EM Resident

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Managing Envenomations

How to Sustain a Career: Peer Support

Guide to ABEM Certification

Small Sicklers

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Welcome to a New Academic Year!

July marks a turning point each year, as new interns arrive in programs throughout the country, newly graduated residents launch the next phase of their careers, and medical students take the next steps in their journey to residency.

DO YOU KNOW HOW EMRA CAN HELP?

Resources for Interns

All EM programs will receive Intern Kits this summer with resources that offer immediate backup for those first nerve-wracking shifts. The high-yield EMRA Intern Kit is sent for free to *all* EM interns (membership not required) and includes:

- *Basics of Emergency Medicine*, 4th ed. (newly updated this year!)
- *Basics of EM: Pediatrics*, 3rd ed. (newly updated this year!)
- EMRA *Trauma Guide*

Plus, log in at emra.org to find resources specific for interns: https://www.emra.org/resident/pgy1

Resources for PGY2+

EMRA residents members receive the nearly 10-pound EMRA Resident Kit upon first joining EMRA. It is packed with clinical resources for every rotation — some you'll need only rarely (but prove to be clutch), and some you'll use every single shift (EMRA Antibiotic Guide, anyone?). Plus, check out:

- EMRA Match for Fellowship: https://webapps.emra.org/utils/spa/match#/search/map (click F for fellowships in the upper left corner)
- EMRA Fellowship Guide: https://www.emra.org/books/fellowship-guide-book/i-title
- PGY2 resources: https://www.emra.org/resident/pgy2
- PGY3+ resources: https://www.emra.org/resident/pgy3

Resources for Students

EMRA shows up for medical students interested in this specialty. From new advising content every month to opportunities for leadership and growth, EMRA student membership is high-yield. Plus, our online resources are unparalleled:

- EMRA Match for Clerkships: https://webapps.emra.org/utils/spa/match#/search/map (click C for clerkships in the upper left corner)
- EMRA Match for Residency: https://webapps.emra.org/utils/spa/match#/search/map
- EMRA and CORD Student Advising Guide: https://www.emra.org/books/msadvisingguide/msag
- EMRA resources for every year of your medical school journey: https://www.emra.org/student/msi (log in as a member to view)

Resources for Fellows and Alumni

You remain one of our #EMRAFamily, long after you've surpassed your residency years. Fellows and alumni receive their own tailored EMRA Member kits upon joining after residency.

Plus, check out:

- EMRA Match for Jobs: https://webapps.emra.org/utils/spa/match#/search/map (click J for Jobs in the upper left corner)
- EMRA Career Resources: https://www.emra.org/residents-fellows/career-planning

From EMRA Match for Jobs to podcast content to contract negotiation tips and CV prep, we've got you covered.

- Log in to get your Fellow-specific content here: https://www.emra.org/emra-fellow
- Alumni, log in and visit: https://www.emra.org/alumni

Resources for All

Check www.emra.org often, as we constantly add new content (podcasts, vlogs, Hangouts, free publications, open-access *EM Resident* online, and more).

Plus, refer often to these critical resources:

- EMRA Wellness Guide: https://www.emra.org/books/emra-wellness-guide/cover
- Emergency Medicine Advocacy Handbook: https://www.emra.org/books/advocacy-handbook/advhbook

Welcome to this new stage of your career! We're glad you're here, and it's our privilege to journey with you. ✶

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EM Resident (ISSN 2377-438X) is the bi-monthly magazine of the Emergency Medicine Residents' Association (EMRA). The opinions herein are those of the authors and not of EMRA or any institutions, organizations, or federal agencies. EMRA encourages readers to inform themselves fully about all issues presented. *EM Resident* reserves the right to edit all material and does not guarantee publication.

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Looking Beyond the Uncertainty

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The first thing I notice about a patient are their eyes. Are they open or shut? Are they bloodshot? Are they avoiding eye contact? When we look in someone's eyes, emergency physicians get an immediate sense of what we will encounter as our time together unfolds. Not only do we begin to gather pertinent information for our physical exam, but we also see the emotions underlying their chief complaints. As I reflect on my time as EMRA president and on my first year as a brand-new attending, I also wonder what people saw when they looked in my eyes.

Did my very first patient see any uncertainty? His chief complaint was dizziness, and his symptoms were vague. I looked in his eyes, searching for nystagmus or any other clues I could use. As I examined him, differentials swirled in my head, and I tried to decide if I should call a stroke alert. That uncertainty left me with a sinking feeling in my stomach, and imposter syndrome began to set in.

We all have that feeling at these times of transition. These transitions tend to happen in summer, as one academic year moves into the next. As we adapt to starting medical school or clerkships or residency, we know our training and preparation are designed to propel us to success, but we also appreciate the magnitude of what we cannot know. I leaned in to my uncertainty and decided to ask for help. I called a stroke alert and spoke to a neurologist. I looked her in the eyes as I described the patient's presentation, and she agreed the presentation made diagnosis difficult. I breathed a sigh of relief, realizing I was not alone in my uncertainty. I was beginning to build what would become strong relationships with our consultants.

Later in the year, as the winter set in and the pandemic worsened, a nurse pulled me into a room. He looked concerned and explained that his patient arrived in respiratory distress, and she was not responding to the BiPAP EMS had provided. I called for respiratory therapy to join us, and I looked in my patient's eyes. I saw fatigue, and I knew she needed immediate help. Between shallow breaths and coughing fits, she shared that her partner recently died from COVID-19, and she wondered if she would survive. I maintained eye contact and did my best to comfort her as we discussed intubation. The pandemic has shown all of us how little certainty we can have in these situations, and balancing the desire to be both honest and reassuring is never easy. As the sedative was pushed, her eyes became glassy, and then they slowly shut. I wondered if they would ever open again. After intubating her, I looked up at the staff around me; the patient was not the only one with fatigue in her eyes.

Navigating uncertainty through challenging times has been a hallmark of the past year, and it became even more apparent in the spring with the release of the report about the changing EM workforce (acep.org/workforce). I see the impact of this when I look in the eyes of EMRA members who have already felt the pressures of these changing workforce trends. We have the opportunity to look this challenge in the eyes and make a decision: **We can sit back and wait to see how it unfolds, or we can lean in to our uncertainty and look for solutions.** Now is the time to have difficult conversations, including conversations about our own residency training models and about the impact of business interests and other providers on our training and practice. EMRA's recent Workforce Town Hall showed that our members are ready to have these difficult conversations.

Whether I am with patients as their doctor, with consultants as their partner, or with you as EMRA's president, I see it as my job to look a person in the eyes and share what I know and what I am uncertain about. I am uncertain about the future, but I know it looks different. I know that we have more power when we band together to make change. I know we have the power to design our future, a future where our generation leads, with new solutions for safe and effective patient care.

As we embrace the uncertainty that summer and its associated transitions inevitably bring, keep in touch. I hope to be able to look you in the eye in person at a conference very soon. In the meantime, email me at president@emra.org to share your ideas for the future. Let's find ways to support each other and work together for a better future. ✶

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U.S.ARMY

Micro-Communities of Practice to Maintain Resilience During Times of Uncertainty

A Sustainable Means to Draw Strength When Depleted

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The impact COVID-19 has had on our personal and professional livelihood cannot be taken lightly. Since the beginning of the pandemic, we have focused our efforts on protecting our patients, our loved ones, and ourselves. We relinquished the connectedness that had once rejuvenated our ethos for emergency medicine. Social creatures by nature, emergency physicians (EP) crave opportunities to share narratives and create meaning from work collectively. Some of us have successfully navigated separation from family and friends by identifying creative ways to maintain connectedness while adhering to social distancing requirements. How then can we practically and sustainably maintain our sanity during times of uncertainty?

In this commentary, we describe lessons learned for maintaining personal vitality and professional connectedness. By creating our very own *micro-community of practice*, we have been able to create a shared space to provide peer-to-peer mentorship and process the ripples of the pandemic to create meaning from chaos — meaning that has been essential to guide us as leaders.

We are three emergency physicians who hold a variety of academic leadership positions at three institutions within the same geographic region. While our kinship began long before the pandemic, the last year strengthened our resolve to maintain regular interactions to support one another — personally and professionally. It did not matter that we represented three very unique health systems; we had one same goal in mind: *Keeping ourselves and our respective teams moving forward through the isolation and frustration of COVID-19.* We have learned that to maintain sanity and nurture resilience during a pandemic requires intentional action. And over the last year, we have identified critical tips that have helped us sustainably navigate uncertainty.

As you travel the many roads and detours over your evolving careers, remember that the relationships you build along the way will be critical to your success. You may need to rely on these relationships during darker days to come. Our overarching message is simple: Expand your relationship circles, both professional and personal, and nurture them with intention. It's worth it. There will come a day when you will need support through difficult work situations, career-altering decisions, or perhaps another pandemic. Start building your micro-communities of practice.

Tips to Maintain Thriving Professional Relationships During Times of Uncertainty

1. **Share resources with one another.** One benefit of networking is utilizing the expertise and resources of that colleague to aid in your daily responsibilities and career development. Through the years we continue to share our resources to help one another. Policies, spreadsheet templates, curricula, you name it, we've sent to each other as samples; and it really helps to reduce work. Why recreate the wheel for some things when a colleague has already done it?
2. **Connect with like-minded individuals, regardless of where they're from.** Join a local or regional group with similar interests or roles as yours. Share your stories and listen to others' — in them, you will find common ground.
3. **Raise your glass.** What's better than sharing a meal with friends? (Sharing a BIG meal with friends!) We made a commitment to make time for this at every regional or national conference, and in between. Strive for connectedness as often as possible. Unfortunately, COVID put the kibosh on getting together at restaurants, but we did this electronically during the pandemic. As restrictions lifted, and we were all vaccinated, we hopped back on the food-train and shared some great meals, provided lots of laughter, and generating great ideas we could take with us to develop for ourselves or with each other. This winter was intimidating; but we eventually decided to have an outdoor dinner thanks to a heated igloo (see pic). There is nothing more memorable than an Arctic dinner with great company!
4. **Embrace laughter.** There are few things as cathartic and nurturing for the soul as a good belly-laugh. Find ways to see things for what they are, even the humor in them. Create spaces for others to rant and complain, and try to find humor in the ridiculous. Find others who allow the same for you. Laughing got us through some difficult times, kept our sanity and further bonded our relationship. It was a safe space; what was discussed between us stays between us.
5. **Watch the forecast.** Tough times lie ahead of any career. Identify triggers for burnout and create sustainable plans to mitigate them. Plan ahead during stable times when waters are calm for those times you'll need a rudder for stability.
6. **Commit to communicate.** Keep in touch and check-in with your group – no matter how silly the communication may seem. The random, occasional funny text or meme within our group text sets off a flurry of return texts, likes, dislikes, laughs, and further conversation. It may also arrive when it's needed most. Don't be afraid to inject supportive messages during the day or week. You never know what the person might be dealing with on the other end. You very well may be helping them through.
7. **Offer support through transitions.** When making life- or career-altering decisions, call on those you trust and who have had to make the same or similar decisions. These are people who understand what you are going through, understand the context and potential roadblocks or pitfalls. And who better to air your dirty laundry and get advice!
8. **Venture off your island.** When things are tough, it's easy to remain isolated. Sometimes it's just easier to turn inward. Periodically check-in on others who might be doing the same. Find ways to leave what's comfortable for you, and find comfort in new or alternate ways of connecting or enjoying life. Purposely choose to meet up somewhere distant from your home or workplace. Physical separation from these places can be refreshing and invigorating. You will leave with a new sense of energy and purpose.
9. **Inspire one another.** Brainstorm or create opportunities that were not obvious to you as an individual. Sometimes you need peers to see areas of growth required in yourself that you do not see. Offer ideas for growth or change and accept that from others. Life can be tough at times, but through it all, three peer mentors found a way to empower one another.
10. **Do Steps #1 through #9 over again.** Do not underestimate the impact a consistent routine can have on your well-being. A consistent routine built into how you connect with your community of peer mentors can satisfy the innate craving for belonging and boost brain dopamine levels.

What's our message? Leave your self-imposed islands and develop your micro-communities to sustainably maintain your sanity and resilience during times of crisis and uncertainty. Basically, **screw COVID**! ✶

Residents' Guide to ABEM Certification

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Editor's note: ABEM Resident Ambassador Panel members serve 2-year terms during residency training and provide a resident perspective to ABEM activities. Working with ABEM over the past year, the 2020-2022 ABEM Resident Ambassador Panel has gained insights into the process of becoming ABEM-certified, and they have outlined those steps from their perspective in an effort to streamline your preparation for the certification process.

Becoming board certified in Emergency Medicine by the American Board of Emergency Medicine is a simple process requiring three steps for residents who are in their final years of training.

1 STEP 1. Applying for Certification

During the last year of a resident's emergency medicine training, graduating residents destined to finish residency by Oct. 31 can access application information by signing into the ABEM initial certification page. ABEM will also send application information to the program director of the residency program, usually in April. Anyone graduating later than Oct. 31 will apply in the next application cycle. For EM residents who graduate between Nov. 1 and the following Oct. 31, it's important to apply in the current application period. **If you delay, you may need additional certification requirements,** including a state medical license, if you do not have one already.

The entire application and fee payment process is online. Applications are processed as soon as they are completed.

Board eligible means that a resident graduated from an ACGME or RCPSC accredited emergency medicine program or an ABEM-approved combined program. Additionally, you must fulfill all medical licensure per ABEM policy. If you are applying directly out of residency, you do not need to hold a state medical license. This starts on the day you graduate from residency and extends to Dec. 31, 5 years after your graduation date.

2 STEP 2. Passing the Qualifying Exam

The second step in becoming ABEM board certified is to pass the qualifying examination, a computerized test with 305 multiple choice questions (with only single best answer choices). The qualifying exam is offered in about 200 Pearson testing centers across the U.S., making it easy to take the exam in the state you graduated from or plan on practicing in. The exam itself is offered during one 6-day period, typically in the fall. In order to take the exam during this time, you must schedule one 8-hour block in this 5-day period.

Should you be unable to attend the exam, you can cancel up to 24 hours before the start of the exam. Please arrive 30 minutes before your exam time and make sure to bring a valid form of identification. This process is similar to many of the other examinations you have taken to get to this point in your career as a physician!

The exam appointment is a total of 8 hours long, divided into 3 testing sections, each about 3 hours and 10 minutes long with a 1-hour break in between. The question topics are based on the EM Model, similar in makeup to the in-training exams, which you have likely already experienced during your residency training.

Once you have completed this qualifying examination, you can expect your score within 90 days of completion.

3 STEP 3. Passing the Oral Board Exam

The third and final step in completing board certification is to pass the oral board examination. To be eligible, you must have passed the qualifying examination and have a state medical license. Once you pass your qualifying examination, you must take the oral board exam the next calendar year.

It's important to note that given the COVID-19 pandemic, there have been some changes to the implementation of this section of board certification. Notably, the examination has been offered on a virtual platform, as opposed to in-person, for the safety of test takers and testing staff.

The oral board examination comprises 6 single patient cases, each 15 minutes long. The examiner will provide pertinent history and offer answers to the examinee's questions. The examiner will track 8 specific markers during these patient cases. These markers include:

- Data acquisition
- Problem solving
- Patient management
- Resource utilization
- Healthcare provided or outcome
- Interpersonal relations and communication skills
- Comprehension of pathophysiology
- Clinical competence

Examiners assign a score from 1-8, with 1 being very unacceptable to 8 being very acceptable.

In addition to the 6 single patient cases, a discussion on your approach to patient care will evaluate your thought processes. Structured interviews are scored as 25 points spread across 8 stages of a typical patient interaction. These include:

- ✓ History
- ✓ Physical exam
- ✓ Differential diagnosis
- ✓ Testing
- ✓ Treatment
- ✓ Final diagnosis
- ✓ Disposition
- ✓ Transitions of care

ABEM typically releases oral board exam results within 45-60 days, and definitely within 90 days. ABEM does not use quotas or percentages to determine a passing score. Instead, after each examination, ABEM testers meet to determine the standard of care for each case and then determine whether testers passed or failed. The final passing score is then sent to the ABEM Board to determine performance expectations for a pass or fail score. ABEM does not allow for rescoring or second scoring any examinations.

Success

Once you have passed the Oral Board Examination, congratulations! You are now an ABEM board-certified emergency physician!

ABEM-certified physicians serve a valuable and irreplaceable clinical role in the care of the critically ill and injured. The delivery of emergency care is best led by physicians with EM training, experience, and ABEM certification. ABEM will support you throughout your career in continuing certification activities and promoting the important and valuable role ABEM-certified physicians bring to emergency care in the ED.

Do you have questions about the certification process? Reach out to your program director or contact ABEM at abem@abem.org. ✶

Be Prepared for Summertime Exposure
Managing Envenomation

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RATTLESNAKE

A 50-year-old female is celebrating Labor Day weekend with you and your friends in New Mexico. She is swimming at a lake, and upon walking out of the water feels a sharp pain around her ankle. You notice a snake under a rock near her foot and take a photo with your phone as shown. You see 2 small dots around her ankle, and you're 3 miles from your car and another 1-hour drive from the nearest hospital. She is neurovascularly intact with a normal exam except for subjective pain.

This woman has sustained a bite from a rattlesnake and possible envenomation. Rattlesnakes are classified as *crotalinae*, also known as pit vipers.[1] The name pit comes from a heat sensing pit behind the nostril used to find prey. When venom is released, local damage is done by metalloproteinases, phospholipase A2, serine proteases, and hyaluronidase which lead to local tissue edema and swelling from capillary damage.[2-4] Approximately 25% of bites are "dry bites," with no venom released. *Crotalus scutulatus*, also known as Mohave rattlesnakes, are indigenous to the deserts of the western United States and Mexico. They contain a neurotoxic-hemolytic venom, which may lead to vision abnormalities, difficulty with swallowing, and even respiratory failure.[5]

In the wilderness setting, local care and evacuation must be initiated. Jewelry should be removed from the entirety of the affected limb. If erythema is present, it may be helpful to mark the leading edge and notice of the time for tracking of progression. If possible, immobilize the limb in a neutral position. Though some may have heard to do the following, you should refrain from catching the snake, drinking alcohol, cutting a wound to suck out the venom, applying tourniquets, or attempting to use an electric shock.[6] Assessment at a medical facility with labs and monitoring is warranted, and typically will consist of labs to assess coagulopathy as well as consideration of antivenom.[3,4]

SCORPION

A 4-year-old girl is brought to an ED in Phoenix, Arizona, with agitation and foot pain. She is tachypneic, restless, and drooling, and on exam you note tongue fasciculations, multidirectional nystagmus, and a small area of erythema on her right foot. What animal is likely responsible for her symptoms?

