have these movements, raising further concern for seizures. He was given 2 mg midazolam, which immediately stopped the movement. His propofol drip was discontinued and he was started on a midazolam drip.

Trauma surgery evaluated the patient and confirmed the suspicion of compartment syndrome. On examination, the right gluteus was firm to touch with overlying skin changes, non-compressible compartments. The patient with myoclonic jerks, on high levels of sedation, could not participate in the exam. The decision was made to bring the patient to the operating room for gluteal fasciotomy.

The patient went to the operating room for a fasciotomy. The deep compartments were tight and bulging and the overlying fascia was opened with satisfactory release of tension. All compartments in the right gluteal area were adequately decompressed. The patient remained intubated and sedated and returned to the MICU. The patient's hospital course was complicated by rhabdomyolysis with acute kidney injury, compartment syndrome status post fasciotomy right lateral gluteal compartment, and MSSA pneumonia. His course was also complicated by ICU delirium/agitation requiring a precedex drip. On hospital day 5, the patient underwent SBT but failed extubation and required re-intubation due to persistent agitation. He developed MSSA pneumonia, for which he received IV piperacillin/tazobactam. On hospital day 10, he underwent surgical closure of fasciotomy, after which he was able to be extubated and was transitioned to high-flow nasal cannula. The wound VAC was removed on hospital day 13. On hospital day 14, the patient was downgraded to intermediate level of care and ultimately left AMA prior to reevaluation by the trauma surgical service.

The patient presented to the emergency department 2 days later due to right lower extremity weakness/difficulty walking. He was found to be anemic, with a hemoglobin of 5.7 and had a palpable hematoma to fasciotomy site. Trauma surgery was again consulted. The patient underwent a CT scan of the right lower extremity with contrast, which revealed a small, elongated hematoma in the soft tissue lateral to the proximal femur at the site of recent surgery.

In the emergency department the patient received 1 unit PRBC transfusion. The patient was booked for operative exploration and hematoma/possible abscess evacuation with trauma surgery, with plans to be admitted to the trauma service afterward. Unfortunately, while waiting to go to the operating room just a few hours after agreeing to surgery and signing his consent for surgery, the patient again left against medical advice.

Two days later, the patient again presented to an outside emergency department with concern for wound dehiscence and worsening lower extremity numbness. He underwent another CT scan showing the hematoma as noted on the previous scan. He also continued to be anemic, with a hemoglobin of 6.6, and received 1 unit PRBC transfusion in the emergency department. Transfer to the main hospital for tertiary-level trauma care was arranged to again be evaluated by trauma surgery. The patient was transferred and admitted to trauma surgery service. The wound was examined at the bedside by the trauma team, and a single suture was removed to allow for evacuation of a large clot just beneath the suture line. His wound was irrigated and the suture was replaced. The wound did not show any secondary signs of infection and the patient's pain was well controlled. A PM&R consultation was requested for concerns for right foot drop. PM&R recommended outpatient nerve conduction studies and a lightweight AFO boot to help with ambulation. He also received education on wound care so that he could do it himself at home. Given his hemodynamic stability and appropriate pain control, he was deemed safe for discharge 19 days after he first presented to the emergency department.

The patient was seen by PM&R almost 6 months later on an outpatient basis. His neurological exam was consistent with severe left sciatic neuropathy, basically in the peroneal distribution. The prognosis for neurological recovery is unclear at this time but there is no significant recovery thus far at 6 months, so recovery is likely to be far from complete as there is certainly a severe axonal component. EMG/nerve conduction studies were considered for prognostic information but unlikely to change treatment and the patient would likely not be able to tolerate this study at this time. As he had significant neuropathic pain, the patient was given ramp-up instructions for gabapentin. If severe pain persists then the patient will likely be referred to a pain clinic. The patient was given instructions for PT for aggressive range of motion at the right ankle and desensitization. If the patient cannot get good range at the ankle, the patient may be a candidate for Achilles tendon release. The patient was placed on an aggressive home exercise regimen.

DISCUSSION

Early diagnosis of gluteal compartment syndrome can be easily delayed in the altered or unresponsive patient. This diagnosis should be considered by the emergency medicine physician in any patient with prolonged immobilization or another pathogenic mechanism such as trauma or drug overdose. The most frequent symptoms of gluteal compartment syndrome include severe pain, especially with passive motion around the affected buttock, tenseness and/or unilateral swelling.[6] However, as noted in this case, without the subjective finding of pain expressed by the patient, this may be easily missed if not considered by the emergency physician. Therefore, the emergency physician needs to consider

References available online.

	Day 1 (ED) 16:00	Day 1 (ICU, pre-op) 21:00	Day 2 (5 hrs Post-op)	Day 3	Day 7	Day 14 (Left AMA)
pH	7.01		7.24			
Serum Creatinine	3.1	2.4	2.0	2.6	2.7	1.8
Serum HCO3	19	16	10	20	23	21
Anion Gap	25	22	25	9	12	15
Serum Potassium	7.0	5.0	5.0	4.1	4.3	4.7
Creatine Kinase	27,671	59,990	62,613	33,233	2,881	
White Blood Count	29.8	15.1	17.9	12.6	14.4	13.7
AST/ALT	164/111	360/173	491/217	410/206	130,16	
Lactate	10.6	9.9	13.5	1.2	1.3	

Table 1.

this diagnosis in any patient with a concerning recent history with other clues such as up-trending serum biomarkers or other evidence of rhabdomyolysis or multiple organ failure if the diagnosis is delayed as noted in this case (demonstrated by Table 1).

It is also imperative to complete a thorough primary and secondary examination of any undifferentiated patient who is altered or unresponsive presenting to the emergency department in order to minimize delay in management of this surgical emergency. As noted with the patient in this case, a delay in the identification of gluteal compartment syndrome may also lead to functional deficits even if the patient recovers from their life-threatening injuries and systemic sequelae.

Gluteal compartment syndrome is largely a clinical diagnosis as there are no standard diagnostic guidelines for this rare condition. If suspected, surgical consultation should not be delayed to try to obtain further imaging or compartment pressures, although this may help in diagnosis or ruling out other causes of a tense gluteal compartment. CT scans and/or MRIs may help distinguish the extent of muscle edema and/or necrosis as well as any hematoma formation of the affected compartment.[6] In addition to early surgical consultation, it is imperative to also manage the systemic sequelae of compartment syndrome, such as fluid resuscitation for rhabdomyolysis and acute renal failure, and to address any concerning electrolyte derangements, although these may also improve after management of the compartment syndrome.

CONCLUSION

Our case demonstrates several rare yet potentially detrimental consequences of drug overdose: respiratory depression resulting in cardiac arrest, prolonged immobilization resulting in gluteal compartment syndrome causing rhabdomyolysis and metabolic derangements, ultimately causing the functional impairment of unilateral sciatic neuropathy and foot drop.[9]

The emergency physician must have gluteal compartment syndrome on their differential diagnosis in any patient who may have had prolonged immobilization with concerning systemic implications and up-trending serum biomarkers when history is not obtainable directly from the patient to avoid ensuing systemic and functional deficits/permanent disability. When this emergent condition is suspected from clinical evaluation, pathogenic mechanism, and further evaluation with laboratory workup, and possibly imaging/compartment pressures, early surgical consultation for management with fasciotomy should not be delayed.[6]

References available online.

Bad Arterial Material:
A Case of Leriche Syndrome

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Aortoiliac occlusive disease, otherwise known as Leriche syndrome, mostly affects older individuals with classic risk factors for peripheral artery disease (PAD).