This child has likely suffered a bark scorpion envenomation. *Centruroides sculpturatus* is endemic to the American southwest, especially Arizona, Texas, Nevada,

California, Oklahoma, and New Mexico.[7] Scorpion venom is a complex mix of many substances, but the most clinically important is a heat-stable and protease-resistant neurotoxin.[8] This neurotoxin targets axonal sodium channels, increasing the firing of axons by widening and prolonging the action potential and leading to increased release of catecholamines.[8] There is a wide spectrum of symptoms including local pain/paresthesia as well as:

- Neuromuscular abnormalities — agitation, skeletal muscle fasciculation, motor hyperactivity (flailing movements, tetanus-like spasms), rhabdomyolysis
- Autonomic dysfunction — tachycardia, hypertension, vomiting, hypersalivation
- Cranial nerve abnormalities — opsoclonus (roving, multidirectional eye movements), blurred vision, tongue fasciculations, dysarthria, stridor, pharyngeal spasm, dysphagia, hyposmia, and dysgeusia.[8-11]

While adults are stung more often, children are more likely to have more severe symptoms.[8,12] The mainstay of treatment is supportive care, analgesia, and possible benzodiazepines if needed to reduce neuromuscular hyperactivity.[8] In severe cases, an FDA-approved antivenom is available for use after consulting with a regional poison control center.[13]

GILA MONSTER

A distraught mother and friend of yours video calls to show you her son with an animal latched onto his arm from the backyard. They recently bought a home with a pool in the Southwest United States. You immediately recognize the color pattern is consistent with a Gila Monster. What do you do next?

This is a Gila monster (*Heloderma suspectum*), is a sluggish lizard commonly found in Southwest United States and Northwest Mexico. Typically biting when agitated, they will latch on and chew onto tissue activating the release venom and can be difficult to remove. This may lead to pain, edema, hypotension, nausea, vomiting, weakness, and diaphoresis.[14]

When bit, it may be difficult to remove. Submersion in water or prying open the mouth may be necessary.[15] There have been historical reports of using gasoline in the mouth, flame on the chin or underbelly, and pulling on the tail but these should not be done.[16,17] Irrigation and medical attention is recommended immediately. There is no antivenom available and treatment is supportive. The affected area should be elevated to heart level. Suction or compression for treatment is unproved, and cryotherapy, tourniquet, and excision can be dangerous.[15]

BLACK WIDOW SPIDER

A 27-year-old woman presents to the ED with muscle pain. She was gathering firewood near her campsite in eastern Texas when she suddenly felt a mild pain on her left arm. About one hour later she developed left arm muscle pain and paresthesia, diffuse abdominal pain, and nausea. On exam she has a tender and rigid abdomen, and you note a small area of erythema with a blanched center and small central punctum on her left volar forearm. What is responsible for her symptoms?

This patient was likely bitten by a black widow spider. Spiders in the *Latrodectus* species are found in warm climates worldwide, with the most common in the United States being the southern black widow (*L. mactans*) and the western black widow (*L. hesperus*). These spiders collectively range throughout the southern and western United States and are easily recognizable by their black bodies with red, hourglass-shaped abdominal markings.[18] Black widow venom contains excitatory neurotoxins which stimulate massive exocytosis of multiple neurotransmitters from presynaptic nerve terminals.[19] Symptoms can include muscle pain and intermittent rigidity, abdominal pain, paresthesias, and autonomic dysfunction. The typical appearance of the bite is a blanched circular patch with surrounding red perimeter and central punctum. Local diaphoresis and lymphadenopathy may also be present. Treatment is generally supportive, consisting of oral analgesia, benzodiazepines, antiemetics, local wound care, and tetanus prophylaxis if indicated. Antivenoms are available and may be used in moderate to severe envenomations after consulting with a regional poison control center.[20]

ASIAN GIANT HORNET

A 38-year-old man presents to the ED after 3 days of progressively decreased urination. He was in Northern India 3 days ago when he was attacked by a group of insects, and since returning to the US yesterday has also developed ankle swelling, fatigue, nausea, and vomiting. He has approximately 8-10 small, tender areas of erythema and edema on his trunk and upper extremities. What animal is responsible for his symptoms?

This patient was likely stung multiple times by the Asian giant hornet, colloquially called the "murder hornet." *Vespa mandarinia* is native to China, India, Japan, Russia, Vietnam, Thailand, Malaysia, Nepal, and other South and East Asian countries, but there have been a number of reports of these wasps in British Columbia and Washington State in 2019 and 2020.[21-23] Wasp venom contains several active components and can result in conditions ranging from local type I hypersensitivity reactions to anaphylaxis.[24,25] Systemic reactions such as shock, rhabdomyolysis, coagulopathies, respiratory distress, hepatitis, and acute kidney injury are possible especially with multiple stings.[24,26,27] Treatment is symptomatic and supportive (often including glucocorticoids, antihistamines, and IV fluids), as there is no anti-venom available.[24,26,27]

Diagnosing snake envenomation is a crucial step in determining which antivenom is to be applied. Each year there are around 2 million cases of snake envenomation and up to 100,000 deaths worldwide. Various anti-venom treatments exist, typically consisting of antibodies or antibody fragments, which neutralize the venom. Anti-venom therapy is designed to treat the hemorrhaging and coagulation effects that venom has on humans.

CORAL SNAKE

"Red on yellow? Red on black? Jack?" Your friend sustained a bite from a snake yesterday while visiting a friend in Florida. He couldn't remember the riddle but started feeling paraesthesias, nausea, and vomiting after 12 hours. He sends you this picture and asks if he should anticipate staying in the hospital.

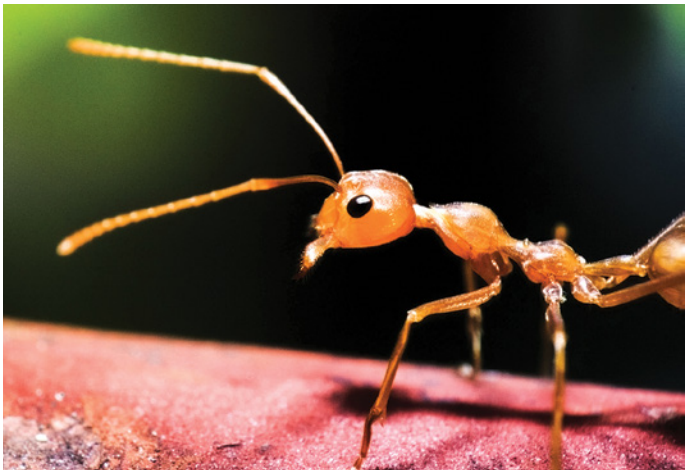
The Eastern coral snake (*Micrurus fulvius fulvius*) as described here with red adjacent to yellow, commonly confused with the Scarlet King Snake or the Milk Snake, which has red adjacent to black. The "Red on Yellow, Kill a Fellow, Red on Black, Friend to Jack" rhyme is generally true but there are morphology variants of coral snakes that do not follow this pattern. They possess small, fixed front fangs and may chew to envenomate. Coral snakes have a venom that binds to acetylcholine receptors and is neurotoxic. Around 20-25% of the time, the bite may be a dry bite.[28,29]

Typically after envenomation, there are minimal local symptoms, and symptoms may develop up to 12 after the exposure.[30] Symptoms can include swelling, paresthesias, nausea, vomiting, euphoria, weakness, dizziness, diaphoresis, muscle tenderness, fasciculations, confusion. In worst case scenarios, bulbar paralytic symptoms, extremity paralysis, and possible respiratory failure.[28]

Treatment involves immobilizing the extremity, marking the erythema, and removing any jewelry. Do not to suck the venom, submerge in warm water, use tourniquets, take NSAIDs, or splint the affected extremity. Some suggest that all need to be admitted to the hospital due to delayed symptoms, and some may need antivenom.[31] Some may advocate for a 8-12 hour monitoring period in the ED with assessments for compartment syndrome and coagulopathies, and if discharged, with strict return precautions.[32]

FIRE ANT

A 72-year-old man with a history of dementia presents to an ED in Mississippi with painful, pruritic lesions on his feet. He was found 20 minutes prior to arrival sitting on a bench outside his assisted living facility. On both of his feet there are numerous wheals with surrounding erythema, which developed into pustules the next day. What animal is responsible for these findings?



This patient was bitten by fire ants. The red imported fire ant (*Solenopsis invicta*), black imported fire ant (*Solenopsis richteri*), and a hybrid species are found in many states in the southern and southeast US, and as far west as California.[33] They are generally aggressive, swarming and stinging even without provocation. Ant bites are more common in children, but less mobile patients are at higher risk; there have been several recent reports of attacks on nursing home residents.[34]

The venom is 95% water-insoluble alkaloid which is cytotoxic and hemolytic, and 5% allergenic aqueous protein solution. The immediate response (IgE-mediated) is a dermal flare and wheal, with papules forming within 2 hours, vesicles within 4 hours, and pustules within 24 hours. There can be large local reactions of pain, erythema, and edema, and they can also cause systemic anaphylaxis. Interestingly, anaphylaxis more commonly occurs in those with prior ant or yellow jacket stings due to venom cross-reactivity.

Fire ant stings can rarely cause serum sickness, nephrotic syndrome, seizures, and exacerbation of pre-existing cardiopulmonary disease. Treatment is firstly to remove any remaining ants, as they can sting repeatedly. Topical antihistamines and corticosteroid creams are sufficient for local reactions, and anaphylaxis should be treated appropriately. The sterile pustules should be left alone but should be cleaned with soap, water, and an antibiotic cream if broken.[33]

STINGRAY

You are walking along the beach and you see a couple taking wedding photos with their feet in the water at a Louisiana beach. The bride shouts and looks down and sees a stingray next to her leg. The photographer catches this photo before it swims away.

Stingrays are found in coastal tropical and subtropical marine waters around the world, with 24 different species found in the US.[35,36] When envenomated, the stinger punctures the skin and introduces a heat labile venom. This commonly occurs when stingrays are stepped on in shallow water and shuffling your feet may decrease your likelihood of getting stung. Effects are typically limited to just local effects including pain, irritation, erythema out of proportion to the wound but vary by species. Maximal pain is usually experienced at 30-90 minutes.[37] If systemic effects occur, they may include nausea, vomiting, diarrhea, diaphoresis, weakness, headache, vertigo, abdominal pain, syncope, cramping, and in worst case scenarios, seizures, respiratory distress, hypotension, convulsions, paralysis, cardiac arrest.[38,39]

Management should start with rinsing in tap water, or if unavailable, salt water. Warm and hot soaks, ideally as hot as tolerated should be applied. Oral narcotics or local anesthesia may be used. The affected individual be evaluated by a medical professional to ensure their tetanus is up to date, evaluated for retained foreign bodies, and supportive care.[37] Prophylactic antibiotics are controversial. Since the venom is heat labile, the wound should be immersed in hot water as soon after the injury as possible, be careful as to not cause further injury by scalding: 45° for 30-90 minutes is recommended. Removal of the barb may be attempted and tetanus should be updated.[40,41] Urine, as well as other historically suggested treatments such as application of numerous substances: macerated cockroaches, fish liver, tobacco juice, cactus juice, gasoline, wine, and cryotherapy are not recommended.[40,41]

Envenomation is the process by which venom is injected by the bite or sting of a venomous animal. Many kinds of animals, including mammals, reptiles, spiders, insects, and fish employ venom for hunting and for self-defense.

BOX JELLYFISH

You and your friends walk across the street to the beach in Florida after eating at a seafood restaurant. Your friend walks out of the water with a jellyfish wrapped around his leg. You immediately run over to provide aid, and you're being asked what can be done to aid the victim, and if urine can be used to "neutralize" the venom.

Classically, jellyfish stings cause local pain up to 8 hours with linear welts which can progress to blistering and necrotic areas. The specific venom components and mechanisms are unclear, but it's thought to affect sodium, potassium, and calcium channels at cell membranes. Classically, it causes local pain up to 8 hours with linear welts that can progress to blistering and necrotic areas. Sometimes, if involving > 10% body surface area, systemic effects may take place, including cardiac arrhythmia, hypotension, shock, tachycardia, respiratory dysfunction, death. These usually manifest within 5 minutes of the sting.[42]

There are many different species of box jellyfish depending on location so following local guidelines are recommended. The American Heart Association-American Red Cross International Consensus on First Aid Science currently advocates vinegar or baking soda slurry followed by the application of heat (or an ice pack if heat is not available) for all jellyfish stings in North America and Hawaii.[43] Be wary of using vinegar because some studies suggests it may worsen symptoms.[44] Try to control the pain with oral agents if available, and hot or cold packs or water may help.[43,45] Topical lidocaine and how water has shown to help in multiple studies.[46] Pressure bandages should not be used.[47] Other potential options with unclear benefit for treating symptoms include commercial products with aluminum sulfate,[48] papain,[49] lidocaine hydrochloride,[44] baking soda,[50] deionized water, seawater, meat tenderizer.[48] In some hospital settings, antivenom has been administered, which is a bovine IgG Fab.[51]

FIRE CORAL

A 16-year-old boy just got a job at a fish aquarium and comes in with intense forearm pain after cleaning a fish tank with coral. Other than local pain, he has no other symptoms and has normal vital signs.

Fire coral (*Millepora alcicornis*) has a white to yellow or green exoskeleton and exists around the world. It contains dactylozooids, which are small tentacles, in the exoskeleton. Envenomation occurs via nematocysts, similar to jellyfish. The venom has hemolytic, dermonecrotic, and cytotoxic effects. Since they are rigid, many injuries occur after abrading the structure.[52,53] The envenomation is immediate and painful, and also is associated with pruritus, urticaria, blistering.[54] In severe cases, pulmonary edema, hypotension, fever, and renal failure may occur.[55,56]

Pain and sensitivity may last months but systemic symptoms are rare. Treatment is mostly extrapolated from nematocysts of other Cnidarians such as jellyfish. Like jellyfish, both hot and cold water are beneficial, as are oral agents, topical lidocaine, and steroids.[52] Lacerations are common, and should be irrigated and closed loosely. Antibiotics are recommended to cover skin and aquatic flora and tetanus should be updated. If no systemic symptoms present, patients may be discharged. ✶

From catfish to Irukandji jellyfish, marine envenomations present a wide and varied picture, in terms of pain, systemic severity, and treatment. While the most lethal and complex envenomations occur in more tropical waters, it pays to be ready to handle any marine envenomation — more than 50% of all venomous vertebrates are fish.

References available online

PERICARDIAL EFFUSION

A Rare Case of a Potentially Dangerous Manifestation of Lyme Disease

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Lyme disease is one of the most common vector-borne diseases in the United States, affecting over 300,000 people annually between June and December and most prominent in the northeastern, mid-Atlantic, and north-central regions.[1] The culprit, Borrelia Burgdorferi, is a spirochete transmitted through deer tick (Ixodes Scapularis) in the Eastern U.S. and the Western blacklegged tick (Ixodes Pacificus) in the West, that gets transmitted to the human host when the tick is attached to the human host for over 36 hours.[1]

Lyme disease affects every organ system and is described in three progressive stages of disease: early localized, early disseminated, and late disseminated or chronic. Stereotypical manifestations include the cutaneous "bull's eye rash" *(Figure 1),* unilateral Bell's Palsy, and Lyme carditis which often causes an AV node conduction abnormality.[1,2,3,8] This case report describes a less common cardiac manifestation of Lyme disease: pericarditis with pericardial effusion.

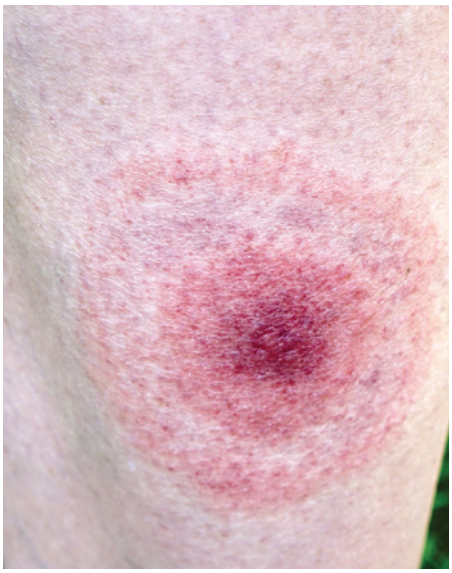

FIGURE 1. Bull's eye rash indicative of Lyme disease

Case

A 61-year-old female was sent to the ED after an incidental finding of pericardial effusion on outpatient echocardiogram. Her medical history was significant for well controlled type 2 diabetes, hypertension, and hyperlipidemia. Upon presentation to the ED, the patient endorsed two weeks of intermittent palpitations, worsening dyspnea that was exacerbated by laying down, and weight gain. She denied chest pain, cough, recent fevers or illnesses, any surgeries or long periods of immobilization. Recent travel history was only significant for spending summers at her cabin in Pennsylvania. She denied any family history of cardiovascular disease as well as tobacco, alcohol, or recreational drug use. On the previous week the patient visited her primary care physician (PCP) with these concerns. At that time, atrial flutter was identified on electrocardiogram (ECG). She was started on a beta blocker, an anticoagulant, and referred for the echocardiogram.

Upon further questioning, patient reported she was in her usual state of health until last summer (4 months prior to presentation) when she had developed a single erythematous lesion on the left upper extremity, which eventually spread over her entire body. A skin biopsy was significant for urticaria and she completed a treatment of steroids. She subsequently developed fatigue, then mild positional dyspnea, and 2 months later she developed a unilateral right 6th and 7th nerve palsy. Extensive workup was non-contributory until a spinal tap demonstrated elevated cerebrospinal fluid protein and lymphocytes, when led her PCP to suspect Lyme disease.

In the ED, her heart rate was 121 bpm, BP 155/65 mmHg, RR 20 breaths/min, and oxygen saturation 100% on room air. Her physical exam was significant for a comfortable appearing woman with an elevated BMI, tachycardia, and moderate bilateral lower extremity pitting edema. Blood work was significant for a WBC 10.9, Hgb 10.5, CRP 33.5, ESR 45. ECG showed atrial flutter with variable block (figure 2), and a bedside echo showed a moderate pericardial effusion without hemodynamic compromise, which was confirmed by formal echocardiography (figure 3). The patient was admitted to cardiology for pericardiocentesis and further work-up.