Although typically a progressive disease, it has been less commonly seen to cause sudden onset critical limb ischemia like in our patient. It is important for the clinician to have a high suspicion for this pathology, consider a patient's risk factors, relevant medical history, and maintain a wide differential when performing the workup.

INTRODUCTION

Leriche syndrome, otherwise known as aortoiliac occlusive disease (AIOD) is due to severe atherosclerosis affecting the distal abdominal aorta, iliac arteries and femoro-popliteal vessels. Risk factors are the same as those for atherosclerosis and include hypertension (HTN), hyperlipidemia (HLD), hyperglycemia, elevated homocysteine, smoking, age, and family history.[1] There can be a wide range of symptoms, from being asymptomatic to intermittent claudication to a triad of claudication, impotence, and absence of femoral pulses.[1] Leriche syndrome is often progressive in onset as the thrombus slowly grows, with time for development of collateral vasculature. Thus, it is not very common to see Leriche syndrome resulting in sudden onset critical limb ischemia without prior symptoms, as is seen with this case. Our patient had held their oral anticoagulation prior to the event for an orthopedic procedure, which possibly triggered thrombus formation and sudden limb ischemia.

CASE PRESENTATION

We present a case of a female in her 80s with an extensive medical history, including congestive heart failure, nonobstructive coronary artery disease, obstructive sleep apnea, atrial fibrillation, left bundle branch block, implantable cardioverter defibrillator (ICD) placement, left atrial appendage thrombus, transient ischemic attack, polymyalgia rheumatica, hypertension, and hyperlipidemia. The patient was recovering from a trigger finger surgery that occurred earlier that morning. She presented at 1 am with acute onset bilateral lower extremity burning pain starting at 11:30 pm, when she got out of bed to go to the bathroom. This quickly progressed to complete motor and sensory loss of both lower extremities.

The patient denied any trauma and per family was in her usual state of health when she went to bed earlier that night. On initial examination, the patient appeared in severe distress with cold and mottled bilateral lower extremities. In the ED, providers were unable to find Doppler-able pulses of her lower extremities bilaterally. She endorsed no sensation in bilateral lower extremities up to her proximal thighs and had no motor function in either leg. The patient did have rectal tone but possible fecal incontinence as there was stool in the diaper.

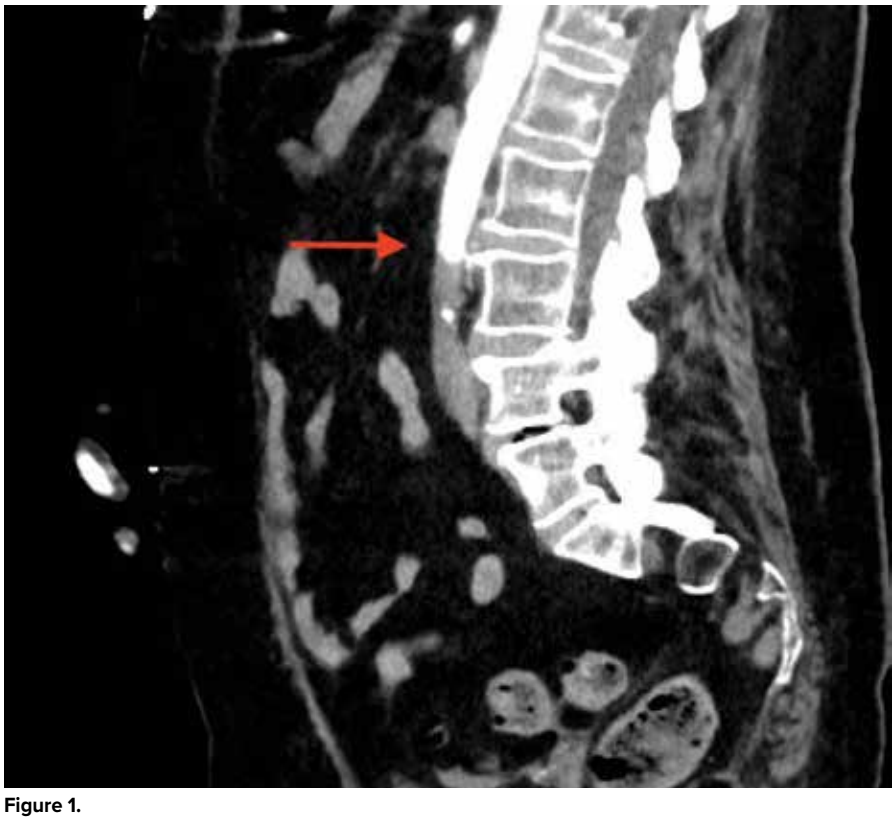

Figure 1.

References available online.

There was an immediate concern for an aortic catastrophe. She was taken for expedited CT angiography (CTA) after IV insertion and pain control. The CTA showed complete occlusion of the infra-renal abdominal aorta and bilateral internal iliacs with preserved external iliacs and 1 vessel run-off to the right lower extremity, 2 vessel run-off to left lower extremity and left renal infarct (**Figures 1 and 2**).

CTA from 4 months prior showed a patent aorta and iliac arteries without disease. Vascular Surgery was paged, and she was taken immediately to the operating room for emergent revascularization. She went to the surgical intensive care unit (SICU) after aorto-bilateral iliac suction thrombectomy, left common femoral artery cutdown and patch angioplasty, bilateral 4 compartment fasciotomies with resolution of thrombus and restoration of flow seen on angiogram.

Her postoperative course was complicated by stress cardiomyopathy leading to cardiogenic shock and reperfusion syndrome with resultant multiorgan failure requiring significant inotropic and pressor support, worsening lactic acidosis, and multiple electrolyte derangements. A goals of care discussion was held with the family by the intensive care and surgery teams, and a DNR order was placed. The patient passed away roughly 48 hours after her initial arrival to the emergency department.

DISCUSSION

Our patient's clinical presentation was consistent with the most severe end stage clinical manifestation of this disease, as she had lower extremity paralysis, non-palpable lower extremity pulses, and complete occlusion of the infra-renal abdominal aorta and bilateral internal iliac arteries. Per chart review from a primary care visit in 2021, she denied complaints of leg claudication. She had an official diagnosis of HLD — however, not of PAD with no prior documentation in the chart of ankle-brachial index measurements or symptoms consistent with a PAD diagnosis. Prior computed tomography (CT) abdomen and pelvis from May 2023 showed a patent aorta and iliac arteries without disease; however, this was not a dedicated CT angiography study. It is possible she was having symptoms that went unnoticed for many years. It is also possible she developed significant collaterals to compensate as many patients are asymptomatic because of the development of collateral networks.[2] This makes it difficult to determine the exact incidence and prevalence of this condition.

Our patient also had a history of atrial fibrillation and had paused her Xarelto for the past 10 days in preparation for her trigger finger surgery that occurred that morning. An acute thromboembolic occlusion of the distal aorta and iliac arteries is more consistent with acute limb ischemia than chronically worsening peripheral artery disease. Per Wooten et al., 80% of these acute occlusions originate in the heart, and 70% of these cardiac emboli are caused by atrial fibrillation.[2] Diagnostic imaging in patients with atrial fibrillation presenting with acute thromboembolic occlusion resulting in critical limb ischemia also revealed the occlusion to be of the distal infrarenal aorta at the bifurcation point (similar to how our patient presented).[3] Both the pathology of the limb ischemia itself as well as the complications resulting from management, renal failure, fasciotomies, amputations, cardiomyopathy, and electrolyte derangements, result in significant pre- and post-procedure mortality.[4,5]

While this is a diagnosis that requires rapid intervention, it is still important to consider other vascular pathology when initially evaluating patients presenting with symptoms of aortoiliac occlusive disease. These include arterial aneurysm, arterial dissection, embolism, Giant cell arteritis and Takayasu, arteritis and venous claudication.[6]

CONCLUSIONS

In summary, aortoiliac occlusive disease is an overall rare presentation but one with high mortality that requires prompt diagnosis and intervention. Strongly consider this in the differential diagnosis with cases ranging from absent lower extremity pulses to intermittent claudication. Remember to review past medical history to determine risk factors that could predispose to this vascular abnormality and be sure to involve vascular surgery consultants as soon as possible when Leriche syndrome is suspected.

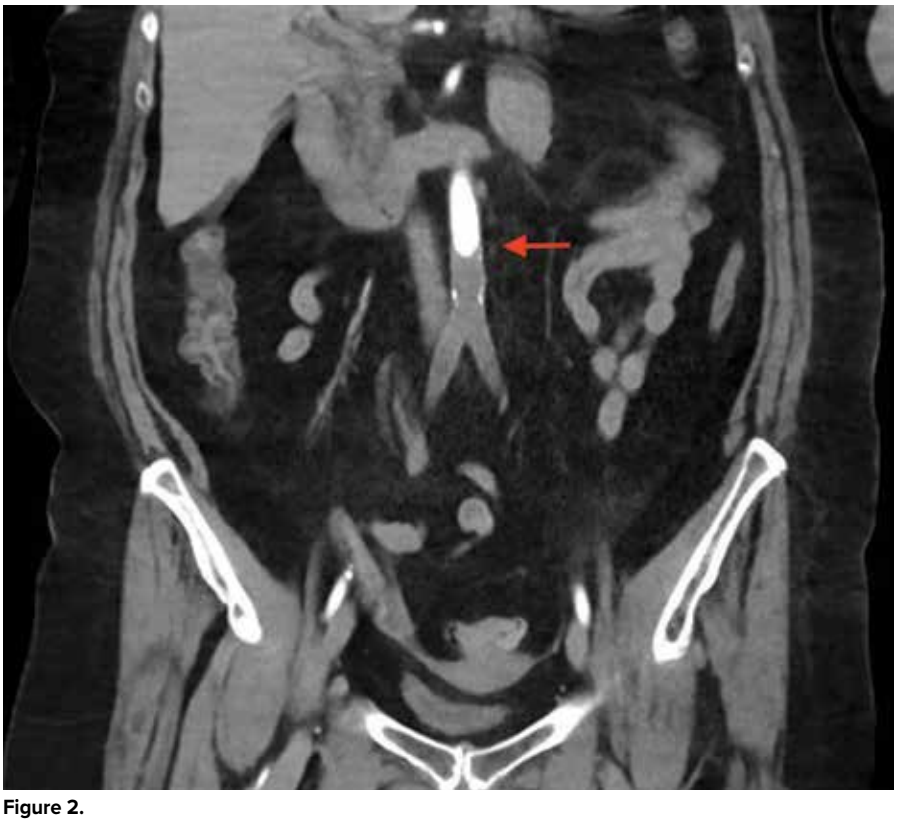

Figure 2.

References available online.

Case Report: Severe Chronic Argyria Secondary to Silver Supplement Use

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Colloidal silver is a popular supplement used for a spectrum of perceived health benefits. However, the FDA does not regulate it and, in fact, issued a final rule deeming it "not generally recognized as safe and effective."

Arising from excessive silver exposure, argyria is a rare condition characterized by a slate-blue discoloration of the skin and mucous membranes. This report highlights a specific dermatologic finding seen in chronic silver poisoning and discusses occupational, environmental, and pharmacologic risk factors.

Recognizing chronic argyria is important to the emergency physician in forming a targeted differential for the "blue patient" and avoiding unnecessary workups or medications that may harm the patient.

CASE PRESENTATION

A 79-year-old female with a history of chronic kidney disease stage V and chronic argyria presented to the ED with subclinical fever, fatigue, and mild joint pain. She had been experiencing these symptoms for 6 weeks and had multiple unremarkable laboratory work-ups in the ED. Incidentally, on physical examination she had deep bluish pigmentation of the face, conjunctiva, tongue, and upper extremities, which she stated is her baseline from her chronic argyria.

This patient's chronic argyria resulted from use of homeopathic silver-containing oral supplements she had been using to treat chronic sinus congestion for 8 years. She denied any chest pain, shortness of breath, or new skin discoloration. Her workup demonstrated a non-specific

Image 1. Skin color changes are a hallmark of chronic argyria

References available online.

leukocytosis of 13.8 thousand/mcL, and a mild anemia of 10.4gm/dL with a mean corpuscular volume of 103 fL. She also had a creatinine of 3.72mg/dL with a GFR of 12mL/min, both of which consistent with her baseline kidney disease. Urinalysis and remaining serology were unremarkable. Given the presence of non-specific symptoms and multiple prior ED visits without a definitive answer, CT imaging with contrast of the neck, chest, abdomen, and pelvis was obtained to evaluate for neoplastic lesions and presence of lymphadenopathy. There was no evidence of lymphadenopathy; however, a pathological T6 vertebral compression fracture with sclerotic lesions was noted. The patient was ultimately discharged with instructions to pursue outpatient follow-up for the sclerotic lesions and compression fracture.

CASE DISCUSSION

Argyria is a rare but important differential diagnosis, especially in emergency medicine, due to its distinctive appearance. Argyria is a characteristic dermatologic finding seen in chronic silver toxicity. The blue-gray discoloration can result from mechanical contact of silver particles on the skin or via inhalation or ingestion of silver particles. Silver particles that are systemically absorbed dissolve into silver ions, which can be reduced by sunlight or sulfur and lead to increased melanin production in light-exposed areas and blue discoloration when alongside selenium ions.[1] Chronic argyria is suspected to signal worsening kidney function, based on limited data.[2] While the link to long-term silver therapy and renal failure has not been definitely linked, effects of such therapy on the kidney should be considered when managing patients with argyria, especially when considering the administration of nephrotoxins. In addition, there are significant impacts to the cardiac, hepatic, and hematopoietic systems seen from silver toxicity, with manifestations such as ventricular hypertrophy, bone marrow suppression, hepatic necrosis, vertigo, seizures, and acute tubular necrosis.[1]

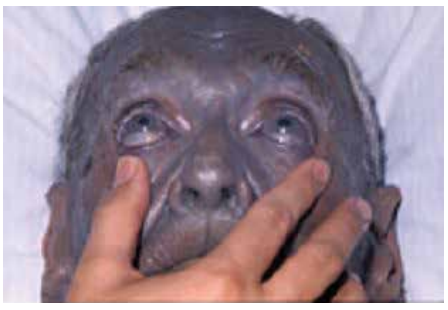

Image 2. Generalized argyria stemming from nose drops containing colloidal silver, resulting in permanent pigmentary changes. (credit Herbert L. Fred, MD and Hendrik A. van Dijk)

Chronic exposure to silver and the subsequent accumulation of these deposits are often due to either occupational exposure or use of silver-containing supplements. A higher degree of suspicion for generalized argyria should be held in those with long-term occupations involving the manufacturing of silver-containing products, including antiseptic agents, porcelain, dyes, and jewelry.[3] Others develop systemic argyria secondary to unregulated homeopathic supplements, as is the case for our patient, as well as buying colloidal silver, photography products, mirrors, plating, inks and dyes, and porcelain over the counter. Treatment of chronic argyria is difficult because it is irreversible and not amenable to chelation; however, there may be some benefit from topical hydroquinone 5% and laser technology.