Pericardiocentesis was performed by interventional cardiology and yielded 500 cc of blood so her anticoagulant was discontinued. There was no cytologic evidence of malignancy, viral, bacterial or fungal growth in the pericardial fluid, but have findings consistent with an exudative process with LDH 234 (fluid/serum LDH > 0.6) and fluid protein 76.2 (fluid/serum protein >0.5). Meningitis and respiratory panels, tuberculosis, syphilis, SARS Cov2, and West Nile virus tests were negative. Her Lyme ELISA test resulted 11 days post-admission and was unequivocally positive.

Given her history and presentation, disseminated Lyme disease was thought to have been the cause of her pericarditis complicated by pericardial effusion, and she was presumptively treated for Lyme with 4 weeks of intravenous ceftriaxone. The acute pericarditis was managed with intra-pericardial steroids, ibuprofen,

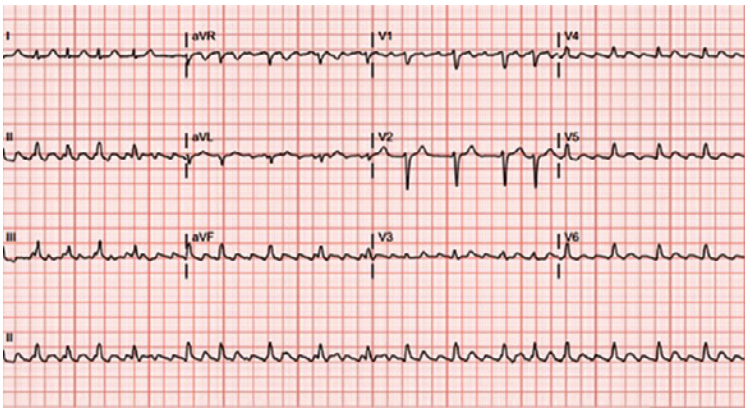

FIGURE 2. ECG at presentation demonstrates atrial fibrillation

and colchicine. Diuresis was initiated, leading to a 7.7 kg weight loss during her hospitalization and another 10 kg in the subsequent 2 weeks. Her hospitalization was complicated by refractory atrial flutter and decreased cardiac function evidenced by a left ventricular ejection fraction (LVEF) of 30% (from 65% on admission) with global dysfunction. She was discharged on digoxin on hospital day 7 and felt better for a month at which time her symptoms started to return. An echocardiogram at that time revealed interval improvement of cardiac function (LVEF 48%) but a moderate pericardial effusion re-accumulation, managed conservatively with high dose prednisone.

Discussion

Lyme disease is often classified into 3 stages:

First — Early Localized Disease

Within days of exposure patients present in the spring or summer with flu-like illness, fever, and a typical rash (erythematous patch with a central scab and blurred margins) *(figure 1)*. The erythema migrans "bull's eye rash" appears within 48 hours and is most apparent 7-14 days after tick is detached.[1]

Second — Early Disseminated Disease

Neurologic manifestations such as Bell's palsy (unilateral or bilateral) or aseptic meningitis may develop. Cardiac manifestations occur in 1-10 % of cases.[1,2,8]

Third — Late Disseminated or Chronic

Encephalopathy, radiculopathy, severe fatigue, psychologic disturbances, mono-arthralgias (often the knee).[1]

Evidently, we met this patient on the second stage. "Lyme carditis" most frequently manifests as conduction abnormalities (eg, AV nodal blocks) due to direct toxic and immune-mediated mechanisms. Less commonly, Lyme carditis has been implicated in coronary aneurysms, congestive heart failure, valve disease, ischemic events, endocarditis, myocarditis, and rarely pericarditis (2-5% of Lyme carditis cases in the U.S. and 23% in Europe[4,5]).

The pericardium consists of thin visceral and parietal layers between which a potential space usually holds 15–35 cc of fluid, but when the pericardium becomes acutely inflamed, a pericardial effusion can ensue and hold up to liters of fluid. Infectious organisms are often the precursor to pericarditis in world, but in north America up to 90% of cases are idiopathic.[7] The most common symptoms of pericarditis with effusion are dyspnea, pleuritic chest pain that is relieved by leaning forward and exacerbated by lying down and swallowing, nausea, abdominal pain, and malaise.[2,7]

Pericardial tamponade is a dangerous complication of the rapid development of a pericardial effusion leading to increased pressure in the pericardial space, decreased ventricular filling and cardiac output leading to hemodynamic collapse. Signs of tamponade can be remembered by the "Beck's triad:" hypotension, muffled heart sounds and jugular venous distention.[7]

The essential workup for pericarditis in the ED includes a physical exam, chest x-ray or bedside ultrasound, ECG, and basic labs. An auscultated high-pitched scratchy "friction rub" is 100% specific for pericarditis.[3] While chest x-ray may detect effusions larger than 250 cc, a bedside echocardiogram can detect as little as 20–50 cc of pericardial fluid[3] and up to 60% of pericardial effusions.[7] ECG with PR depression or ST changes that do not match anatomic regions, low voltage, and electrical alternans suggests a large pericardial effusion and should prompt evaluation for tamponade physiology. Classically, the ECG findings seen with pericarditis evolve through 4 stages:

1. Diffuse concave upward ST-elevations (typically 2-4 mm) and PR segment depressions diffusely in all leads other than aVR and V1 which will show PR segment elevations and

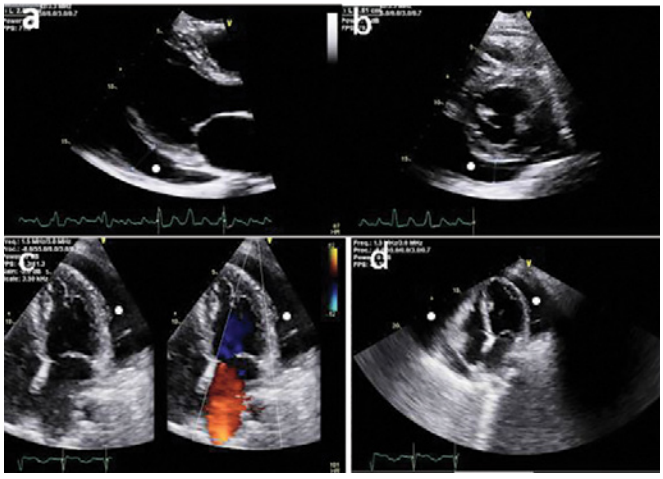

FIGURE 3. Pericardial effusion. Transthoracic echocardiogram demonstrates a large pericardial effusion (+) a: Parasternal long axis view demonstrates typical interposition of the pericardial effusion (+) between the heart and the descending thoracic aorta. b: Parasternal short axis at the level of the papillary muscles. c: In the apical 4-chamber view, note that the pericardial effusion surrounds the cardiac apex. d: Subcostal view.

ST-segment depressions
2. Normalization of ST and PR segments plus T-wave flattening
3. Diffuse T-wave inversions, typically in the leads that previously had ST-segment elevation
4. ECG normalizes or may have some persistent T-wave inversions[2]

Bloodwork is non-specific or sensitive, but may show leukocytosis, elevated ESR and CRP, while positive cardiac enzymes would indicate myocardial involvement.[2,3,5,7]

There is no specific work-up for Lyme disease in the ED. The Infectious Diseases Society of America (IDSA) supports a clinical diagnosis and subsequent treatment of erythema migrans.[8] Serologic tests should only be performed 6-8 weeks into the suspected disease after antibodies have developed. The CDC recommends a 2-tier testing for Lyme Disease, which includes an initial enzyme immunoassay (EIA) such as enzyme-linked immunosorbent assay (ELISA) or immunofluorescence assay (IFA) followed by a Western blot. If the immunoassay is negative, the diagnosis of early disseminated or late Lyme disease is unlikely. If the immunoassay is positive, the Western blot should be performed and interpreted accordingly:[8]

Early Disseminated Lyme Disease

Detectable IgM and IgG antibodies to *B. burgdorferi*. An isolated positive IgM Western blot may represent a false positive test. If there's evidence of erythema migrans, the recommendation is to treat empirically and repeat testing in 3 weeks.

Late Lyme Disease

IgG should be positive. IgM may or may not be positive and should not be used for diagnosis. Serology might be positive for months to years after infection.[6,8]

Although non-diagnostic, bloodwork may show leukocytosis, anemia, thrombocytopenia, ESR>30 mm/hr, elevated liver enzymes (notably γ-glutamyl transferase). In Lyme meningitis, CSF analysis will reveal pleocytosis and elevated protein, but CSF spirochete antibodies are not recommended.[3,8] In Lyme monoarthritis, joint effusion cryoglobulin may be increased 5-fold compared with serum.[3,8]

If the most common cardiac manifestation of Lyme is observed (first- degree heart block), admission may be warranted as patients may rapidly decompensate into second and third-degree atrioventricular block, and some patients require a temporary pacemaker.[2,7]

Treatments

Treating the early manifestations of Lyme disease is important to prevent development of complications. Oral antibiotics (doxycycline, cefuroxime or amoxicillin) may be used for early localized disease or mild cardiac involvement, while intravenous antibiotics (ceftriaxone or penicillin G) are suggested for disseminated and more severe cardiac manifestations.[1,2,3,7,8] Prophylactic use of antibiotics after a tick bite is controversial, but a one-time dose of 100 mg oral doxycycline for children and 200 mg for adults may be administered.[8]

TABLE 1. Lyme Disease Antibiotics

Medication	Adult dose	Duration
Ceftriaxone	2 g IV daily	14 days
Penicillin G	20 million units IV daily	14 days
Doxycycline	100 mg PO 2x daily	14-21 days
Cefuroxime	500 mg PO 2x daily	14-21 days
Amoxicillin	500 mg PO 3x daily	14-21 days

Acute pericarditis treatment with non-steroidal anti-inflammatories and colchicine should be started in the ED. Refractory pericarditis treatment include low to moderate doses of corticosteroids with taper and colchicine for at least 6 months.[2,3,8] Patients who are stable, afebrile, without large pericardial effusions and reliable follow-up may be managed outpatient as most patients tend to recover within 3 weeks.[7]

Cases of pericarditis that are resistant to NSAIDs, colchicine and corticosteroids are extremely difficult to treat and may require pericardiectomy.[7] Unless the patient presents with cardiac tamponade, ED pericardiocentesis is not recommended and should be performed by interventional cardiology.[2,3]

TABLE 2. Pericarditis Medications

Medication	Oral Adult dose	Duration
Aspirin	1200-1800 mg daily	1-2 weeks
Ibuprofen	600-800 mg every 8 hours	1-2 weeks
Indomethacin	75–150 mg daily	1-2 weeks
Colchicine	0.5-1.2 mg twice daily	3-6 months
Prednisone	0.2–0.5 mg/kg/day	1 month

Summary

Effective management of Lyme disease and its related complications begins with recognition and diagnosis. ✶

TAKE-HOME POINTS

- As with all undifferentiated patients in the ED, taking a good history is of utmost importance to diagnose both Lyme disease and pericarditis.
- Although there are no signs or symptoms specific for pericarditis, the diagnosis can be made if the patient has a constellation of symptoms including preceding illness, low grade fever, dyspnea exacerbated by laying down, and pleuritic chest pain. ECG is helpful but unreliable and lab tests are not specific. An auscultated friction rub is very specific for pericarditis, yet it comes with environmental challenges in a busy ED.
- To diagnose a pericardial effusion, the single best tool we have in the ED is the ultrasound.
- Although not every patient with Lyme carditis requires hospital admission, even first-degree heart blocks can quickly decompensate into third-degree and the decision about disposition should be made with our consulting cardiologists.
- Treatment with NSAID and colchicine should not be delayed for suspected pericarditis.
- The same holds true for initiating antibiotic treatment for Lyme disease since delayed treatment can lead to progression of disease and confirmatory tests might take days to weeks to result.

An Unexpected Electrical Injury

A Case Report

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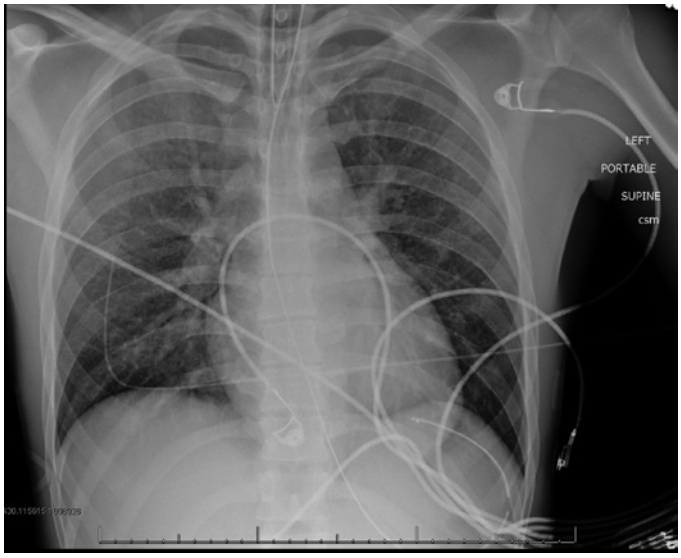
Electrical injuries range in severity from minor cutaneous burns to life-threatening internal organ damage and death. These injuries account for at least 30,000 non-fatal incidents and 1,000 fatalities in the U.S. every year.[1] The mechanism of an electrical injury varies with age, with younger children accounting for household electrical injuries via cords and outlets, older children via high-voltage power lines, and adults via work-related accidents. Two-thirds of all electrical injuries occur in electrical and construction workers.[2]

Case

A 20-year-old male was brought to the ED by EMS in cardiac arrest after an electrical injury while working as a residential construction worker. The patient was carrying an aluminum ladder that contacted overhead wires running to a house (240 volts). The homeowner had started CPR approximately 3 minutes after initial downtime. Upon EMS arrival, the patient was in ventricular fibrillation (VF). They delivered 4 rounds of defibrillation and 2 rounds of epinephrine en route to the ED, but attempts failed to achieve return of spontaneous circulation (ROSC). The estimated downtime prior to ED arrival was 25 minutes.

On arrival to the ED, the patient was in asystole with agonal respirations. He had a Glasgow Coma Scale of 3, no palpable pulses, cool extremities to touch, fixed and dilated pupils, and disconjugate gaze, with the right eye deviating laterally. Resuscitation was continued adhering to Advanced Cardiovascular Life Support guidelines. After the first round of CPR, VF was again detected, he was defibrillated, and ROSC was achieved with conversion to normal sinus rhythm. He was subsequently intubated. Vital signs were BP 164/80, HR 86, RR 16, T 35.6C (96.3°F), spO_2 97%, on 35% FiO_2. Secondary exam demonstrated erythema of the palmar aspect of the right hand, ecchymosis of the left popliteal fossa, and bilateral great toe linear "exit wound" burns with necrotic centers. His EKG showed wide QRS complexes with nonspecific ST-T changes. Chest x-ray and chest CT showed right upper and middle lobe pulmonary contusions.

Upon returning from imaging, he was noted to have equal and reactive pupils with aligned gaze. During the remainder of his ED stay, telemetry showed fluctuations from atrial fibrillation to sinus tachycardia, but no further chemical or electrical interventions were required.



Discussion

There are 2 types of electrical current: direct current (DC) found in batteries and lightning, and alternating current (AC) used in electrical wires. AC and DC can be further categorized into low-voltage (<1,000 volts) or high-voltage (>1,000 volts). When traveling through the human body, the current follows the path of least resistance. This resistance is variable based on the type of organ or tissue it is going through with the highest to lowest resistance being bone, fat, tendon, skin, muscle, blood vessel, and nerve.[3] The severity of the injury due to low-voltage AC can be increased by tetany causing prolonged contact, which is seen more in low voltages, as compared to higher voltage DC that may cause one large single muscle contraction. Many different organ systems are affected by an electrical injury, but we are going to limit the discussion to those injured in our patient.

Cardiovascular

The heart is the internal organ most commonly affected by electrical injury as it is located along many paths between entrance and exit of the electrical current.[4] Cardiac injuries can occur due to direct damage from the electrical current to the myocardium, extensive catecholamine release, myocardial infarction, severe hypotension leading to hypoperfusion, and myocardial contusion due to prolonged CPR. EKG findings may demonstrate arrhythmias, myocardial damage, and conduction abnormalities. Typically, high-voltage DC causes ventricular asystole and AC causes death via VF. Delayed arrhythmias after an initial

presentation of NSR have been seen up to 12 hours later.[5]

It is important to note that normal cardiac markers cannot rule out heart damage, as shown in our patient who had normal CK and troponin levels despite clear signs of myocardial injury. There should be high suspicion that the electrical pathway may have cardiac involvement in cases with loss of consciousness or entrance wounds on the hands. The most significant clinical predictor of cardiac injury is evidence of a vertical pathway of electricity, mapped by a line between the entrance and exit wounds.[6] Our patient was found to have a vertical pathway between his right hand and feet.

Nervous

Neurological deficits can be present throughout the central (CNS) and peripheral (PNS) nervous systems, often due to cerebral hemorrhage and infarction. The etiologies of these injuries may include but are not limited to direct damage, thermal damage, tissue separation whether due to electrostatic measures or force, and ischemic changes.[7] Acute-onset neurological deficits have a more favorable outcome than those which are delayed.[8] Immediate injuries are typically seen in the CNS with loss of consciousness as well as respiratory and motor paralysis. Delayed injuries are more often documented in high-voltage injuries with spinal cord injuries taking as little as a few hours and up to a few years to present.[9] Albeit rare, these long-term effects have included amyotrophic lateral sclerosis and other demyelinating disorders.[7] Dysfunction of cranial nerves associated with the eye as seen in our patient can occur due to reversible autonomic deficits and should not be used as indication of poor prognosis.[10]

Respiratory

Pulmonary injuries rarely present after a low or high voltage contact and are more often seen after a lightning strike. Our patient showed multiple pulmonary contusions on chest CT in his right upper and middle lobes. As he had no previous pulmonary disease, no signs of thoracic or abdominal wall trauma, or concern for respiratory infection, we suspect the pulmonary contusions were most likely caused by electrical injury, specifically the flow of electricity from his right hand to his lungs. In addition, prolonged tetany of the diaphragm and intercostal muscles may have contributed to his respiratory arrest. The pathology is consistent with two other cases describing low-voltage pulmonary contusions, making this the third case reported in English literature worldwide.[11,12] These injuries can be a result of either non-thermal effects (electricity causing cellular changes) or due to thermal effects (electricity heating surrounding air causing a blast).