TREATING THE "BLUE PATIENT:" WHAT ELSE COULD IT BE?

Apart from argyria, the differential for the "blue patient" is broad yet important for the ED physician to consider. Methemoglobinemia, a disorder of hemoglobin during which ferrous (Fe^{2+}) iron in hemoglobin is oxidized to the ferric (Fe^{3+}) state, is classically attributed to toxicity from local anesthetics, nitrates, dapsone, phenazopyridine, quinones, sulfonamides and other pharmaceuticals; however can also be seen in patients with sequelae of severe fire injuries, as well as those with hereditary conditions such as NADH methemoglobin reductase deficiency.[4] High-flow oxygen and methylene blue are the mainstays of treatment. It should be noted that large doses of methylene blue, typically greater than 7 mg/kg, have been reported to rarely cause initial blue skin discoloration and paradoxical methemoglobinemia.[3]

Blue skin pigmentation from chrysiasis may be seen in patients who have undergone prolonged parenteral gold therapy.[5] In cases of skin discoloration secondary to long-term amiodarone toxicity, medication review and addressing cardiac concerns are vital as blue pigmentation from amiodarone use has been shown to be reversible.[6] Overall, the ED plays a crucial role in diagnosis, initiation of appropriate treatments, and ensuring patient safety and comfort.

EM TAKEAWAYS

- Chronic argyria, characterized by severe blue skin discoloration due to excessive silver exposure, is a rare but noteworthy condition for emergency physicians to consider in the "blue patient."
- This case underscores the importance of a thorough evaluation and differential diagnosis in the ED, given the broad differential of dramatic blue skin discoloration.
- Long-term silver therapy can affect multiple systems, including cardiac, hepatic, hematologic, and kidneys, warranting careful consideration in patient management.

References available online.

EMRA ULTRASOUND POCUS For The Win!

Quadriceps Tendon Rupture

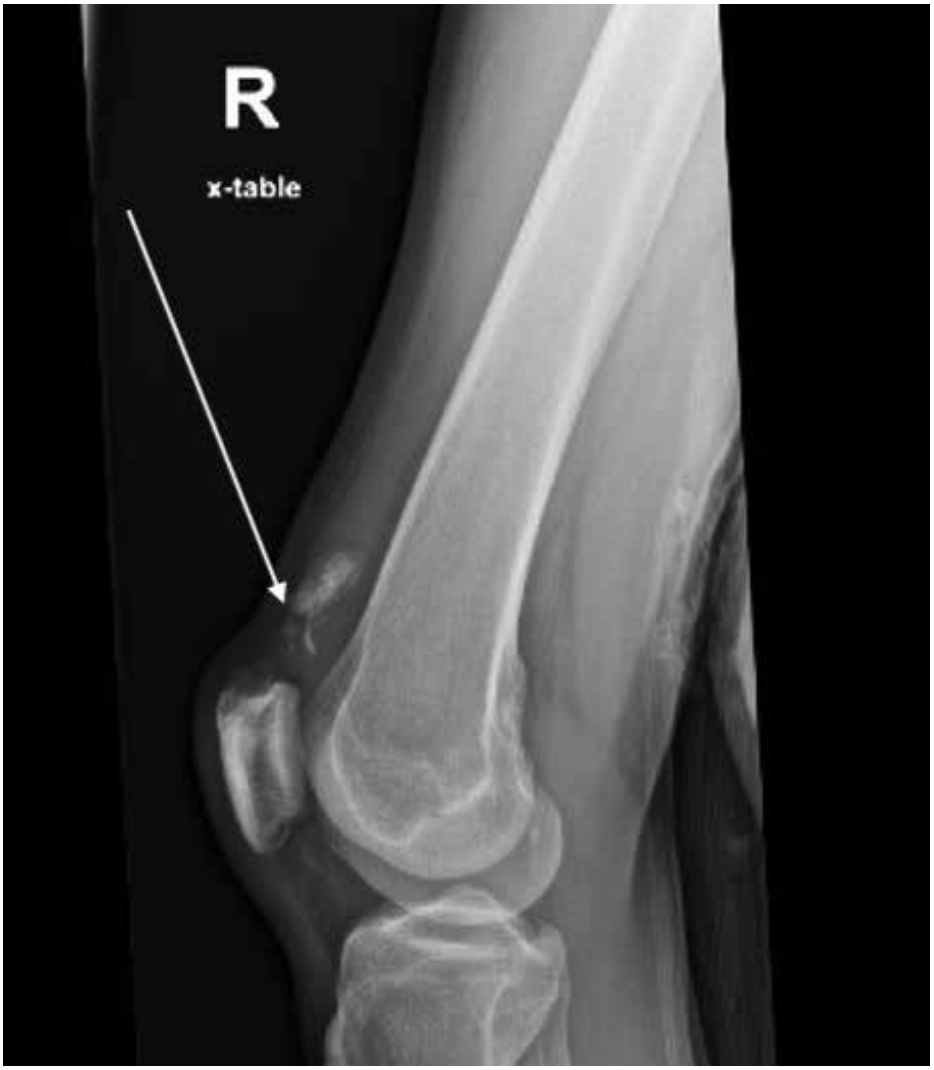

Image 1. Lateral X-ray of right knee showing bony avulsion of the superior pole of the patella

A 61-year-old male presents to the emergency department after feeling a pop in his right knee while planting his foot playing competitive table tennis. He has no known previous injuries to his right lower extremity. The patient reports pain and inability to bear weight or straighten his leg. Physical exam reveals a visible depression superior to the patellar pole (gap sign). He is unable to actively extend his knee against gravity. His distal pulses and sensation are intact. Radiographs and point of care ultrasound (POCUS) were used to assess for any fracture or soft tissue injury.

DIAGNOSIS:

Quadriceps tendon rupture (QTR) with avulsion of patella.

Radiographs were remarkable for a large ossicle anterior to the distal femur representing a fractured enthesophyte or superior pole patella avulsion fracture. (**Image 1**) POCUS was performed with

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References available online.

a linear probe (3.0 - 20 MHz) using the musculoskeletal preset. A long axis view was obtained revealing a disruption in the tendon's normal fascicular pattern consistent with a QTR. (**Image 2**) Hematoma formation is visible as an anechoic area deep to the tendon rupture. The avulsed patella appears as an irregularly shaped hyperechoic structure adjacent to the injured tendon.

ULTRASOUND TECHNIQUE

To perform POCUS of the quadriceps tendon (QT), have the patient supine in a position of comfort with the knee slightly flexed, using a rolled towel posteriorly for support. Use the high frequency linear probe and select the musculoskeletal preset. To optimize the image, further adjustments can be made to the depth and gain. The structure of interest, in this case the QT, should be evaluated in both the axial and longitudinal plane.[1] The axial view is obtained with the probe marker to the patient's right, starting three-quarters of the way down the patient's thigh and from there sliding the probe down to the patella. The fibrillar echotexture of four muscle structures of the quadriceps, including the rectus femoris, vastus lateralis, vastus intermedius and vastus medialis, is initially visualized. The four muscle bellies are visualized converging into the QT as the probe is slid distally to the patella. To obtain the longitudinal view, begin scanning in the same location as the axial view with the probe rotated 90° with the probe marker facing cephalad. Slide the probe caudally until the insertion site into the patella is visualized. Healthy tendons have a striated appearance with multiple, closely spaced hyperechoic parallel lines with echogenicity distinct from the surrounding soft tissue. Disorganized appearing fibers are suspicious for tendon injury. Hypoechoic or anechoic areas within the tendon represent disruption of the fibers, bleeding, or hematoma formation which are also indicative of tendon injury. In the longitudinal view, it is important to have the probe perpendicular to the tendon. If not completely perpendicular, hypoechoic areas may appear within the tendon leading to a false positive result. This phenomenon is known as anisotropy.[2] The integrity of the QT may also be examined dynamically by having the patient attempt to extend the knee while directly visualizing the tendon in the longitudinal axis. After thorough investigation of the QT, use the same technique to visualize the patellar tendon, evaluating its course from the patella to insertion into the tibial tuberosity. To complete the exam, examine the patient's non-injured knee and compare findings.

DISCUSSION

QTR is rare, typically occurring in patients 40 years old or greater after sudden changes in direction or jumping and landing.[3] Early diagnosis and surgical repair along with a strong rehabilitation program correlate to better outcomes.[4] Although MRI is the gold standard for diagnosing complete QTR, this is not always practical in the ED. Ultrasound is a time-efficient, economical approach to assessing acute knee injuries, especially when evaluating the integrity of tendons.[5] However, patients may require MRI for definitive diagnosis and operative planning as ultrasound has been reported to have false positive results in obese and muscular patients.[6] As with any POCUS, results are operator dependent and false positives may result from factors such as anisotropy or inability to visualize the injured area of tendon. Therefore, POCUS is most appropriately used in conjunction with a physical exam including straight leg raise, presence of a "gap sign", radiographs, and consideration of MRI. In this patient, we identified QT disruption, hematoma, and bony avulsion of the patella with POCUS. The patient was placed in a knee immobilizer and followed up with an orthopedic surgeon in his hometown.

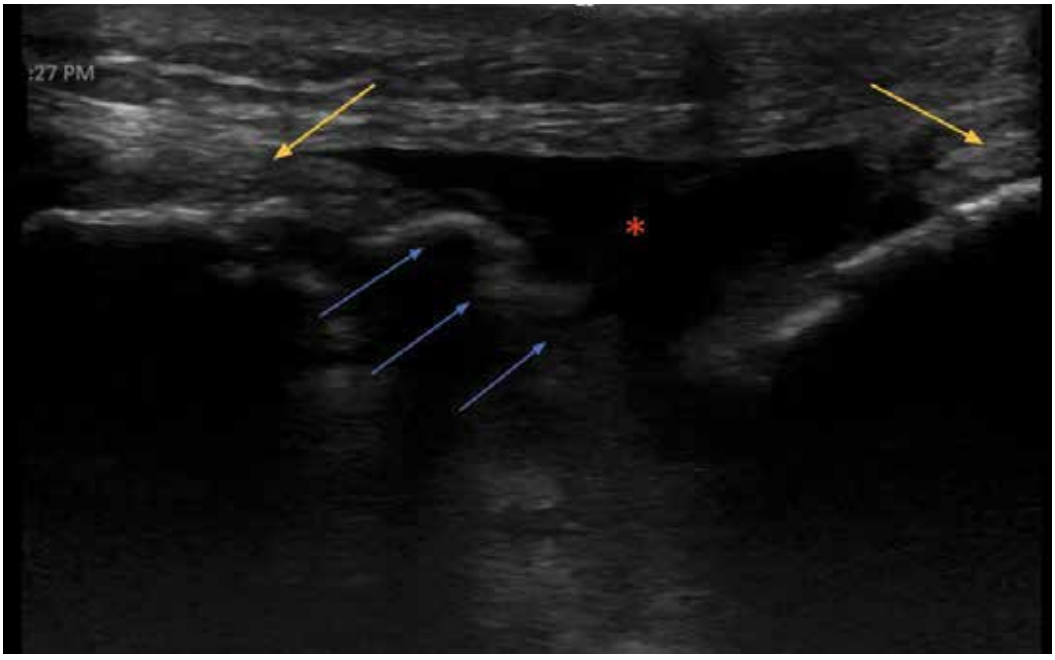


Image 2. Long axis view of the superior right knee. Asterisk (*) indicates hematoma, yellow arrows demonstrate discontinuity of the quadriceps tendon, and blue arrows demonstrate hyperechoic patellar avulsions.

References available online.

A Deep Dive with Angela Fiege, MD

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

empower
Sharing Our Stories

This edition of EMPower, we are seeking to learn from Dr. Angela Fiege.

When I did an elective rotation at IU Health last year, I was pleased to meet Dr. Fiege and have the distinct pleasure to work with and learn from her throughout the month. I was happy to receive an email recommending that I interview her for this publication! Though I learned a lot about her over the course of the month from her accomplishments on race tracks around the country, I was excited to learn more about her work with the non-profit she created and highlight it for others who are interested in learning more, sharing the work, and possibly doing similar work in their careers.

Dr. Fiege holds many titles in addition to Critical Care and Emergency Medicine physician at IU Health Methodist. She is the Medical Director for the Indy NXT series and deputy medical director for the NTT Indycar series, a track physician for the Indianapolis Motor Speedway, and Founder of the nonprofit **Rachael's First Week**. She serves as one of the nighttime critical care physicians at IU Methodist Hospital, where she cares for the sickest of the sick. She has worked as a track physician at the Indianapolis Motor Speedway since 2007. In the past, she has served as the Medical Director for the NASCAR / AMR Track Safety Team traveling weekends to NASCAR races across the country. Yet if you ask Fiege what her greatest accomplishment is, she will tell you it is founding Rachael's First Week, a non-profit organization she created as a legacy to her late daughter, Rachael.

FIRST THINGS FIRST, WHY EMERGENCY MEDICINE?

I think most people choose EM for the breadth — you get a little bit of everything. Because I started out as a flight nurse, I had EM in my blood already. There is always an adrenaline kick, but the speed at which you have to operate and the flexibility is quite fun. I couldn't see myself doing anything else. My critical care is very "emergency medicine" in style. It is the perfect blend when I'm taking care of high acuity patients.

IF YOU WERE RESTARTING RESIDENCY, WHAT ADVICE WOULD YOU GIVE YOURSELF?

Spend more time investigating the gaps in your learning. When you come onto shift, it's oftentimes that you're working as hard and fast as you can. Residency training is a great chance to be mentored and to do that self-introspection before you take on that role as an attending. It's important to work to find that balance between patient care and education. Use case-based learning when you can as well, because not everyone will have the same experience in residency and the same patient opportunities.

References available online.

WHAT IS YOUR GREATEST ACCOMPLISHMENT?

My daughter's legacy. Without a doubt.

TELL ME ABOUT RACHAEL'S FIRST WEEK. HOW DID YOU GET THE IDEA TO START THIS?

Rachael went to Indiana University. I had just finished a night shift when I got a phone call from the hospital that she was there. I couldn't fathom why she'd be in the emergency department, so I asked if she was on a ventilator, and they said yes. I got in my car and started driving. When I got to her bedside, I lifted up her eyelids, and I saw that her pupils were fixed and dilated. I knew at that time that her prognosis was poor. She had gone to an off-campus party with some people she knew. She fell down a flight of stairs. There was alcohol involved, and she was staggering around, so they brought her up to lay on the couch. When they checked on her later, she wasn't breathing, so they called EMS. She arrested a few times before they could get her to hold a blood pressure. They called me at that point. It was a complete life-changing experience, which led to me reevaluating everything in my life. I've been to pretty much every hospital in Indiana. It was the beginning of the college time period, and it became newsworthy. If you knew my daughter, the kind of person she was was always taking care of the less fortunate. She was very giving, and I wanted to honor the kind of person she was. I kept thinking about that, and I came up with the idea for an educational program. The other people at the party meant well; they just didn't know any better. The idea is for nonjudgmental learning points. It has grown from teaching at high schools all over. The entire residency class at IU came to the first one. We used the Youth Risk Surveillance Survey to come up with the curriculum.