Other low-voltage pulmonary injuries have included cardiogenic and neurogenic induced pulmonary edema as well as ground-glass opacities with thickening of interlobular septa and bronchial walls. With high-voltage electrical injuries, pulmonary embolism and infiltrates have been described.[12-16]

Integumentary

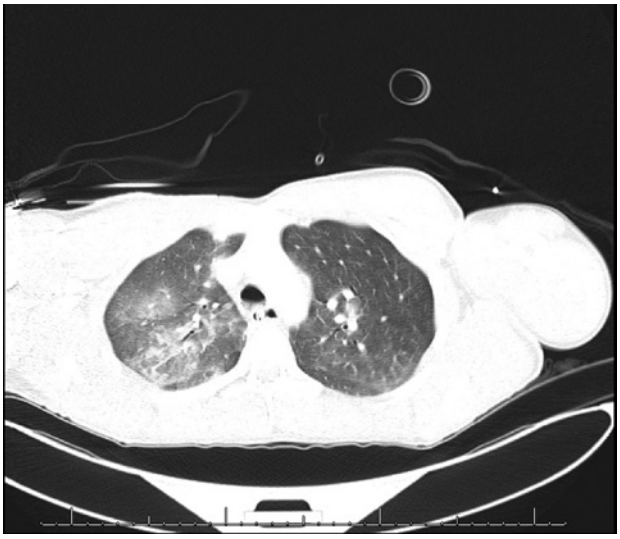
The severity of skin manifestations after an electrical injury can vary, but it is important to recognize that the level of cutaneous involvement is a poor predictor of the extent of internal injury. When present, burn marks are more commonly seen at the point of contact and where the electrical current exited the individual.[1] Our patient was noted to have an entrance wound on his right palm and bilateral exit wounds on his big toes. Kissing burns may also be seen in individuals as a result of limb flexion and the electricity arcing from one surface to another.[17] An electrical burn can be treated similarly to a thermal injury.

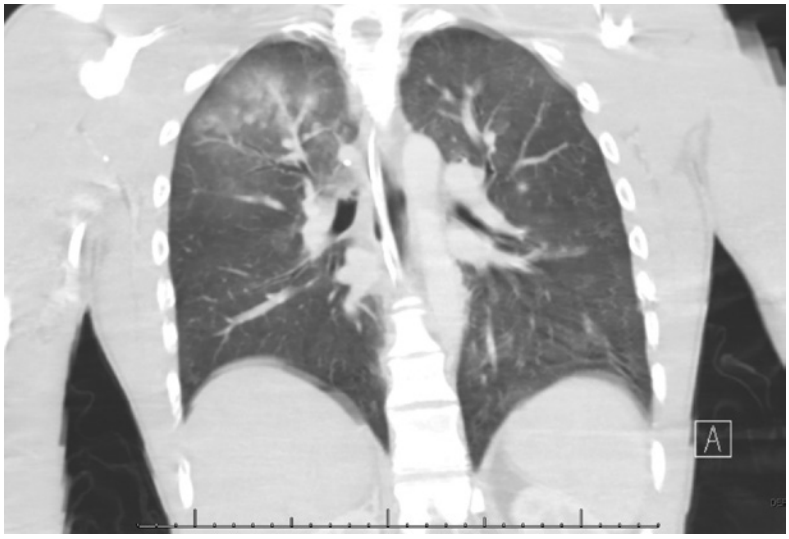
Case Resolution

After being stabilized in our community ED, the patient was transferred to a burn center. Prior to transfer, he began to have spontaneous movements requiring sedation. Upon arrival at the burn center, the patient was subsequently cooled secondary to VF arrest. By the end of hospital day one, he was more alert and was extubated without issue. His hospital course was complicated by elevated troponin levels, but he was ultimately cleared by cardiology and discharged from the hospital four days later with full neurologic recovery. ✶

TAKE-HOME POINTS

- ✓ Limited external injuries should not limit a thorough evaluation for internal injuries.[1]
- ✓ Ocular dysfunction should not be used as an indicator of poor patient outcome.[10]
- ✓ Cardiac insults should be considered with low voltage electrical injuries.[5]





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A Case of Excipient Lung Disease

Pulmonary Manifestations Following IV Injection of Crushed Suboxone

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Case

A 38-year-old male presented to the emergency department via ambulance with a 4-day history of worsening agitation, confusion, altered mental status, fever and cough. The patient's wife reported seeing a text on the patient's phone about possible recent intravenous injection of buprenorphine with naloxone. Upon arrival to the ED, the patient was afebrile (37.5°C) but extremely agitated. Initial vitals were notable for marked hypoxia (SpO2 74%) and tachycardia (140 bpm) with stable blood pressure (146/74 mmHg). On physical exam, the patient appeared to be a young, uncooperative man with diaphoresis, increased work of breathing, and accessory muscle use. Track marks were noted on his bilateral upper extremities. Due to worsening respiratory failure and agitation, the patient was emergently intubated. A CT angiogram showed a distinct tree-in-bud pattern with findings of pulmonary hypertension (Figure 1). The patient received vancomycin, piperacillin/tazobactam, and was admitted to the intensive care unit.

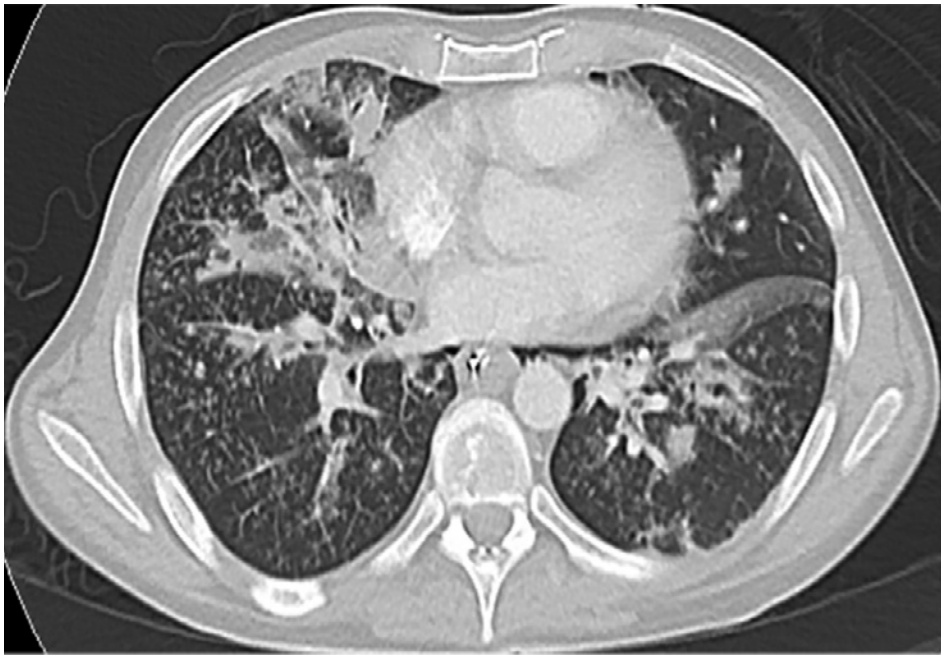

FIGURE 1. Axial cuts of computed tomography (CT) scan of patient's chest suggesting excipient lung disease. Dashed circles indicate centrilobular periarteriolar micronodules that create a tree-in-bud pattern. Solid arrows show centrilobular micronodules in the lungs corresponding to perivascular granulomas, also often seen in excipient lung disease.

Discussion

Excipient lung disease (ELD) occurs when foreign substance particles are lodged into pulmonary arterioles and capillaries and trigger a potentially fatal reaction referred to as pulmonary foreign body granulomatosis.[1,2] The exact pathophysiology of this lung disease is based on the type of pulverized agent injected. Oral tablets contain excipients, which are insoluble particulate filler materials that bind and protect the active drug during production as well as shape/lubricate the tablet for easy swallowing. Common excipients include talc, microcrystalline cellulose, crospovidone, and starch.[2] When these oral tablets are injected intravenously, these excipients may cause varying presentations of ELD. Patients with ELD are at increased risk of acute and chronic pulmonary complications such as pneumonia (10-fold increased risk), septic embolization, noncardiogenic pulmonary edema, foreign body granulomatosis, emphysema, interstitial lung disease, pulmonary vascular disease, pneumothorax, and increased incidence of fatal asthma exacerbation.[3]

Patients with ELD may initially present with nonspecific complaints such as dyspnea, cough, hypoxia with altered mental status. Advanced cases may present as acute respiratory disease syndrome (ARDS), panlobular emphysema, cor pulmonale, or acute pulmonary hypertension due to pulmonary arterial occlusion.[4] In mild cases, the physical exam may reveal only bibasilar end-inspiratory crackles. Patients presenting with more advanced disease may show evidence of acute pulmonary hypertension, including augmentation of the second heart sound, a right ventricular heave, and/or peripheral edema.[4] Physicians should also look for secondary signs of injection drug use such as needle marks, cutaneous abscesses, and hyperpigmented scars at the sites of previous injections.[5]

The initial evaluation of a patient with a history concerning for ELD should include chest radiograph, continuous pulse oximetry and arterial blood gas analysis. The chest radiograph in ELD typically shows widespread, small (2-3 mm) well-defined micronodules, often occurring in the midlung zones.[6] The typical CT findings include numerous centrilobular micronodules in the lungs (Figure 1: solid arrows), corresponding to perivascular granulomas. Although centrilobular nodules typically reflect bronchiolar disease, in the setting of excipient lung disease, they reflect embolic arteriolar disease.[7] Centrilobular periarteriolar micronodules can also appear as a tree-in-bud pattern (Figure 1: dashed circles), further mimicking bronchiolar disease.[7] The "tree-in-bud pattern" described in the case refers to small centrilobular nodules of soft tissue attenuation connected to multiple branching linear structures of similar caliber that originate from a single stalk. (See Table 1 for differential diagnosis of "tree-in-bud" pattern on CT imaging.)

In addition to centrilobular nodules, other CT findings such as enlargement of the pulmonary arteries from pulmonary hypertension and other secondary signs of right heart strain should raise concern for excipient lung disease once pulmonary embolism and chronic pulmonary hypertension have been excluded.[8,9] Echocardiography should be considered for further estimation of the pulmonary artery pressures and to rule out concomitant endocarditis.[8] If a diagnosis is still unclear following CT imaging, a transbronchial biopsy may be performed, with the specimens being sent for microbiologic culture and histopathologic examination.[10]

TABLE 1. Differential Diagnosis of Tree-In-Bud Pattern on CT Imaging[3]

Differential Diagnosis for "Tree-in-bud pattern" on CT imaging
Bronchioalveolar Infection
Congenital disorders (cystic fibrosis, Kartagener's syndrome)
Idiopathic disorders (obliterative bronchiolitis, panbronchiolitis)
Aspiration pneumonitis
Inhalation of foreign substances
Immunologic disorders (allergic bronchopulmonary aspergillosis)
Connective tissue disorders
Neoplasms

Management of ELD

The treatment of ELD is largely individualized and based on the severity of symptoms and degree of respiratory impairment. In patients who do not present with acute respiratory distress, supportive care measures may be sufficient to improve symptoms that can last from days to weeks. Following resolution of the acute episode, periodic reassessment with repeat chest imaging and echocardiography is recommended as secondary lung fibrosis and pulmonary hypertension may develop over months to years.[8]

The use of systemic steroids in patients who develop granulomatosis has limited supporting data and is generally not recommended.[11] Patients with intravenous drug use should also be counselled on their habits, treated for associated illnesses, and referred for outpatient counselling when appropriate.

The foreign body granulomatosis associated with ELD can lead to increased long-term morbidity because of the complications of angiothrombosis, pulmonary hypertension, severe emphysema, chronic hypoxia, and progressive interstitial lung disease.[8] It is also suggested that individuals who have injected a higher number of crushed pills may have a worse prognosis.[4] Lung transplantation has been performed in patients with advanced pulmonary hypertension secondary to foreign body granulomatosis.[12]

Case Conclusion

Given the initial CT findings, the patient underwent an extensive work-up for possible infectious causes. A bronchoscopy with bronchoalveolar lavage was notable for Methicillin-sensitive Staphylococcus aureus (MSSA) and blood cultures were positive for Streptococcus pneumonia and MSSA bacteremia. Respiratory PCR analysis was positive for Influenza A. A transthoracic echocardiogram was negative for valvular vegetations concerning for endocarditis but did reveal biventricular heart failure.

The patient was treated with oseltamivir for severe influenza and a prolonged course of antibiotics for sepsis secondary to bacterial pneumonia. The patient was eventually extubated on hospital day 10 without further complications. Repeat transthoracic echocardiogram showed normalization of biventricular function, and subsequent thoracic CT showed interval improvement of the tree-in-bud pattern. The patient was discharged to his home on hospital day 25 with outpatient referral for polysubstance abuse rehabilitation.

Excipient lung disease can manifest from many different injected substances and can present with clinical manifestations ranging from asymptomatic to acute respiratory failure. Knowing and recognizing radiologic patterns, in conjunction with the patient's history and physical exam, will help emergency physicians and radiologists narrow the differential diagnosis and provide appropriate treatment. ✶

TAKE-HOME POINTS

- Include excipient lung disease (ELD) in the differential diagnosis when evaluating and treating a patient with a history of intravenous drug use presenting with respiratory failure and typical findings on chest imaging.
- Patients with acute presentations of ELD may also have superimposed bronchoalveolar infections, endocarditis, and acute pulmonary hypertension; ED clinicians should keep these in mind when evaluating these patients.
- Treatment for acute presentations ELD include airway/respiratory support as indicated and supportive care. Data on the use of steroids or other interventions is limited.
- Physicians should consider a wide differential for patients with tree-in-bud pattern on CT. (Table 1)

References available online

Shockingly Dangerous Hypokalemia

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Case

A 65-year-old female presented to the ED for evaluation of generalized weakness and hyperglycemia following a syncopal episode. The patient was seated in a chair while visiting another patient in the hospital when she suddenly had a syncopal episode without reported trauma. She denied experiencing any associated chest pain or shortness of breath. She reported nausea and vomiting following the event, as well as malaise and diarrhea in the days prior to the event. She said her blood glucose had been poorly controlled in the past week. She denied any recent fevers or chills.

Her extensive medical history included coronary artery disease, congestive heart failure with depressed ejection fraction and bi-ventricular implantable cardiac defibrillator, diabetes, chronic kidney disease, depression, and tobacco abuse. She had multiple prescribed medications, notably including hydrochlorothiazide and furosemide.

Vitals on initial presentation were unremarkable, and the patient was afebrile. Other than appearing generally ill with intermittent non-bloody vomiting, the patient's physical examination was unremarkable.

During the initial evaluation, the patient began having rigid, convulsive body movements and became unresponsive with snoring respirations. Cardiac telemetry monitoring showed ventricular tachycardia. The patient's implanted cardiac defibrillator delivered a shock, leading to resolution of the ventricular tachycardia, and the patient immediately became alert and oriented. Ventricular tachycardia with subsequent ICD activation occurred several more times over the next 30 minutes.

Serial ECGs (Figures 1-3) showed inverted T-waves, wide QRS complexes, and prominent U-waves with frequent premature ventricular contractions. Point of care laboratory testing revealed: K 1.9 mmol/L, Mg 2.4 mg/dL, glucose 440 mg/dL.

Intravenous potassium was started immediately; she received 70 mEq total in the ED. An amiodarone bolus followed by infusion was also provided. The patient continued to experience several more episodes of unstable ventricular tachycardia and resolution with ICD activation. Emergent central access was obtained via femoral central venous catheter, and the patient was intubated without complications to ensure airway protection during recurrent defibrillation. Serial laboratory tests showed incremental improvement of hypokalemia, with appropriate stabilization of the patient's myocardial irritability and normalization of her ECG. This patient was admitted to the ICU and extubated the next day.

Cardiac electrophysiology was consulted for ICD interrogation and found that the patient had 14 defibrillation events in total in the ED; 7 were triggered by ventricular tachycardia and 7 were ventricular fibrillation. Additionally, electrophysiology noted the patient had an ICD discharge on each of the 2 days prior to her ED episode.

It was determined that the patient's severe hypokalemia was likely precipitated by a combination of factors: the use of hydrochlorothiazide and furosemide, several days of poor oral intake, diarrhea, and vomiting. The inpatient team discharged the patient 6 days after admission with discontinuation of hydrochlorothiazide and the addition of amlodipine for hypertension control.

TABLE 1. Common Hypokalemia Precipitants[1]

Renal Losses	Nonrenal Losses	Decreased Intake	Intracellular Shift	Endrocrine
Diureitcs (hydrochlorothiazide, furosemide)	Sweating	Ethanol abuse	Hyperventilation	Cushing's disease
Steroids	Diarrhea	Malnutrition	60%-94%	Insulin use
Metabolic acidosis	Vomtitng			
Renal tubular acidosis	Laxaitve use			
Diabetic ketoacidosis				

TABLE 2. Potassium Repletion Plan[2]

KCl Dose, maximum	Fluids	Route
60 mEQ	IL normal saline	Peripheral
10 mEQ	100-200 cc normal saline	Peripheral
40 mEQ	100-200 cc normal saline	Central

Clinical Manifestations of Severe Hypokalemia

Hypokalemia can be precipitated by a number of factors (Table 1). Severe hypokalemia can present similarly to hyperkalemia; therefore, a thorough history is paramount. When serum potassium levels fall below 2.5 mEq/L, patients may experience severe muscle weakness, muscle cramping, and even rhabdomyolysis and myoglobinuria.[1] If rhabdomyolysis is present, intracellular potassium is released and may mask the severity of the underlying overall hypokalemia.

Additionally, patients may experience respiratory muscle weakness that may lead to respiratory failure, as well as vomiting and diarrhea that will potentiate further potassium losses.[2]

As seen in this case, severe hypokalemia can result in life threatening cardiac arrhythmias. Consider hypokalemia if EKG findings include premature atrial contractions, premature ventricular contractions, sinus bradycardia, atrioventricular blocks, ventricular tachycardia, or ventricular fibrillation.[3]

There are several EKG manifestations that are characteristic of hypokalemia. Early EKG changes may include increased amplitude and width of P-waves, PR segment prolongation, T-wave flattening and inversion, ST segment depression, prominent U waves in the lateral precordial leads. There may also be an "apparent" long QT resulting from the fusion of the T and U waves, termed "long QU".[3] Be alert for concomitant hypomagnesemia in patients with hypokalemia. Patients with

FIGURE 1. EKG on arrival. Note PVCs (pink arrows), wide QRS, long QT, and developing U waves in the precordial leads (red arrows). Potassium 1.9 mmol/L.

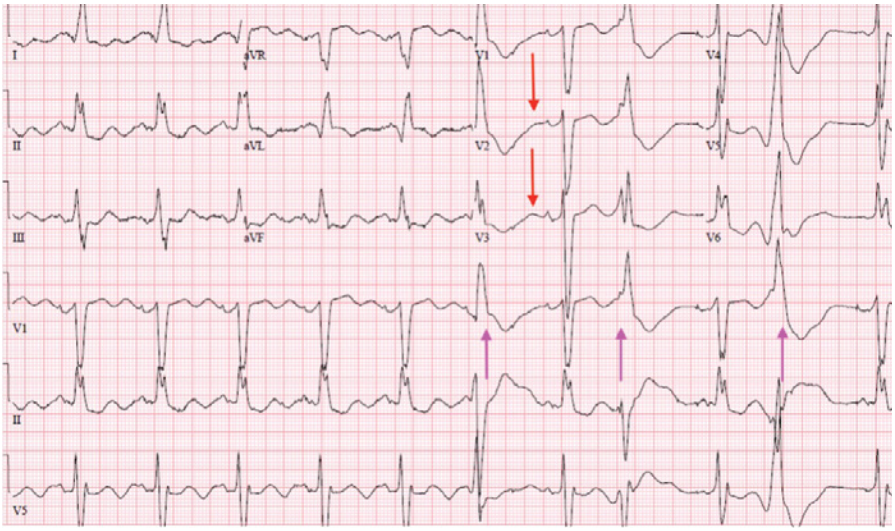


FIGURE 2. EKG after beginning infusion of IV potassium chloride. Note prominence of inverted T waves (blue arrows), long QT (green lines), U waves (red arrows) and resolution of PVCs. Potassium 2.9 mmol/L.

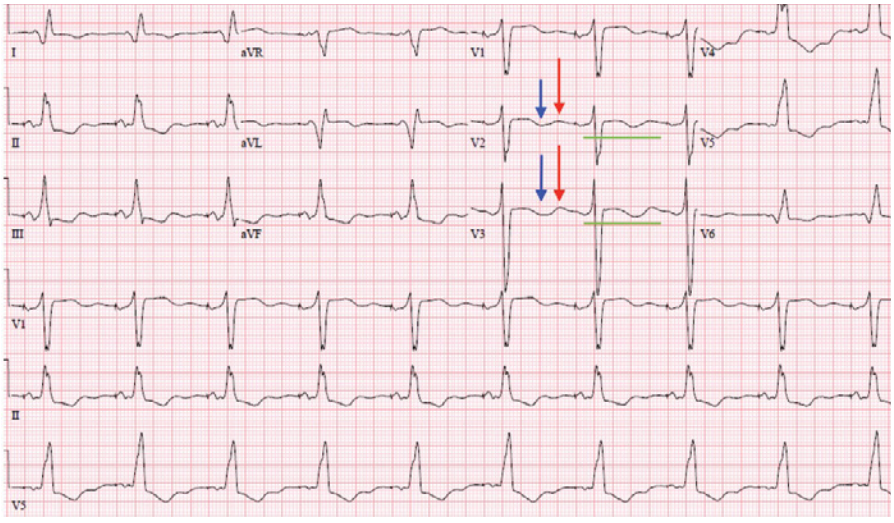
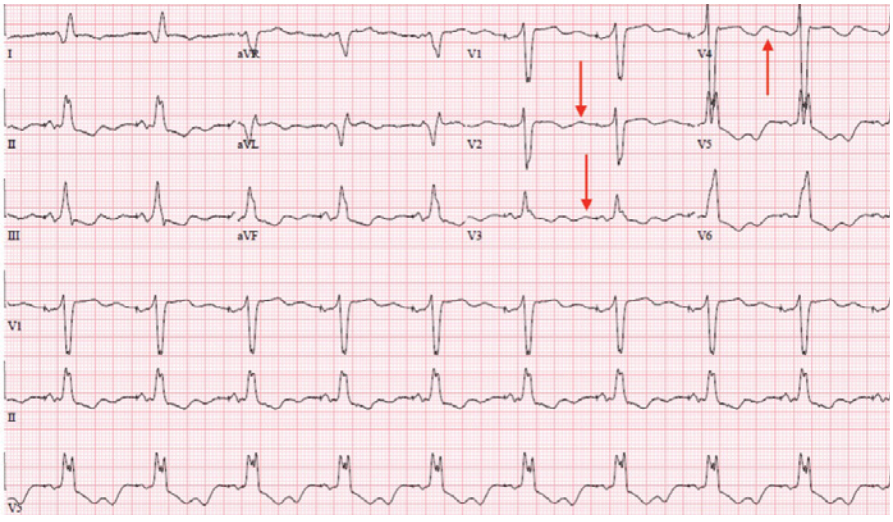


FIGURE 3. Third EKG after infusing potassium chloride with amiodarone. Again demonstrating a wide QRS, long QT, prominent but improving U waves (red arrows). Potassium 3.0 mmol/L.



both hypokalemia and hypomagnesemia have an increased risk of developing polymorphic ventricular tachycardia.[2]

Severe Hypokalemia Treatment

The treatment of hypokalemia consists of several strategies that may be utilized depending on the chronicity and severity of the disturbance. The following treatments are intended for acute life-threatening hypokalemia when serum levels fall below 2.5mEq/L or if patients are symptomatic.

In the setting of severe, life-threatening hypokalemia, IV potassium repletion should be initiated. Oral repletion may be considered as an adjunct if the patient can tolerate oral medications, however systemic absorption is slower.

Potassium chloride (KCl) is the preferred choice for IV repletion as it has faster onset than potassium bicarbonate. KCl should be administered in an isotonic saline solution *without* dextrose. The use of dextrose-containing fluids will prompt an insulin release, driving potassium into the cells resulting in further reduction of serum potassium levels.[2]

Patients with life-threatening hypokalemia secondary to GI losses, as in this case, should have potassium repleted between 10-40 mEq/hr.[1] This can be accomplished in several ways (Table 2).

It is important to be mindful of potential complications and risks associated with potassium infusions. Pain at the site of infusion and phlebitis occurs typically at rates > 10 mEq/hr when run peripherally. When faster rates or higher doses are needed, central vascular access should be obtained.

Infusing large amounts of potassium can inadvertently result in severe hyperkalemia. Caution should be taken, especially in patients with acute or chronic renal dysfunction. Normal serum potassium in the extracellular space is 50-70 mEq; rapid infusions of 40-60 mEq can result in serum concentrations that exceed safe levels. Therefore, close cardiac monitoring as well as serum potassium level checks every 2-4 hours are recommended. Repletion should be continued until serum potassium concentrations are consistently above 3-3.5 mEq/L and symptoms related to hypokalemia have resolved. At that time, dosing may be reduced, typically after the patient has already been admitted. Depending on the severity of hypokalemia, admission to a critical care service should be considered, especially if there is any indication of hemodynamic instability or if the patient will need close cardiac monitoring. ✶

References available online

A Case Study of Juvenile Ovarian Cancer and the Importance of POCUS in the ED

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Author's note: Author is father of the patient. No other disclaimers.

This case study describes the presentation of an 8-month-old female who had been misdiagnosed with constipation twice in the previous 36 hours before being correctly diagnosed with ovarian torsion and a tumor, identified as a Juvenile Granulosa Cell tumor. The overall incidence of granulosa cell tumors varies from 0.4 to 1.7 cases per 100,000 women.[1] The juvenile form of this tumor is even rarer, being only 5% of these cases. The incidence of ovarian torsion among females ages 1-20 years is estimated to be 4.9 of 100,000.[2]

The purpose of this case report is to show the importance of using appropriate imaging before settling on a diagnosis.

Case Presentation

An 8-month-old female with no past medical history presents to the pediatrician with abdominal distention (noticed by parents as having tighter clothes/diaper and visually large abdomen), fever up to 103°F, decreased urinary output, and decreased activity. This physician diagnosed the patient with constipation after ordering an abdominal x-ray to look for stool burden. This was read as normal. A few hours later, the symptoms worsened, so the patient was brought to a pediatric ED. No imaging was obtained. The patient was again diagnosed with constipation and discharged. The following day, the patient developed increased work of breathing, livedo reticularis, and increased abdominal distention. There had been no urinary output for 24 hours. She was brought to a different pediatrician who observed lack of bowel sounds. The patient was then referred to a different pediatric ED than the day before.

At the ED, triage showed a temperature of 39.9°C and RR 44. Initial labs showed a WBC of 23.8, hemoglobin of 9.4, platelet of 720, and CRP of 190. Labs and vitals were otherwise unremarkable. Abdominal exam was documented as "soft, no organomegaly, abdominal distention, bowel sounds absent. Moderated diffuse tenderness throughout." A formal ultrasound was ordered to rule out intussusception. This revealed a mass measuring 9.2 × 7.5 × 6.3cm in the lower abdomen, likely arising from the left ovary, as well as a large amount of ascites (seen in Figure 1). This was confirmed with CT (seen in Figure 2). The CT also suggested ovarian torsion.

Management and Outcome

The patient was brought to the operating room for diagnostic laparoscopy and ovarian detorsion. 400cc of bloody ascites were also drained. Three days later, an additional 800cc of bloody ascites were drained, and removal of the mass and ovary was performed. The mass was identified as a juvenile granulosa cell tumor, and the patient was referred to the local pediatric oncological hospital.

Discussion

This case demonstrates the importance of point of care ultrasound in the ED. Because no imaging was obtained during the first ED visit, the patient had to experience an additional 24 hours of discomfort caused by ovarian torsion secondary to the mass. The delayed diagnosis could also have contributed to a prolonged hospital course and increased time in the PICU. Also, an x-ray might not be sufficient imaging to implement in a case of abdominal distention, as it proved to be ineffective in this case. Abdominal x-ray has sensitivity/specificity of 82/83% for small bowel obstruction[3] and 84/72% for constipation.[4]

It is a well-established practice to use abdominal x-ray as an initial diagnostic test, but if this proves to be insufficient in providing a diagnosis, an ultrasound is a reasonable next step in workup. Ultimately, POCUS is fast and cheap and should be integrated into pediatric emergency medicine training just as it has in general EM training. Using POCUS in cases of abdominal distention could help prevent misdiagnosis. ✶

FIGURE 1. US of the ovarian mass during 2nd ED visit

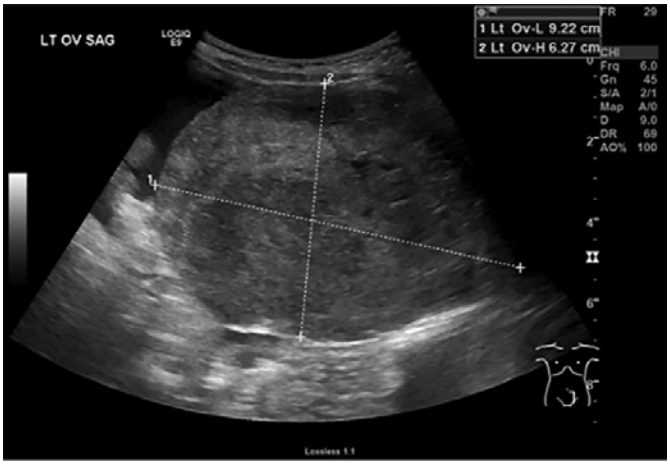
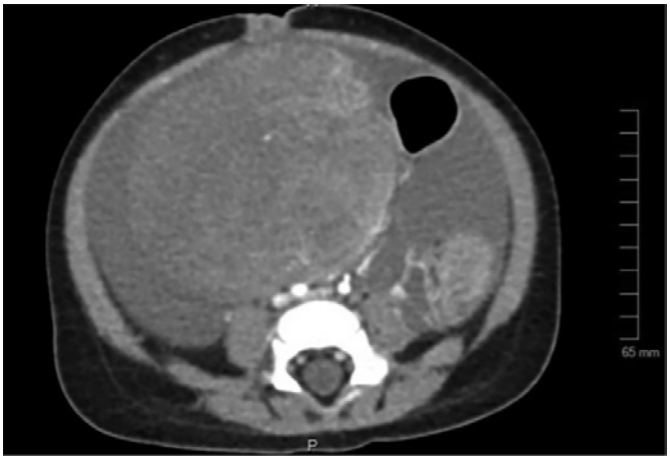
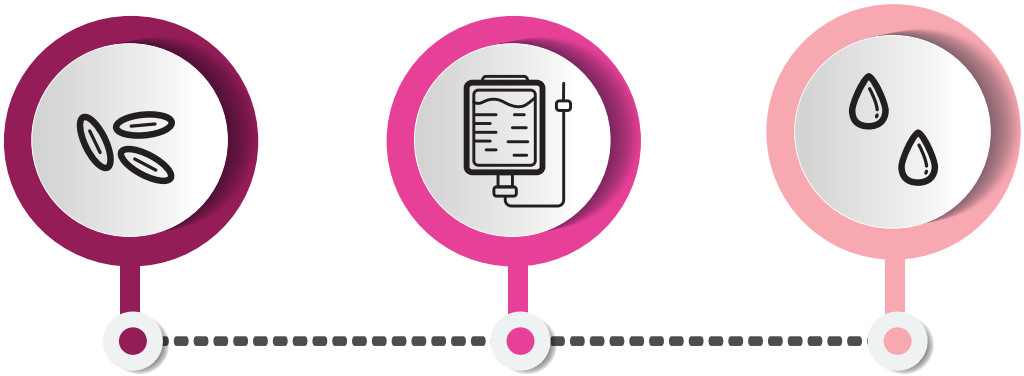


FIGURE 2. CT of the ovarian mass during the 2nd ED visit





World Sickle Cell Day, June 19

Sickle Cell Disease

Overview of Complications Seen in Pediatric Sickle Cell Disease Patients and How to Manage Them in the Emergency Department

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Epidemiology and Pathophysiology

It is estimated there are around 100,000 individuals living in the United States with sickle cell disease (SCD). The majority are of African ancestry with a minority being Hispanic, Middle Eastern, or Asian Indian descent. In addition, there are estimated to be 3.5 million people in the U.S. who are heterozygous carriers.[1]

Patients with SCD predominantly have sickle hemoglobin present in their red blood cells because of the amino acid substitution of valine for glutamic acid at the 6th position on the beta-globin chain. This substitution causes the red blood cells to develop a sickle shape and become inflexible in deoxygenated conditions, leading to increased blood viscosity as well as abnormal interactions with other cells in systemic circulation. Patients with sickle cell disease are predisposed to a variety of complications.[1]

Things to ask every patient with sickle cell disease

Any patient with a history of SCD should be asked what their typical pain crisis looks like and how their current pain varies. When obtaining a review of systems, it is important to ask if the patient has recently had a fever. Knowing the patient's transfusion history and baseline hemoglobin level will help guide clinical decision making.[2]

PEARL. Of note, patients with SCD (hemoglobin SS disease much less common HbSβ0-thalassemia) typically present earlier and with more severe symptoms.[1]

Clinical Cases to Highlight Complications of SCD *Pain*

A 16-year-old male presents to the ED for acute pain crisis. He has a history of Hemoglobin SS disease. He is on hydroxyurea for suppression therapy. He reports his current home pain regimen of first line ibuprofen and second line oxycodone have not controlled his current pain episode. He typically has severe bilateral lower extremity pain with his previous crises. Today he is presenting for similar symptoms.

Acute pain crisis (APC) is pain caused by vaso-occlusion and can involve any body system. Fever and leukocytosis frequently occur with APC and warrant an infectious investigation if present, given SCD patients' high susceptibility to pathogens.

Established pain protocols

The National Heart, Lung and Blood Institute (NHLBI) have established guidelines for the management of APC. They recommend initiation of analgesia within 60 minutes of registration or 30 minutes of triage to the ED.[4] The use of IV opioid is considered first line for APC pain management. NSAIDs should be used in conjunction with opioid therapy. The use of APC patient-management protocols are recommended to give a standardized approach for providers in the same hospital system.[2] Studies show the protocols decrease time to delivery of pain medication, decrease frequency of ED visits, result in fewer hospital days, and increase utilization of patients' primary provider for follow-up services.[4]

Pain management strategies that need further investigation

Akinsola et al. found that use of intranasal fentanyl decreased the time to first parenteral opioid in the emergency department, but overall did not show significant decrease in overall pain scores or admission rates. The investigators concluded intranasal fentanyl may be a useful temporizing measure for pain control until IV access can be established.[5]

Things to avoid: The use of supplemental oxygen is not recommended unless oxygen saturations are less than 92%.[2] Euvolemia should be maintained. The use of normal saline boluses has not been shown to reduce pain scores or admission rates.[6] Excessive hydration can lead to atelectasis, hyperchloremic metabolic acidosis and pulmonary edema.[2] Ketamine infusions are currently being utilized and investigated as a pain control option. Hagedorn et al performed a literature review which showed ketamine was useful in reducing pain scores, but

not enough data is currently available for specific clinical recommendations.[7]

Discharge can be considered if pain is adequately controlled. Patients should be discharged home with an oral equivalent of the IV pain regimen provided while in the ED. If adequate pain control cannot be achieved the patient should be admitted for further management.[2]

FEVER

A 2-year-old female with history of hemoglobin SS disease presents with fever of 102^{0}F at home. Family has been intermittently compliant with home penicillin prophylaxis. Family reports no recent cough, congestion, or other URI symptoms, and no sick contacts. Toddler is non-toxic on exam with no focal findings.

Fever in infants < 60 days: Proceed to complete full work-up per institutional guidelines. Use of Rochester criteria or home institution algorithm can help guide limited workup with discharge versus full workup with antibiotics and admission. All infants under 29 days of age warrant a CBC, CMP, CRP (or other inflammatory marker), blood culture, UA with urine culture and lumbar puncture. Providers should consider viral testing (to include HSV) and CXR on a case-by-case basis.[8]

PEARL. The NHLBI recommend oral penicillin prophylaxis until 5 years of age in all children with hemoglobin SS disease.[3]

Fever in infants > 60 days through 5 years: Children under age 5 with SCD should be on daily prophylactic penicillin. Compliance with this regimen should be elicited during the H&P to help risk stratify these children. Vaccination history is also an important component since these children are at increased risk for bacterial infections.[10] Given their increased risk for *Streptococcus pneumoniae* infections along with *Haemophilus influenzae, Neisseria meningitides,* and *Salmonellae* infections, all children under 5 with hemoglobin SS disease should receive antibiotics that cover pneumococcus such as ceftriaxone (50-75 mg/kg/dose every 24 hours).[9,13]

PEARL. Urine culture should be obtained in any febrile pediatric patient with SCD complaining of urinary symptoms.[3]

Fever in children over 5 years: Any child presenting with a fever of 101.3 ^{0} F warrants prompt evaluation to include history and physical, CBC and blood culture. If urinary symptoms are present a urine culture should also be obtained. Most patients with SCD who lack high risk criteria can be managed as an outpatient after administration of IV ceftriaxone. Patients are considered high risk if: white blood cell could greater than 30,000 or less than 5,000, fever is greater than or equal to103.1^0 F or they are ill-appearing.[3]

PEARL. Any SCD patient presenting with a fever ≥ 103.1^0 F who are ill-appearing warrant hospital admission with IV antibiotics.[1]

PULMONARY

A 6-year-old male with history of SCD presents with fever, cough and difficulty breathing. He is tachypneic with course breath sounds on exam.