TELL ME ABOUT THE IMPACT YOU'VE SEEN RACHAEL'S FIRST WEEK HAVE IN THE COMMUNITY.

We've talked to most of the colleges in Indiana, the freshman classes, and sororities and fraternities. We also talk to high schools at the end of their years before they graduate. The topics include the Indiana Lifeline law (amnesty law for students who call on behalf of another student if the user has alcohol involved), alcohol, stimulants, THC, sexual assault, mental health, and creating a new culture of looking out for each other. You're not with your parents anymore or anyone who will rescue you. It's about being a responsible human being. We use polls everywhere, and we compile the data for school counselors to have a pulse of what is going on with their students. We have two podcasts as well — SafeTea and Three College Idiots. It's hard to quantify prevention, but over 20,000 people have been spoken to about this topic. It's not infrequent to get a letter from a student or a parent, saying that something happened to them and that they remembered Rachael's story and called 911.

WHAT DO YOU THINK EM RESIDENTS CAN LEARN FROM RACHAEL'S STORY?

Find your passion. I would hate that anyone else would have to go through what I did to find their passion. If you chose this specialty, more than likely, you have a heart for advocacy. We're seeing every patient has a need, so find your niche, and try to make a difference. It doesn't have to be a huge project; the small projects also make a huge impact.

HOW HAS YOUR DAUGHTER'S STORY CHANGED YOUR PRACTICE?

I think it taught me how to talk to families and concerned others during the bleakest moments of their life. Sharing bad news is very important and very hard. I think having gone through that experience has made me more in tune to what people are going through in times of stress and how to support them. I've seen the whole spectrum of response, and I would not be the kind of physician I am without this experience. Showing people grace at a bad time in their life is very comforting during their suffering.

WHAT IS YOUR BEST TIME MANAGEMENT TIP?

Sleep. One word. I've been a night shifter for so long. You can't remember things or maintain a schedule when you are exhausted. Maximize your sleep, because residency is grueling.

WHAT IS THE BEST ON-SHIFT SNACK?

Whatever the staff have prepared for the patients! Be lucky if you can eat and pee on some shifts.

WHAT IS THE MOST RECENT BOOK YOU READ?

Playing in Traffic — about the Indycar Flagstand Start, who has autism. It covers how he went from feeling awkward as a child to overcoming adversity.

WHAT IS YOUR FAVORITE SONG TO HYPE YOU UP BEFORE A SHIFT?

I love listening to podcasts more so, like *Crime Junkie*.

References available online.

What's Important to You?

The EMRA Representative Council Fall Meeting takes place Sept. 8, with resolutions due July 25. This means now's the time to start thinking about what's important to you and how you want your professional organization to act on your behalf.

"[RepCo is] the single location in EMRA where ALL of the voices across the organization can be heard," said Speaker Jacob Altholz, MD. "No matter the geographic area or particular interest at hand, RepCo comes together and decides how we as an organization are going to see or advocate on certain issues, with all voices present."

You hear this multiple times per year: *Help set the direction of EMRA. Use your voice. Tell RepCo what you believe and how to advocate*.

EMRA Representative Council 2025 Fall Meeting

SEPT. 8, SALT LAKE CITY

- Each program sends 1 voting representative and 1 alternate councilor.
- All members are encouraged to show up and participate.

RESOLUTION DEADLINE: JULY 25

- Visit www.emra.org/repco for a resolution formatting template.
- Virtual resolution review takes place Aug. 25.

But does it actually work?

Consider these EMRA actions — all of which started with RepCo:

- **Full, active support of both 3-year and 4-year residency training;**
- **Support for better leave policies surrounding the birth or adoption of a child, or a death in the family;**
- **Advocating for better work schedules and working conditions for pregnant doctors;**
- **Opposition to the Standardized Video Interview (if you don't know what this is, Google it and then thank RepCo).**

"I also like to bring the AMA example: no smoking on airplanes started as a resolution from medical students," said Vice Speaker Ian Brodka, MD. "We can make a change in the world around us for the better."

Policymaking is not difficult, nor does it need to be boring. All it takes is an interest in the world around you.

"My dream 'slate' is a set of resolutions that are informed, cutting edge, and really seek to advocate for changes on issues affecting trainees directly," Dr. Altholz said. "Formatting can change, wording can change, but the identification of the issue and a possible solution is the single most difficult step in resolution writing. The rest can get hashed out."

And like emergency medicine overall, it's a team effort.

"We are happy to help, every step of the way if you reach out to us!" Dr. Brodka said. "Have thoughts about what you think your residency experience should be with rotations and procedures? Tell us with policy! Think EMRA should have more supporting stance on certain hot button topics and there's a gap in our current policies? Draft and submit what you want us to believe! Have opinions about AI use in the ED and think we should take the same stance? By all means, make policy for us to stand by!

"We always love to see involvement with our RepCo and to know what the membership of this organization wants to see from us."

Step Up as an EMRA Leader!

Join the governing force behind EMRA by running for a Board position. As an elected Board member, you'll steer the organization's future while representing your peers in emergency medicine.

2025 Elections

- **Candidacy deadline: July 25** *(Candidates can run from the floor of the meeting as well.)*
- **Election Day: Sept. 8** *(must be present)*

Positions to Be Filled

- **President-elect**
- **Vice Speaker**
- **Board Secretary/EM Resident Editor-in-Chief**
- **Director of Education**

What You Need to Know

TERMS & ELIGIBILITY

- **All positions require a 2-year commitment (3 years for President-elect).**
- **Any current resident or fellow can run and serve their full term — even if you graduate during your time in office.**

ROLES & RESPONSIBILITIES

- **Lead projects and represent emergency medicine at key events, plus actively participate in regular virtual meetings.**
- **Expect a significant travel commitment that complements your residency duties.** *(Travel is funded by EMRA.)*

CAMPAIGN ESSENTIALS

- **Discuss your plans with your program director, chief residents, and your family — support is crucial!**
- **Secure the necessary endorsements and prepare your application:**
  - A concise, 200-word platform statement
  - Your CV and a professional headshot
  - A letter of support from your program director
- **Familiarize yourself with campaign rules.**
- **Attend the Rep Council Fall Meeting, where you'll deliver a 3-minute speech, followed by a position-specific Q&A.**

CRAFT YOUR PLATFORM

- **Choose 2-3 key issues that resonate with EMRA's mission and strategically frame your campaign.**
- **Hone your speech with ample practice and mentor feedback to make a confident, compelling case for your candidacy.**

Are You Our Next National Leader?

Embracing a leadership role on the EMRA Board is a bold step toward shaping the future of emergency medicine. If you're ready to balance your residency with impactful service, now's the time to lead!

- **Talk to current officers to understand the ins and outs of the role firsthand.**
- **Scan the QR code to learn more about EMRA's mission, goals, and available Board roles.**

EMRA at ACEP25: Sign-ups and Save-the-Dates

EMRA's events at Scientific Assembly open the door for you to network, build your skills, and make incredible memories. We have something for everyone! EMRA events are free (except the MedWAR team fee) and do not require ACEP25 conference registration.

It really is the most high-yield conference time you can have. But why take our word for it?

Why EMRA?