Acute chest syndrome (ACS) has a classic triad of fever, hypoxia, and a new infiltrate on chest x-ray. Any SCD patient presenting with respiratory symptoms accompanied by chest pain and are hypoxic should alert the provider to consider ACS in the differential diagnosis. Children with SCD can also have pulmonary acute pain crisis. In pulmonary APC children present with a constellation of chest pain, fever, and shortness of breath like that seen in ACS but do not have an infiltrate on CXR and are not hypoxic. Pain should be managed aggressively in these patients since splitting can lead to atelectasis and pneumonia. Patients diagnosed with ACS should be admitted for close monitoring and symptom management. Antibiotics for community acquired pneumonia should be started and aggressive pulmonary toilet should be utilized.[2]

PEARL. SCD patients presenting with chest pain, hypoxia and respiratory distress who lack fever or specific chest x-ray findings should warrant consideration of obtaining a CT to evaluate for pulmonary embolism.[10]

NEUROLOGIC

A 16-year-old female with history of hemoglobin SS disease presents with new onset slurred speech and right sided weakness. She was in her normal state of health until she started experiencing these symptoms at school earlier today. EMS was called and she was transported to your department. She has no recent history of head trauma and is non-toxic on exam. Vital signs are within normal limits. Physical exam exhibits an aphasic teenager who is cooperative but anxious on exam. She has 3/5 strength in her upper and lower right extremities. Normal strength on the left. Cranial nerves are grossly intact, she has no ataxia or abnormal cerebellar testing. Her blood glucose is 70 by POC glucometer.

Stroke: Stroke and silent cerebral infarcts are the most common permanent sequelae of SCD in children and adults. Acute stroke evaluation should be considered in any SCD patient presenting with severe headache, altered level of consciousness, seizures, speech problems, and/or paralysis. A neurology consult should be obtained as well as head CT and MRI as well as MRA when available.[3] ASH guidelines recommend blood transfusion to achieve a hemoglobin of 10 g/dL or exchange transfusion in any child with SCD presenting with acute neurological deficits including TIA. The decision to transfuse should not solely rely on MRI results but the entire clinical picture.[11]

Subarachnoid hemorrhage: Children with SCD are at increased risk for cerebral aneurysms which can rupture and result in subarachnoid hemorrhage. Aneurysms are more commonly found in posterior or vestibular circulation when compared to the general population. This diagnosis should be considered in any patient presenting with sudden severe headache, nausea, vomiting, symptoms of meningeal irritation, photophobia or other visual changes, behavior changes or loss of consciousness. If neurosurgical intervention is required, it is recommended to give a blood transfusion to prevent anesthesia complications.[10]

PEARL. Consider cerebral venous sinus thrombosis in symptomatic patient with SCD and negative neuroimaging.[12]

OPHTHALMOLOGIC

A 15-year-old male presents with decreased vision in his right eye. Exam reveals fluid in the anterior chamber; the right eye is injected and actively tearing. He reports significant pain and says he was in a fight at school.

Acute glaucoma after eye injury: Patients who sustain direct or blunt force trauma to the eye orbit are at risk of hyphema. The accumulation of blood in the anterior chamber allows for sickling and the blockage of trabecular meshwork, which can lead to acute closed angle glaucoma. Patients present with a painful red eye and blood may be seen on inspection or with slit lamp exam. Consult Ophthalmology immediately.

Central Retinal artery occlusion: Presents with sudden painless vision loss in one or both eyes. No specific therapy has been characterized for management, but goal should be to optimize oxygen delivery and blood flow to prevent further ischemia while obtaining an ophthalmology consult. Most individuals affected by this will only have partial if any vision recovery.[10]

Orbital wall infarct: Typically, a younger patient, due to increased marrow space in facial bones, will present with eye pain, periorbital edema, proptosis, visual acuity changes, fever and/or headache. Symptoms overlap with periorbital or orbital cellulitis and orbital abscess. Imaging includes CT or MRI. Treatment is supportive with IV hydration and pain control and ophthalmology consultation.[10]

GASTROINTESTINAL

A 10-year-old male with SCD presents with new onset RUQ pain. He has had nausea and non-bilious, non-bloody emesis but no diarrhea or urinary symptoms. He is tender to palpation in RUQ without rebound or guarding.

Children with SCD can have any of the common etiologies of abdominal pain such as constipation, reflux, or acute gastroenteritis but they also are at risk for multiple other abdominal pathologies. In the setting of new onset abdominal pain, the clinician must consider: cholelithiasis, cholecystitis, acute intrahepatic cholestasis, acute sickle hepatic crisis and acute hepatic sequestration.[2] Children with SCD are also at increased risk of pica which in rare instance can lead to ingestion of nonfood items that accumulate and cause a bezoar.[14] In addition to a thorough history and physical exam initial studies should include a CBC, liver function test, coagulation studies. Further evaluation with imaging should include an ultrasound or CT.[2] Children with SCD are at increased risk of gallbladder and hepatic pathologies due to the increased hemolysis leading to the formation of pigmented gallstones and hepatic congestion. Complications:

Acute intrahepatic cholestasis: Sickled red blood cells can cause vascular stasis in the hepatic sinusoids. Patients can present with isolated hyperbilirubinemia, RUQ pain with or without other liver function derangements. Renal failure and coagulopathies can be present. Consider consulting Hematology for possible exchange transfusion.[2]

Acute sickle hepatic crisis: Patients present like cholecystitis to include RUQ pain, fever, leukocytosis and transaminitis in addition to hepatomegaly. Management is conservative with pain control and possible blood transfusion.[2]

Acute hepatic sequestration can occur with acute sickle hepatic crisis. Includes the symptomatology mentioned as well as an acute drop in hemoglobin and hematocrit with a reticulocytotic. Consult Hematology for simple versus exchange transfusion.[2]

GENITOURINARY

An 8-year-old male with history of SCD presents for an erection lasting greater than 4 hours.

Priapism: Ischemic priapism is the most common form of priapism encountered in SCD patients. It is due to the low flow or venous occlusion. Patients present with rigid, painful corpus cavernosa. Ischemic priapism is a medical emergency and can be distinguished from non-ischemic priapism with a corpus cavernosum venous blood gas. The blood gas typical shows an acidosis with pH < 7.25, PO2 < 30 and PCO2 of > 60. Treatment involves needle aspiration of blood from the cavernosa followed by intercavernosal injection of a sympathomimetic such as phenylephrine. Urology consultation is recommended for providers unfamiliar with this procedure.[2]

Sickle cell nephropathy and renal infarct: Presentation depends on the part of the kidney affected. If the renal medulla is involved the patient will present with flank pain and costovertebral angle tenderness on exam.[2] If there is papillary necrosis the patient will present with painful gross or microscopic hematuria.[14] Either presentation can result in renal dysfunction which should be managed with IVF hydration. Serial examination of renal function is recommended.[2]

HEMATOLOGIC

A 3-year-old female with history of hemoglobin SS disease presents for pallor, difficulty breathing, and fatigue. Per family, the patient had not appeared ill but woke up looking pale, uninterested in playing, and wanting to rest while appearing to breathe harder than usual. Exam reveals palpable spleen 5 cm below the costal margin; she is tachycardic and tachypneic.

Splenic sequestration: A drop in hemoglobin ≥ 2g/dL below baseline and acute increase in spleen size with elevated reticulocyte count. Patients report abdominal pain and/or fullness, pallor, and lethargy. Exam can show splenomegaly, tachycardia, and other signs of hypovolemic shock.. Treatment is with blood transfusion.[14]

Aplastic crisis: Infection with parvovirus B19 can induce transient red cell aplasia. Due to the shortened lifespan of red blood cells in SCD patients they are at increased risk for significant drops in hemoglobin. Patients can present with a viral syndrome of gradual onset of pallor, fatigue, and headache. In extreme cases patients may present in hypovolemic shock. This clinical presentation can be differentiated from splenic sequestration with normal spleen size and low reticulocyte count. Treatment is slow blood transfusion.[10]

Summary

Patients with SCD presenting to the ED are at risk for unique complications but can also have garden-variety diagnoses. It is important for the clinician to take into consideration the entire clinical picture and obtain appropriate diagnostics based on recommended practice guidelines. ✶

References available online

BE WARY OF THE BRUISE

Don't Forget the Full-Body Exam

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You are in the emergency department and a 1-month old female presents with her father for dyspnea. According to the father, she was being removed from the bath the night before when she slipped out of his hands, hitting her face on the nearby counter. He was able to grab her by the arm before she hit the ground. When asked why he did not seek medical care, he stated "I thought she was fine." Physical exam showed an abrasion to the bridge of the nose with underlying ecchymosis, ecchymosis circumferentially around the left arm, and an abrasion to the inferior gum line. Her lungs were clear to auscultation bilaterally with no tachypnea or retractions. What would you do if this patient presented to your ED?

Between the overflow of patients recently, the ED has been in chaos recently. The combination of social distancing, abundant precaution, and fear of Covid-19 have contributed to a drop in our pediatric census. In a recent New York Times article, author Nikita Stewart reported, "In the first eight weeks of spring 2019, New York City's child welfare agency received an average of 1,374 cases of abuse or neglect to investigate each week. In the same period this year, that number fell to 672, a decline of 51 percent."[1] With the added physical and mental stress of a pandemic affecting parents all over the U.S., it is interesting to note that we have not seen an increase in reported abuse and neglect. Has abuse really diminished? Or does the decrease in pediatric presentations mean fewer children are receiving medical care and appropriately indicated work-ups? Before the pandemic it was estimated that between 2-10% of children visiting the ED were victims of child abuse or neglect.[2] In the coming months and year we must be on the lookout for these patients and have a high index of suspicion for child abuse.

As emergency medicine resident physicians, we must be vigilant for these patients. A study of 44 children who died of child abuse showed that 20% of them were evaluated by a physician within a month prior to their death. It was determined that 71% of those evaluations were in an ED.[2] At times we are the only medical care these high-risk patients receive and possibly their only chance for intervention. How, then, do we get better?

The most important part of these evaluations is the physical exam. Although the caretaker can be dishonest, your physical exam is an objective measure of truth. In a study with 200 infants who had experienced confirmed severe abuse, 55 (27.5%) had a sentinel injury before the episode of severe abuse. Of the 55, 80% had bruising and 11% had intraoral injury, often bruising to their frenulum.[3] All children under one year of age, regardless of chief complaint, should be placed in a gown to facilitate a full-body exam. This is an easy way to facilitate more thorough examinations in a chaotic emergency department. Bruising should be used as a screening tool for child abuse, and careful attention should be paid to location of the bruise, age of the child, and distribution of ecchymosis. **The TEN 4 FACES P** decision rule can be a useful clinical aide when suspecting abuse.

Location

Bruises that were predictive of abuse were located on the torso, ear, or neck of a child younger than 4 years of age. Bruising in any region on an infant younger than 4 months of age was also predictive. The sensitivity of this decision rule was 97% and the specificity was 84% for predicting abuse.[4]

Patient Age

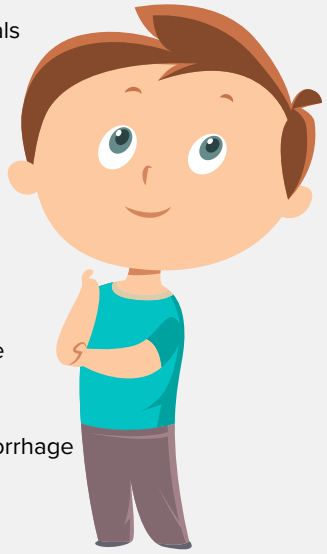
Those that don't cruise rarely bruise.[5] One study on prevalence of bruising in infants in the pediatric ED found a significant difference in bruise rates for infants younger than 5 months old compared to those older than 5 months old. The prevalence of bruising for infants 5 months and younger, in comparison to those older than 5 months, was 1.3% to 6.4%.[6] Infants younger than 5 months old should not present to your ED with bruising. As soon as the child starts to crawl or walk, bruising will become more common. These bruises should not be located in the aforementioned locations.

TEN 4 FACES P decision rule:[4] any bruises present in any of the following locations should be concerning for child abuse.

T Torso including genitals
E Ears
N Neck

F Frenulum (bruising from forcing a bottle into a child's mouth)
A Angle of the mandible
C Cheek
E Eyelid
S Subconjunctival hemorrhage

P Patterned bruising



4 Any bruising in a child less than 4 months of age

Patterned Bruising

Patterned bruising should always be concerning. Hand imprints are common, as are the imprints of objects like belts, cords, shoes, and kitchen utensils.[7] Look for bruising that is consistent with the items mentioned above.

Bruise Age

What if the bruise looks old? In a study of fifty children with accidental bruises, EM pediatricians accurately estimated bruise age (within 24 hours of the actual age) 47.6% of the time.[8] Your judgement alone is not a reliable enough way to determine whether a bruise is younger than 24 hours old.

Is non-accidental trauma the only cause of bruising? Of course not. Patients with concerning bruising should be admitted to the hospital for a full workup, including further testing for other causes of bruising such as bleeding diatheses, coagulopathy, infection, thrombocytopenia, and other etiologies. We must not be afraid to advocate for children and report possible child abuse. A bruising prevalence study conducted in three pediatric EDs found that only 23% of pediatric patients found with bruising (88/2344) had an abuse evaluation. According to the author this was far fewer than expected.[6] Although a bruise increased the threshold for evaluation, it was not enough. Most children with suspicious bruising should be reported to the proper authorities and evaluated for further injury. It is more acceptable to report child abuse and be wrong than it is to not report and risk a child dying or sustaining serious injuries from child abuse.

In the case mentioned at the beginning of this article, the patient was admitted for further evaluation and found to have multiple new and healing rib fractures, retinal hemorrhages, and a subdural hemorrhage. All findings were consistent with non-accidental trauma. Although with younger children illness and injury is not always obvious, we must stay vigilant of the bruise, and keep non-accidental trauma on our minds and in our differential. If you do not look for it, you will never find it. ✶

References available online

ONE BAD HAND OF POKER

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A 77-year-old man presented to the emergency department after being punched in the throat over a dispute involving a card game. He reported immediate odynophagia, abnormal phonation, and small volume hemoptysis, but had no difficulty breathing.

Physical exam was notable for anterior neck pain over the thyroid cartilage with palpable crepitus. Bedside nasopharyngolaryngoscopy (NPL) was performed (Figure 1), followed by a computed tomography (CT) of the neck with intravenous contrast (Figure 2), confirming the diagnosis.

Diagnosis

Tracheal cartilage fracture with airway edema

Discussion

Tracheobronchial injury (TBI) is a rare, but potentially life-threatening, complication of neck trauma. It represents 1 in 30,000 ED visits per year. Fracture of the tracheal cartilage is most commonly the result of direct anterior blunt trauma, and can result in significant airway edema, tracheal laceration, hemoptysis, pneumothorax, and disruption of adjacent vascular structures. Cricoid cartilage fracture is associated with recurrent laryngeal nerve damage. NPL may demonstrate airway edema or bleeding. If the patient is stable, CT with contrast should be obtained to evaluate for significant tracheobronchial and vascular injuries in symptomatic patients following anterior neck trauma. Patients with tracheobronchial trauma have a high risk of other injury, especially with motor vehicle accidents, so imaging for C-spine injury and skull fractures should be obtained.

Early intubation with a fiberoptic scope, to ensure placement of the cuff distal to the site of injury, should be considered given the risk of progressive airway edema. An emergency cricothyroidotomy may also be necessary depending on the degree of trauma to the airway. Blind intubation should never be attempted due to the high risk of creating a false passage.

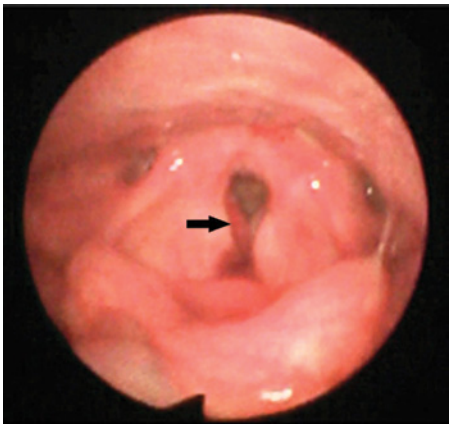

FIGURE 1. Iberoptic NPL demonstrating significant supraglottic edema and hooding of the false vocal cords (black arrow).

Surgical consultation should be obtained if the injury is severe enough to cause disruption of basic functions such as swallowing or phonation. Surgical options include observation, plating of fractures, or stenting of the airway. Displaced fractures will typically require open reduction in the operating room; 21% of patients who undergo surgical repair have phonation difficulties post-op.

Nonsurgical management options include elevation of the head of the bed to reduce edema and manage secretions, voice rest, cool humidified air to improve ciliary management of secretions, steroids for reduction of edema, and proton pump inhibitors to reduce laryngeal inflammation from acid reflux. Antibiotics may also be used as prophylaxis.

The most common cause of blunt neck trauma is motor vehicle accidents. Many of these accidents involve collision of the patient's neck against the steering wheel or windshield. Other possibilities include sports accidents such as clothesline tackles. History and physical exam will typically reveal the symptoms seen in our patient, but can also involve respiratory distress, an expanding hematoma, edema, ecchymosis, or distorted anatomical landmarks. Adult thyroid and cricoid cartilages will typically fracture in multiple places due to ossification, whereas in children, they tend to fracture in one place. In children younger than age 3, the cricoid cartilage sits higher at C4, versus at C7 in most adults.

The neck is divided into 3 anatomic zones, with zone 1 being sternal notch

Schaefer Classification System of Laryngeal Injury

Group	Injury	Treatment	Comments
0	Normal	None	
1	Mild hematoma or laceration without fracture	Observation, humidified air, medical management	
2	Moderate edema, hematoma, mucosal disruption without exposed cartilage, non-displaced fractures	Tracheostomy, direct laryngoscopy, esophagoscopy	Serial examinations due to frequent worsening over time. Usually do not require tracheostomy
3	Major edema or lacerations, exposed cartilage, displaced fractures, or vocal cord immobility	Tracheostomy, exploration/repair	Usually requires surgical repair.
4	Group 3 and disruption of anterior larynx, unstable fractures, two or more fracture lines, or severe mucosal injuries	Tracheostomy, exploration/repair, stent required	Require tracheostomy and stenting to maintain larynx.
5	Complete laryngotracheal separation	Surgical repair	Require an airway emergently placed directly into the trachea through the neck below the injury

to cricoid cartilage, zone 2 being cricoid cartilage to the angle of the mandible, and zone 3 being above the angle of the mandible. Hard signs of vascular injury, including expanding hematoma, bruit or thrill, or cerebral ischemia require immediate surgical consultation. In a stable patient such as ours with soft signs of injury (hemoptysis, dysphagia, dysphonia, subcutaneous air, and crepitus) a computed tomography angiogram is indicated.

Aerodigestive injuries can be very difficult to identify on initial presentation but should be pursued if clinically indicated (dysphagia, blood in gastric contents, or crepitus). These patients will typically require endoscopy or barium swallow to identify injuries. Antibiotics might also be indicated if suspected due to gastric contents.

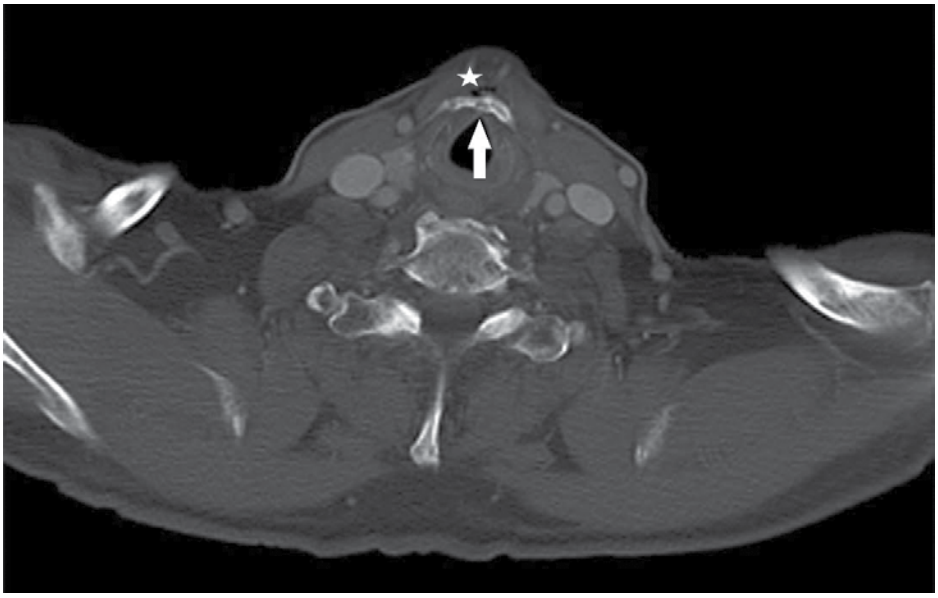

FIGURE 2. CT of the neck demonstrating tracheal cartilage fracture (white arrow) and mild anterior subcutaneous emphysema (white star).

Case Conclusion

In our patient, bedside NPL demonstrated significant supraglottic edema and hooding of the false vocal cords. CT confirmed tracheal cartilage fracture, with evidence of subcutaneous emphysema indicating an injury to the trachea. The patient was given dexamethasone and admitted to an ICU for airway monitoring and serial NPL examinations. His swelling improved after 2 days, and he was discharged with uneventful follow-up. He regained normal phonation and swallowing function several weeks after discharge. It is unclear if he ever played cards again with the same group that caused him to spend a night in the intensive care unit. ✶

Sick as a Dog

A Case of Overwhelming Post-splenectomy Infection

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Case

A 57-year-old woman with a history of antiphospholipid syndrome, ITP, emphysema, and recurrent mesenteric vein thrombosis status-post bowel resections and splenectomy presented to the emergency department with fever, chills, and vomiting for 1 day. The patient reported multiple episodes of non-bilious non-bloody emesis over the prior 24 hours without abdominal pain. She had several loose stools, but no melena or hematochezia. She denied cough, shortness of breath, chest pain, sore throat, neck pain, or headache. Over the day prior to presentation, she had become increasingly weak and confused, prompting her partner to bring her in to the ED for evaluation. She reported no recent travel or sick contacts, no toxic ingestions or substance abuse, and multiple pets in her home.

Her triage vital signs were BP 123/100, HR 136, RR 24, temperature 97.5°F, and oxygen saturation 100% on room air. On physical exam she was ill-appearing. HEENT exam was unremarkable. Cardiovascular exam was significant for tachycardia with a regular rhythm. She was tachypneic but had an otherwise normal pulmonary exam. Abdominal exam was non-tender throughout, but did reveal multiple healed scars from prior surgeries. She was confused about the date but had no focal neurologic deficits and was answering questions. Her extremity exam showed no peripheral edema. Her skin was noted to be pale with delayed capillary refill of >3 seconds. Petechia and early purpura were noted on her back.

What is your working differential diagnosis? How would you treat this patient?

Workup

An initial point-of-care blood glucose demonstrated critical hypoglycemia to 25 mg/dL. iSTAT lactate was elevated at 10.8 mmol/L. Venous blood gas showed a pH 7.25, pCO2 31.7 mmHg, PO2 12 mmHg, HCO3 14 mmol/L, and Base Excess of -13. Laboratory studies returned with an abnormal CBC including WBC 3.1 K/uL, Hb 12.1 g/dL, platelets 28 K/uL, which also showed Howell-Jolly bodies and many intracellular bacteria. Chemistry resulted with a sodium of 138 mmol/L, potassium 6.4 mmol/L, chloride 98 mmol/L, CO2 10 mmol/L, urea nitrogen 56 mg/dL, creatinine 2.87 mg/dL, anion gap of 30, calcium of 9.8 mg/dL, bilirubin 0.6 mg/dL, AST 116 U/L, ALT 56 U/L, alk phos 66 U/L, albumin 4.0 g/dL, protein 7.0 g/dL. Prothrombin time was 25.4 seconds, INR 2.3, partial thromboplastin time 201.2 seconds, D-dimer was 7,695 ng/mL FEU, and fibrinogen 400mg/dL. Influenza PCR was negative.

With dextrose administration glucose improved to 236. Blood gas in the subsequent 2 hours worsened to 7.20/CO2 35/O2 17, HCO3 13.9/ BE -14, with a mild improvement in lactate to 9.03 after volume resuscitation and antibiotics. Chest x-ray did not reveal a source of infection. CT of the abdomen did not show any acute cause of severe sepsis. Arterial vasculature was patent, portal vein and SMV showed chronic thrombus seen on imaging 10 years prior. Non-contrast CT of the head was unremarkable. The patient developed worsening respiratory distress and confusion, ultimately requiring intubation while still boarding in the emergency department.

Within 6 hours of arrival, aerobic blood cultures grew *Capnocytophaga canimorsus*.

Case Discussion

Background

The patient described in this case developed overwhelming post-splenectomy infection (OPSI) with septic shock and multi-organ failure due to *Capnocytophaga canimorsus* bacteremia. This gram-negative bacterium is commonly isolated from the oral microbiota of dogs and less commonly in cats.[1] There are 7 species of Capnocytophaga, all of which can be found in the oral cavity of both humans and domestic animals. It is estimated that somewhere between 8% and 41% of canines have a colonized oropharynx.[1,2] *Capnocytophaga canimorsus* infections in humans are most commonly associated with dog and cat bites, and to a lesser extent, scratches and licking.[3] The first human infection was described in 1976, and there are very few documented cases each year.[4,5]

Clinical Presentation

A patient with a remote splenectomy may not recognize initial symptoms of infection, and may be unaware of how quickly infections can become fulminant. In addition, they may not associate a canine bite or other common exposures with symptoms of infection, so a careful

history is essential in all ill-appearing splenectomy patients. In the case of *C. canimorsus* infection, symptom onset typically occurs within 5 days of bite, and hospitalization occurs on average at 7 days after exposure.[1,6] The infection may present in a variety of ways, from localized skin and soft tissue infection to systemic symptoms of septicemia.[1] In those who develop sepsis, the most common symptoms are fever (78%), chills (46%), vomiting (31%), diarrhea (26%), abdominal pain (26%), dyspnea (23%), confusion (23%) and headache (18%).[7]

Sepsis due to *C. canimorsus* can be devastating, with mortality rates estimated to be 30%.[5] It is essential that bacteremia is considered early and antibiotics that treat *C. canimorsus* are administered promptly. The rapid progression of disease suggests that the organism may be able to evade the immune system.[1] While it is possible for severe sepsis to occur in immunocompetent individuals, in 33% of cases there is a history of splenectomy, which is also associated with a higher risk of death.[1,8,9] Besides splenectomy, other risk factors for severe disease include immunosuppression and alcohol abuse.[5]

In the case of this patient, fever, vomiting, and confusion were the primary symptoms, although she was afebrile upon presentation. She had three SIRS criteria and a qSOFA score of 2, which initiated work up and treatment for sepsis. Her confusion, tachypnea, purpura, and extremely high lactic acid were all ominous signs of a life-threatening infection. On review of the patient's chart, she had a remote history of splenectomy which predisposed her to infection with encapsulated bacteria, intraerythrocytic parasites, and gram-negative bacteremia such as *C. canimorsus*.[10] While there was no specific history of animal bite, her exposure to pets may have put her at risk for this infection.

ED Management

The first step in ED management is consideration of life-threatening infection and atypical pathogens in a patient with a history of splenectomy who presents with sepsis or septic shock.[1] *C. canimorsus* is one of the pathogens which may be considered in such patients, and the emergency physician should take a careful history, asking the patient about any exposure to animals or recent bite wounds.

The spleen is responsible for phagocytosis of bacteria and production of antibodies. Asplenic patients are at risk for serious infection from not only encapsulated bacteria, but also other pathogens like *C. canimorsus*.[10] Patients may develop OPSI which is characterized by initial vague symptoms (fever, malaise, headache, vomiting, diarrhea) followed by septic shock with disseminated intravascular coagulation and mortality of 50 to 80%, even with appropriate antibiotics. Mortality can be reduced by if patients seek medical care and are treated immediately, which is why early recognition in the ED is essential.[11]

The ED work up should include a complete blood count, chemistry, lactate, and blood cultures. Peripheral blood smear examination may demonstrate extracellular and phagocytosed bacilli.[12] Blood cultures often confirm the diagnosis of *C. canimorsus* (88% of cases).[13] However, because blood cultures may not become positive during the ED stay, it is essential for the EM physician to have a high index of suspicion for bacteremia in a patient presenting with sepsis. If there is a concern for meningitis, a lumbar puncture should be performed to obtain cerebrospinal fluid (CSF). The cerebrospinal fluid culture may take an extended period of time to become positive (median of 5 days, with a range of 1-19) so it is important to continue culture growth beyond five days.[14] Antibiotics should be given based on clinical concern and the physician should not wait for a clear source of infection to be identified in laboratory work up, cultures or CSF.

The patient should be given a broad-spectrum antibiotic that covers *C. canimorsus* while awaiting susceptibility testing. For the initial treatment of severe infections, empiric antibiotics should include a carbapenem or a beta-lactam beta lactamase combination such as pipercillin-tazobactam, though the bacterium may end up being susceptible to a variety of other antimicrobials such as penicillin G, linezolid, chloramphenicol, third generation cephalosporins, fluoroquinolones, erythromycin, doxycycline, and metronidazole.[1,15] It is unlikely that a diagnosis will be confirmed during the ED stay, and thus for severe infections broad-spectrum antibiotics should be administered while the differential remains open. Non-severe infections may instead by treated with oral amoxicillin-clavulanate or clindamycin. For confirmed cases, duration of treatment ranges from 14-21 days.[6]

Finally, in order to prevent life-threatening infection from *C. canimorsus* and other bacteria, prophylactic antibiotics (usually amoxicillin-clavulanate) should be given to asplenic individuals who have been bitten by a dog, even in the absence of infectious symptoms.[1,5] Currently there is not clear utility on the testing of canines, since it does not guarantee that particular dog will stay infection-free. Furthermore, isolation from canine saliva or bite wounds is challenging.[1]

Case Conclusion

Patient was admitted to the MICU for septic shock due to Capnocytophaga canimorsus *bacteremia. Her course was complicated by anuric renal failure with severe metabolic acidosis requiring initiation of CRRT. Hemodynamics continued to worsen, and within hours the patient progressed to PEA arrests and ultimately passed away despite maximal support.* ✶

TAKE-HOME POINTS

- All post-splenectomy patients who present with fever or other signs of sepsis should be treated with broad-spectrum antibiotics while work-up is pending, as infections may become rapidly fulminate (overwhelming post-splenectomy infection).
- Although it is rare, it is important to recognize *C. canimorsus* sepsis, as it is associated with high mortality rate and the patient can deteriorate rapidly.
- *C. canimorsus* bacteremia results from bites, scratches, and licking by dogs and cats among patients at risk, such as those with splenectomy, history of alcohol abuse, or immunosuppression.
- Gram negative rods may be seen in the blood smear for patients with *C. canimorsus* bacteremia.
- For severe cases, preferred antibiotics to treat *C. canimorsus* include carbapenems or a beta-lactam beta lactamase combination.

EM Use of Sphenopalatine Ganglion Block for Migraine Headaches

Tickle Your Brain to Numb the Pain

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The most prevalent medical problems around the globe are in the sphere of headache disorders. Between 50% and 75% of adults between ages 18-65 will have headaches. Worldwide, the disability and loss of productivity due to headache disorders being ranked third in the number of years lost to disability.[1] However, primary headache disorders, of which migraines are the most prevalent, only account for up to 4% of chief complaints for emergency department (ED) admissions.[1,2] Migraine therapies revolve around preventive measures in anticipation of migraines and adjuvant treatments for associated symptoms of migraines such as nausea and emesis. Another key therapy for migraines is abortive treatment. Preventative treatments include antidepressants, antihypertensive, ant antiseizure medications. However, triptans and NSAIDs are the mainstay of abortive treatment, which must be delivered at the onset of symptoms.[1] This article intends to discuss the procedure, use, and relevance of Sphenopalatine Ganglion (SPG) blockade in the abortive treatment of migraines and other noteworthy primary headaches not due to trauma. The research behind SPG blocks and the application to ED use will also be discussed.

Research Behind SPG Blocks

The culprit behind headache disorders are largely the nerves that converge at the sphenopalatine ganglion (SPG), an extracranial parasympathetic ganglion.[3] The SPG is located behind the nasal bony structures, giving rise to the superior, inferior and posterior lateral nasal branches, nasopalatine nerve, greater and lesser palatine nerves, the pharyngeal branch of the maxillary nerve, and orbital branches that innervate the lacrimal gland.[3,4] During a headache meninges become inflamed, leading to activated pain receptors per the trigeminal nerve. These pain receptors transmit impulses through the SPG to autonomic nerves.

SPG involvement results in lacrimation and nasal discharge noted in migraines and other primary headaches. In the midst of a migraine, parasympathetic outflow from the SPG results in cranial vasodilation. Inflammatory mediators are able to activate meningeal pain receptors leading to migraine pain.[3]

The SPG relays sensory, sympathetic, and parasympathetic pathways.[4,5] Sensory pathways for pterygopalatine branches with the maxillary nerve to terminate at the trigeminal ganglion, innervating parts of the head and neck. Sympathetic pathways arise from the superior cervical ganglion to converge at the SPG to innervate vasoconstriction in the nasal cavity, upper pharynx and palate. The parasympathetic pathways arise from the superior salivatory nucleus to join the SPG, innervating the lacrimal, nasal, and other oropharyngeal glands.[4,5] As such, blockade at the SPG theoretically should propagate relief of symptoms associated with a headache state: meningeal vessel inflammation, lacrimation, rhinorrhea.

SPG blocks have been shown to be effective in the reduction of headaches, including but not limited to cluster headaches, chronic migraine, refractory trigeminal neuralgia, and post-operative analgesia for endoscopic sinus surgery.[5] Approaches within the studies considered in the literature do not all use the procedures noted in the SPG Procedures and Steps section of this article. Variable approaches included intranasal, intraoral, transcutaneous as well as the use of radiologically guided needles to achieve the desired a SPG block.[5]

Immediately following the procedure, the patient may already be

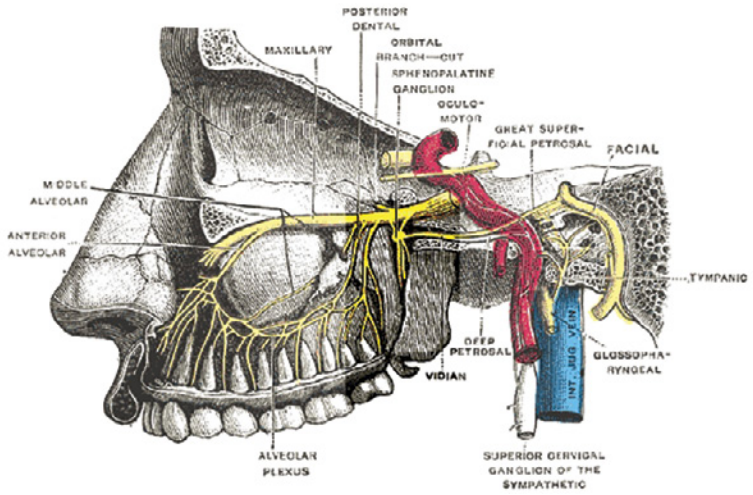

Image obtained from Wikimedia Commons; picture is public domain. All other images were taken with a volunteer in a non-clinical setting.

feeling relief! If they have numbness in the posterior pharynx, advise them not to eat or drink until full feeling returns.

Note: There are several proprietary applicators for blocking the SPG, with the SphenoCath® being the most studied. It can still be performed with an inexpensive simple cotton tip applicator but studies will need to be performed to determine any difference in efficacy.

Verdict: Is this realistic for use in EM?

Research behind SPG blocks is consistent in demonstrating efficacy in reducing or even resolving migraine headaches. It should be noted that many of the studies supporting SPG blocks are based in outpatient settings such as pain clinics. Anecdotally, these blocks' efficacy can be externalized to the EM setting, but more research will need to be done to prove this.