EMRA Art Gallery 2024 People's Choice Winner Rafael J. Carillo Torres, MD

Dr. Carrillo's music entry, "armED", won the People's Choice Award at the EMRA art show in 2024. (Listen to it and see the full gallery here: https://www.emra.org/emresident/article/emra-art-gallery-2024.)

The whole experience of attending the EMRA events at Scientific Assembly was energizing, he said.

"I had the best time — it was amazing," Dr. Carrillo said. "It's the best. I'm so glad I did it! But I almost didn't make it," he added. "The flight got delayed and I was stressing, but it all worked out.

"If you have a chance to go, you should go," Dr. Carrillo said. "There's so much to do and so many people to meet!"

EMRA Events at ACEP25
Sept. 5-10 | Salt Lake City, Utah

SimWars: The Legend

Gather your team, get your flair (boring scrubs? nope), and come steal the championship at EMRA SimWars 2025.

- **Team sign-up deadline: June 8**
- **Competition date: Sept. 8**

Fall Medical Student Forum

Get your questions answered by program leaders & faculty, through general sessions and breakouts tailored to your phase of training.

- **Free event**
- **Date: Sept. 6**

MedWAR 2025

Head outside after a week of conferencing to compete in one of the most unique events in EM.

- **Team sign-up deadline: July 10**
- **Competition date: Sept. 10**

EMRA Residency Program Fair

Meet the programs! This is the original — and largest — EM program fair in the nation, and it's a can't-miss chance for programs and candidates to connect.

- **Free for attendees**
- **Date: Sept. 6**

Case-con

This is a golden opportunity to present at a national conference.

- **Application deadline: June 15**
- **Presentation date: Sept. 7**

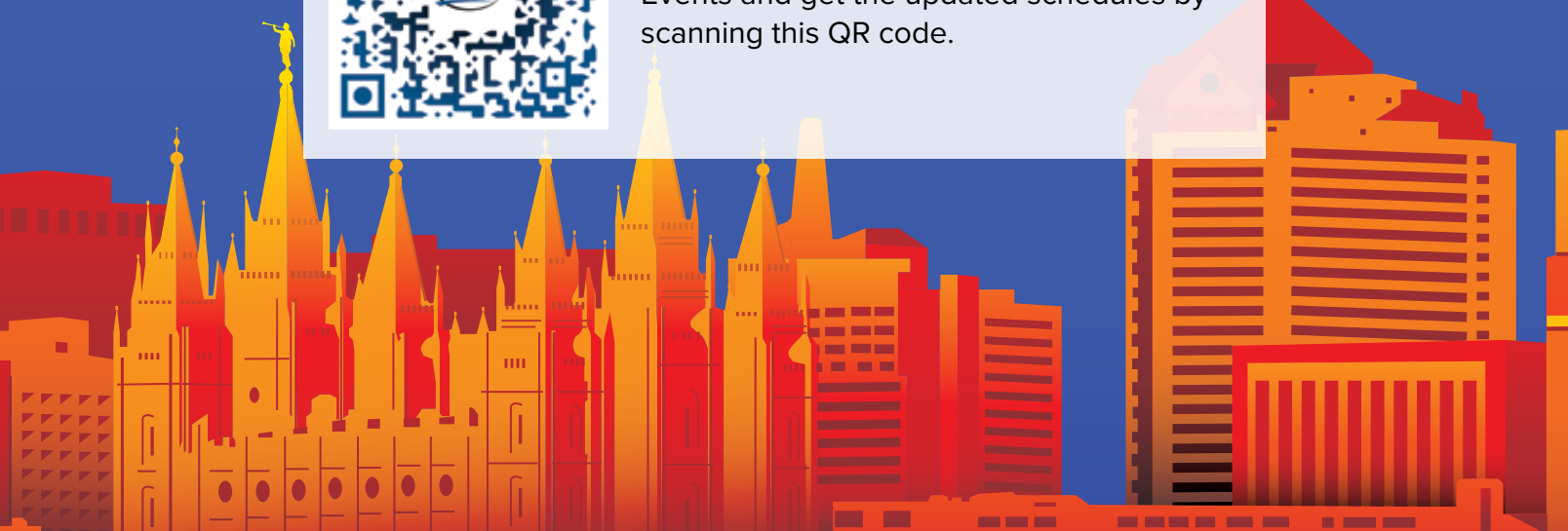
emCareers Job & Fellowship Fair

First brought to you by EMRA in 1990, this fair helps EM physicians find the next opportunity in their careers.

- **Free for attendees**
- **Date: Sept. 7**

EMRA Party, New Events, and More!

We've got big plans in the works, and you don't want to miss out. Find all our EMRA Events and get the updated schedules by scanning this QR code.



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EMRA ECG Challenge

Saira Khan, MD
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Jeremy Berberian, MD
Associate Director of Emergency Medicine
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Department of Emergency Medicine
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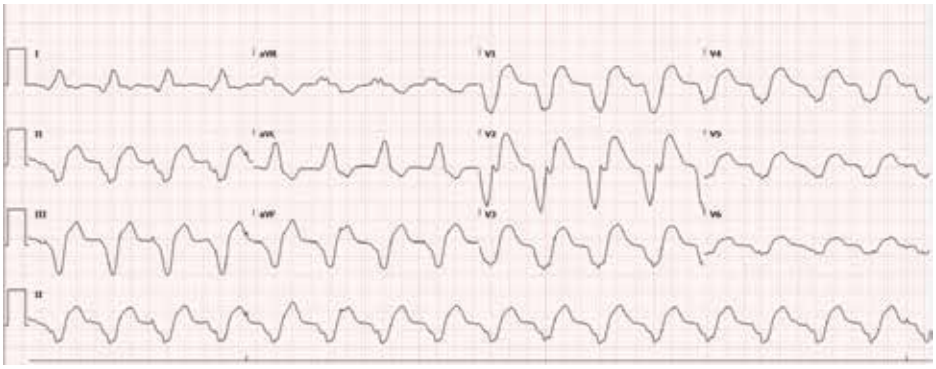
CASE

AN 89-YEAR-OLD MALE WITH A PAST MEDICAL HISTORY OF HYPERTENSION, CHRONIC KIDNEY DISEASE (CKD), AND ATRIAL FIBRILLATION PRESENTS TO THE EMERGENCY DEPARTMENT WITH COMPLAINTS OF GENERALIZED WEAKNESS.



WHAT IS YOUR INTERPRETATION OF HIS ECG?

ANSWER ON PAGE 54

References available online.

ECG Challenge

ANSWER:

This ECG shows a regular wide complex rhythm with a ventricular rate of 99, left axis deviation, no consistent P-waves, an extremely prolonged QRS complex duration with a LBBB-like morphology, and a prolonged QTc interval.

The causes of a QRS complex duration > 200 msec includes hyperkalemia, sodium channel blocker toxicity, and ventricular rhythms (e.g., AIVR, VT). Given this patient's history of CKD, he was treated with IV calcium for presumptive hyperkalemia with no change in the ECG. Further history was notable for the patient being on mexiletine, a Class 1B sodium channel blocker, and his dose had recently been increased.

The classic ECG triad seen with sodium channel blocker toxicity incudes a prolonged QRS complex duration, a prolonged QTc interval, and right axis deviation. Sinus tachycardia is also a very common finding in sodium channel blocker toxicity, so this triad should really be a tetrad, but historically medicine prefers triads (with the occasional pentad). Other ECG findings seen with sodium channel blocker toxicity include a dominant R-wave (> 3 mm) or an R/S ratio > 0.7 in lead aVR.

The cardiotoxicity from sodium channel blockers comes from their blockade of the fast sodium channels. Sodium channel blocking drugs demonstrate a rate-dependent effect, which means there is an increase in the sodium channel blockade with increasing heart rates. In other words, the QRS complex duration and QT interval are more prolonged at faster ventricular rates. This effect is seen with therapeutic levels and is not a sign of toxicity. In the setting of sodium channel blocker toxicity, a prolonged QRS complex duration > 100 msec is associated with an increased risk of seizures, and a prolonged QRS complex duration > 160 msec is associated with an increased risk of ventricular dysrhythmias. In general, mortality increases as the QRS complex duration increases.

Although tachycardia is a common finding in sodium channel blocker toxicity, it is not caused by the sodium channel blockade. Many sodium channel blocking drugs, such as TCAs and diphenhydramine, also have anticholinergic properties, and the muscarinic effects typically result in sinus tachycardia. Other sodium channel blocking drugs, such as cocaine, have sympathomimetic properties that cause tachycardia. These effects compete with, and predominate over, the sodium channel blocking effects of decreased pacemaker cell automaticity. This dynamic changes with severe toxicity when the sodium channel blocking effects predominate, resulting in bradycardia. When bradycardia is present, severe toxicity should be assumed.

Treatment involves rapid assessment of the ABCs and aggressive resuscitation, as many patients present altered with unstable vital signs that warrant establishment of an advanced airway and hemodynamic support. Treatment with sodium bicarbonate is indicated for a QRS complex duration > 100 msec. For cases refractory to treatment with sodium bicarbonate, lipid emulsion can be considered, as many of the drugs that cause sodium channel toxicity are highly lipophilic. Unfortunately, the highly lipophilic nature of these drugs makes them poor candidates for removal through dialysis. If available, ECMO is another consideration for refractory cases.

CASE CONCLUSION

The patient was ultimately treated with sodium bicarbonate for suspected sodium channel blocker toxicity with subsequent narrowing of the QRS complexes on repeat EKGs.

SODIUM CHANNEL BLOCKER TOXICITY LEARNING POINTS

- **EKG triad of prolonged QRS complex duration, prolonged QTc interval, and right axis deviation**
- **EKG features include:**
  - Tachycardia
  - QRS complex duration > 100 msec
  - Right axis deviation
  - Prolonged QTc interval
  - Dominant R-wave in lead aVR (> 3 mm)
  - R/S ratio > 0.7 in lead aVR
- **Tachycardia is common**
  - Due to competing muscarinic, serotonergic, or sympathomimetic effects
  - Bradycardia is typically a sign of severe toxicity
- **QRS complex duration > 100 msec is associated with toxicity**
  - Mortality increases as QRS duration increases
- **Treat with sodium bicarbonate**
- **Sodium channel blocking drugs include:**
  - Anesthetics: bupivacaine
  - Anticonvulsants: carbamazepine
  - Antidepressants: bupropion, mirtazapine, venlafaxine
  - Antiarrhythmics: Class IA (procainamide), IC (flecainide), and II (propranolol)
  - Antihistamines: diphenhydramine
  - Antimalarial drugs: chloroquine, hydroxychloroquine, quinine
  - Cocaine
  - Phenothiazines: prochlorperazine, chlorpromazine
  - Tricyclic antidepressants: amitriptyline, nortriptyline

References available online.

Board Review Questions

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1. A 52-year-old man with a history of exploratory laparotomy 5 years ago presents with severe abdominal pain, nausea, and vomiting. His last bowel movement was 3 days ago. Placement of a nasogastric tube yields copious bilious material. Given the presumed diagnosis, which abdominal X-ray finding is most likely?

A. Bowel wall thickening
B. Dilated bowel with prominent haustra
C. Dilated bowel with prominent plicae circulares
D. Distended U-shaped loop of bowel

2. Which statement describes the clinical manifestations of hypoglycemia?

A. Bradycardia occurs frequently
B. Dry skin is present
C. Focal neurologic deficits can occur
D. Pulse pressure is decreased

3. An intoxicated 52-year-old man presents with altered mental status after using phencyclidine. His laboratory evaluation is significant for a CK level of 5,100 U/L and tea-colored urine. What is the most appropriate treatment?

A. Alkalinization of urine
B. Diuresis with mannitol
C. Intravenous antibiotics
D. Intravenous hydration

4. Which drug is used as a first-line agent for smoking cessation?

A. Clonidine
B. Nortriptyline
C. Sertraline
D. Varenicline

5. What is a contraindication to ultrasound-guided thoracentesis?

A. Creatinine level 5.2 mg/dL
B. Loculated effusions
C. Mechanical ventilation
D. Skin infection overlying the site

PEERprep For Physicians

ANSWERS
1. C; 2. C; 3. D; 4. D; 5. D

References available online.


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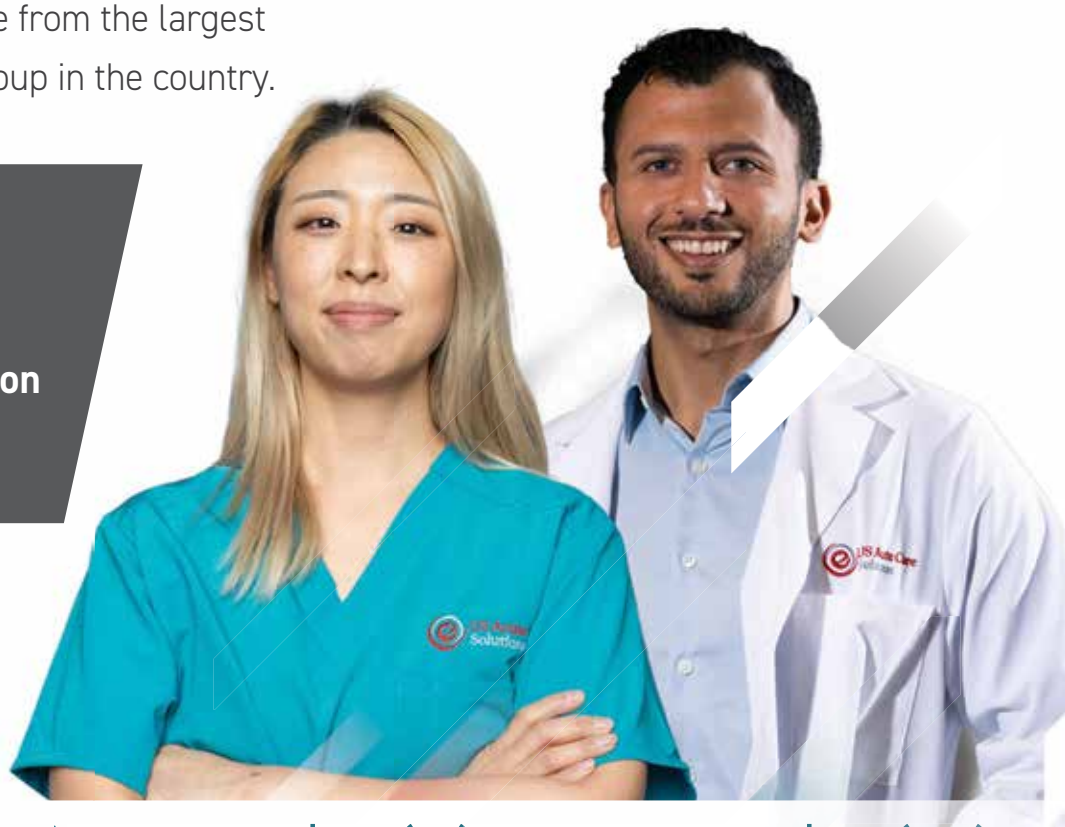

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