Even if you are sold on the SPG block as an effective treatment for migraine headaches, it will not likely be your first approach to every patient with a migraine. Ordering prochlorperazine and diphenhydramine will remain an effective and relatively easy treatment for most migraines, and the medications take less time to order than it does to perform the SPG block procedure.

One strength of the SPG block, however, is that it has been shown to work relatively quickly. As much as 70% of migraines improved after 15 minutes in one case series, although bilateral blocks were utilized in this study.[7] Other treatment remedies may have similar times to treatment success but would be more sedating and could require longer ED stays. Besides the time involved in the procedure, another downside of the SPG block is proposing to a patient that a cotton tip applicator be placed several inches into their nostril. However, given how prevalent placing nasal swabs in nostrils has become over the past year, this just may be time for the SPG block to shine. ✶

SPG BLOCK DESCRIPTION & STEPS

Step 1. Gather the following materials:
- Cotton tipped applicator, ≥ 6 inches
- Anesthetic of choice
  - Lidocaine, 1-4%
  - Bupivicaine, 0.25-0.50%
  - No difference in efficacy between the two anesthetics[6]
- Small cup or container for anesthetic
- 5 mL syringe with needle to draw up anesthetic

Step 2: Position patient in the supine position with the neck extended.

Step 3. Soak cotton tipped applicator in the anesthetic solution, and draw up anesthetic into the syringe.

Step 4. Place cotton tipped applicator in the nostril of the affected side, advancing through the middle turbinate until resistance is met.

Step 5. Use the needleless syringe to drip 1-2 mL of anesthetic solution down the shaft of the cotton tipped applicator. Note: some providers' preference is to only use the anesthetic on the cotton tip and not drip any into the turbinate.

Step 6. If this procedure is used for a bilateral headache such as a known cluster headache, repeat steps 3-5 on the other side. However, take care to not administer SPG blocks on bilateral headaches of unknown cause so as to miss new life-threatening pathology.

Step 7. Allow the patient to rest supine for 10-15 minutes. Keep patient on cardiac monitor due to the nature of the procedure.

Step 8. Remove the cotton tipped applicator(s). Monitor for side effects, most commonly epistaxis.

Patient Outcomes in ED Observation Units for Syncope

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Mount Sinai Morningside – West
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Syncope is a complex presenting chief complaint spanning several physiologic systems. Its differential includes several "can't miss" etiologies, making diagnosis and disposition sometimes difficult for the emergency provider. Per The Framingham Study, the estimated incidence of syncope is approximately 6.2 per 1000 person years, accounting for approximately 1% of all ED visits in the United States.[1,2] It is estimated that syncope workup and management costs the US healthcare system an annual 2.4 billion dollars[3]. The development of ED Observation Units, or EDOUs, have allowed patients and hospitals alike to seek specialized care that is less costly and, in some instances, just as efficient at diagnostics as a full hospitalization. The literature review below will focus on patient-centered outcomes in EDOUs for syncope.

Review of Literature

1) Syncope Evaluation in the Emergency Department Study (SEEDS): a multidisciplinary approach to syncope management. (Circulation, 2004)

This prospective, single-center study randomized patients in an intermediate-risk group for syncope (met by pre-determined clinical features) into admission versus EDOU for syncope. Notably, the intermediate group contained patients with high-risk features — 43% had a history of CAD, and 57% had an abnormal ECG. In phase 1, patients were randomized to standard care in the ED versus syncope unit — which was equipped with 24-hr telemetry monitoring, TTE, orthostatic BP qhr, and tilt-table testing. The latter group had a higher diagnosis rate (meaning diagnosis for the etiology of syncope). At two years, nearly all metrics measured between the two groups were equal — namely, probability of survival at 2 years, recurrent syncope at 2 years, and probability of being free of a syncopal event. The authors conclude that a dedicated syncope observation unit leads to decreased hospital and patient costs and shortened stays, while not affecting all-cause mortality or recurrent syncope.

2) Randomized clinical trial of an emergency department observation syncope protocol versus routine inpatient admission. (Ann of Emerg Med, 2014)

This randomized control trial of 124 patients across 5 EDs compared an

ED observation protocol to inpatient admission for adults over 50 presenting with syncope. The observation unit led to shorter hospital stays (29 versus 47 hours) and serious outcomes were equivalent at both 3 month and 6 months. Zooming in on patient-centered outcomes, there was no significant difference found in patient satisfaction between the two groups. Notably, this paper estimates an average cost for syncope hospitalization at approximately $2420, compared to a mean cost of EDOU observation status of $1400.

3) Comparison of 1-Day Emergency Department Observation and Inpatient Ward for 1-Day Admissions in Syncope Patients (J Emerg Med, 2016)

Several texts presented above provide evidence for the benefits of EDOU admissions for syncope — notably cost savings and equivalent patient centered outcomes. This study, however, sheds light on a metric that may correlate with patient satisfaction, presuming that patient satisfaction correlates with having a formal diagnosis at time of discharge.

This retrospective cohort study analyzed 351 ED patients admitted to either the hospital or the EDOU for syncope workup. Importantly, none of the EDOU patients had serious etiologies of syncope compared to 38% of admitted inpatients (who tended to be older with more comorbidities, and thus more likely to have serious etiologies of syncope.) Additionally, EDOU patients were much more likely to be discharged without a diagnosis. In this regard, this study points to a potential flaw of EDOU observation workups for syncope, in that patients may be less likely to receive a formal etiology of their syncope compared to their admitted counterparts.

4) Role of emergency department observation units in the management of patients with unexplained syncope: a critical review and meta-analysis (Clin Exp Emerg Med, 2017)

This systematic review of EDOU for syncope analyzed a total of six studies — four observational and two RCTs. The authors make a point to comment favorably on the SEEDS trial, included in their analysis and outlined above, for this trial demonstrated that a dedicated syncope workup unit in the ED led to a higher diagnosis rate than hospitalization. The authors comment on two of the main drawbacks to current literature on this subject that require a closer look — namely, who qualifies as 'intermediate risk' syncope and would thus benefit from an observational stay as opposed to a full hospitalization, and what qualifies a syncope observational unit (as tilt-table testing is not practical nor cost effective for most hospital systems). Similar to most of the individual studies analyzed, little weight is placed on patient-centered metrics such as patient satisfaction.

Procedures List in Syncope Observation Study

Procedure	APC code	APC cost
Insertion of heart pacemaker	00089	7811.77
Echocardiogram	00269	402.39
Implantable defibrillator placement	00107	23404.61
Intracoronary stent placement	00104	5655.53
Coronary angiography	00080	2726.85
Carotid artery duplex/ ultrasound	00267	152.99
Troponin	84484	18.72
Chest x-ray	00260	45.04
Holter monitor	00097	66.25
Tilt-table evaluation	00101	293.90
Cardiovascular stress test	00100	178.42
Perfusion-ventilated chest scan	00378	319.92
CT of brain/cervical spine/ face/pelvis	00332	193.85
MRI of brain	70551	342.93
Electroencephalogram	00213	166.64
Lower extremity venous ultrasound	00266	96.28
Electrocardiogram	00099	27.26
Venipuncture	36415	3.00
Complete blood count test	85025	14.79
Metabolic panel	80053	20.10
Cardioversion	00679	371.97
Chest CT angiogram	00662	338.53
Observation unit visit	08002	714.33

From supplemental material for Study 2: various costs of syncope work up related procedures.

Summary of Literature

There have been limited high-quality papers investigating the role of EDOUs for syncope, but the evidence suggests that they lead to lower healthcare costs and shorter hospital stays. In addition, long-term serious outcomes do not appear significantly different between those admitted to hospital versus observation units.

Looking Forward/What's Next

It is unclear whether observation units have as much diagnostic value as a full hospitalization in the differentiation of syncope, and this is likely based, in large part, on the degree of complex testing available to the observation unit. There is little evidence in the literature evaluating patient satisfaction in observation stays versus full hospital stays. Further research should emphasize bounce-backs, or repeat visits, between those admitted to a full hospitalization versus syncope observation unit. Nevertheless, this review demonstrates the utility of observation units, for syncope but also much more broadly a diverse array of chief complaints, in reducing both patient and hospital cost and achieving shorter length of stays.

Looking ahead, syncope risk stratification tools may further aid emergency providers in determining disposition for patients who present with syncope. A recent systematic review of nine syncope risk stratification tools found the Canadian Syncope Risk Score to have statistically significant high likelihood ratios. See references for this paper to check out the Canadian Syncope Risk Score.

EMRA Administration & Operations Committee Links, Guides and Opportunities

- Check out what's happening with our group at the EMRA A&O landing page.
- Looking to gain a mastery of this and other relevant operational knowledge? Consider applying for EMRA's ED Directors Academy scholarship. ✶

References available online

EMRA as the MVP

The regulatory acronym soup can be difficult to grasp; EMRA opportunities can help it make sense.

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Times are changing for EM health policy and reimbursement with the recent shift toward value-based care and away from fee-for-service models. Currently, the Centers for Medicare & Medicaid Services (CMS) uses the Merit-based Incentive Payment System (MIPS) as the program to evaluate clinicians on quality, cost, improvement activities, and promoting interoperability.

Dependent on performance, these evaluations and scoring on quality measures are used to alter Medicare Part B reimbursements to clinicians by up to 9%. Stakeholders, including physician groups, have noted significant limitations in the MIPS due to the quality measures not being particularly meaningful to individual specialties. As a result, CMS has developed the MIPS Value Pathway (MVP) framework to be implemented in performance year 2022 with aims to:[1]

- Develop connected meaningful measures for clinicians
- Include measures that are valuable to patients and caregivers
- Include the patient voice
- Reduce barriers to alternative payment model (APM) participation
- Support the transition to digital quality measures.

This acronym soup can quickly become dizzying and difficult to follow, but opportunities through EMRA helped form the foundation of my understanding on these topics and other programs that will inevitably impact our specialty significantly in the coming years.

When in residency, I was fortunate enough to be selected for the EMRA Emergency Medicine Basic Research Skills (EMBRS) Scholarship as well as the EMRA/ACEP Resident-Fellow Health Policy Award in Washington, D.C. Respectively, these opportunities provided me a fundamental understanding of research techniques as well as a "learning of the language" needed.

Recently, as a fellow, I was nominated and selected to lead the ACEP MVP Task Force, assembled to develop an EM-specific MVP that would include meaningful quality measures that EM clinicians should be judged and reimbursed on.

Using those developed skills, the Task Force rapidly gathered feedback from a wide range of clinicians within ACEP and implemented findings into a candidate MVP. While still under review with CMS, the goal of the MVP is to focus on the "bread and butter" complaints we see daily — those being the high-risk undifferentiated complaints of chest pain, abdominal pain, headache, and back pain. Including available quality measures around those complaints will allow EM clinicians to report on measures that are meaningful, while also offering opportunities to identify areas of practice variation ready for improvement.

Lessons Learned

From these unique experiences I've learned a few lessons that I think are translatable to many EM residents trying to find their way and identify their subject niche.

First: If you don't shoot, you can't score.

While I was fortunate to be selected for the opportunities listed above, I also have applied for and *not* been selected for significantly more, including EMRA Health Policy Director. That's OK, other doors will open as a result.

Second: Say yes to opportunities early in your training.

You might have a bit of impostor syndrome, but you likely have more time to sink your teeth in and drive a project forward in comparison to several other uber-busy folks.

Third: Do your homework and be prepared.

You only get one first impression. Understand what has been written on the topic at hand and by whom.

Finally, and potentially most important: Don't reinvent the wheel.

Be strategic and identify those people you respect, and look at their path through CVs, leadership, publications, and service to see how that journey might be adapted to meet your goals. To have impact, don't just think 1 year in advance, but instead think about where you want to be in 10-15 years, and be intentional about your actions today. ✶

Speaking Up for Speaking Plainly
Why EM Physicians Should Care About Health Literacy

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You're in the middle of a busy ED shift when a new patient arrives in bed 7. Her breathing is labored and her lungs are riddled with crackles. She tells you she was diagnosed with "a heart problem" a few months ago. She recently ran out of her medications and did not refill them because she "wasn't sure what they were doing anyway." You ask if she sees a cardiologist. "A what?" she responds.

As emergency clinicians, we encounter scenarios like this one more often than we might hope. At times it is easy to label patients as "noncompliant" and assign them blame for their conditions, but providing compassionate and equitable care requires us to analyze the underlying cause. We must consider what circumstances precipitated this presentation. What can be done differently this time to help prevent a repeat scenario? Many underlying issues stem from flaws in the healthcare system that we cannot individually fix, but optimizing communication and promoting health literacy is something that we *can* easily do.

Health literacy is defined by the U.S. Department of Health and Human Services as the ability to access and understand basic health information and services needed to make appropriate health decisions.[1] A national survey performed in 2003 revealed that over one third of US adults have basic or below-basic health literacy.[2] Health literacy has gained recognition in recent decades for its association with a multitude of health outcomes. In the US, low health literacy is associated with higher rates of hospitalization and emergency care use, greater healthcare costs, less frequent use of preventive services such as screenings and vaccines, improper medication use, and greater morbidity and mortality.[3-6] It is important to note that low health literacy disproportionately affects individuals of minority, low-income, and low-education groups.[2] These groups, often impacted by other social determinants of health, are at further risk of health inequities when additionally affected by low health literacy.

While health lit eracy was traditionally thought of as an individual trait, the definition has evolved over recent years to incorporate the role of clinicians and healthcare systems. Health literacy is now described as occurring when society and its healthcare system "provide accurate health information and services that people can easily find, understand, and use to inform their decisions and actions."[7] **Emergency physicians are equipped to play an important role in addressing and promoting health literacy.** We often take care of patients who struggle with proper medication use, demonstrate limited understanding of their conditions, or are unable to access appropriate health services. We have the ability to provide clear and helpful information in order to enhance patient understanding and increase health potential — or we can deepen a patient's confusion.

The COVID-19 pandemic illuminated the detrimental power of misunderstanding health information. As the virus swept through society, so too did misinformation, disinformation, and confusion. Uncertainty regarding how and when to wear masks, the importance of social distancing, and vaccine safety overwhelmed communities and social media. One thing was plainly evident: clear and effective communication plays a critical part in societal health literacy. Equally important to quality information is trust. Much of the confusion surrounding the pandemic resulted from a lack of adequate information, but other qualms were deeply rooted in well-founded mistrust of the healthcare system. Building public trust is essential in mounting an effective response to this global crisis. Likewise, our patients may be more willing to share their lack of understanding and ask questions when the patient-physician relationship is founded on trust. It is well worth our while to dedicate a few minutes to listening to patients, addressing any gaps in understanding, and engaging in patient-centered communication.

Best Practices for Communication

Quality communication is important for both patient care decisions in the ED and patient self-care after discharge. Emergency physicians are often pressed for time, and may not have the ability to engage in lengthy conversations with patients or review discharge instructions in great detail. But practicing simple communication skills that promote health literacy is an efficient and effective way to demonstrate empathy, increase patient understanding, and improve patient outcomes. Best practices for communication include avoiding medical jargon, demonstrating concepts when possible, distilling information into key points, writing clear instructions, utilizing the teach-back method, and inviting patient questions.

Plain language is terminology that anyone can understand upon first use, which requires avoidance of medical jargon. Even terms that seem simple, such as "contraceptives," can be confusing for patients and should be replaced with clearer alternatives like "birth control." Discussion of self-care measures can also be simplified through the use of demonstration. Instead of using words or

HEALTH LITERACY COMMUNICATION PRACTICES

USE PLAIN LANGUAGE

Plain language is communication all patients can understand the first time they hear it. Avoid technical jargon!

DISTILL INFORMATION

Emphasize the top 1-3 things your patient should know about his/her condition and its management.

GET VISUAL

Demonstrate how to use a medication or draw a picture to explain a concept — simpler is better!

WRITE CLEAR INSTRUCTIONS

Ensure that discharge instructions are easy to read and clear. Explicitly highlight any follow-up measures.

USE TEACH-BACK

Check that you have communicated clearly by asking your patient to repeat back what he/she understands.

INVITE QUESTIONS

Ask "What questions do you have?" instead of "Do you have any questions?"

written instructions to describe how to use an inhaler, demonstrate proper technique.

Another method of maximizing the amount of information patients walk away with is distilling material into a limited number of high-importance points. The sheer volume of new information often relayed to patients during a hospital visit can be overwhelming beyond the point of comprehension. Focusing on 3-5 key concepts and next steps increases the likelihood of retention. It is helpful to highlight these in discharge instructions as well, which should be written in clear and concise language.

Use of the teach-back method and an invitation for questions are ideal ways to close a patient encounter. The teach-back model is a means of ensuring patient understanding. It employs a cycle of relaying information to the patient, asking her to repeat back what she understood, and clarifying any missed points until mutual understanding is achieved. It should be emphasized that teach-back is used to ensure clear communication by the provider, not to measure patient intelligence. This can be illustrated to the patient by introducing the concept with an opening along the lines of, "*To make sure that I have done a good job explaining everything to you, can you tell me what you understand about…*" Nursing staff typically receive more education on teach-back than physicians, and may be able to assist with this.

Finally, questions should be elicited from patients in an open-ended and judgement-free manner. Instead of the potentially intimidating "*Do you have any questions?*", try "*I know that you've been through a lot today; what questions do you have for me?*" This demonstrates the *expectation* for questions and makes patients feel more comfortable asking them.

We can now apply some of these communication principles to our patient in bed 7. When talking with her, we can avoid jargon and explain her condition in simple terms. Instead of asking about her cardiologist, we can use the term "heart doctor." We can end the encounter with emphasizing the key 3 things she needs to do to manage her condition, and use teach-back to ensure her understanding. Our discharge instructions should provide easily digestible information, as well as resources when available. Questions should be elicited in an inviting manner and fully addressed. Our patient will leave the ED with a better understanding of her disease and how to manage it thanks to our patient-centered communication that promoted health literacy. ✶

TAKE-HOME POINTS

Care that does not prioritize patient understanding is suboptimal care. We cannot place the onus solely on our patients to understand everything we spent years in school learning. It is our responsibility to communicate clearly in a manner that promotes comprehension and addresses patient questions and needs. We do our patients a disservice when we do not take the time to properly explain a diagnosis or appropriate management strategies.

On the flip side, we have the ability to use simple communication techniques to make a real impact on patient understanding. Use some proven practices:

- Avoid jargon
- Distill information
- Use teach-back
- Invite questions

Promoting health literacy not only reduces healthcare costs and otherwise avoidable overutilization of the ED, but also empowers patients and improves health outcomes. Knowledge is power, and health literacy is a vehicle for patient empowerment that allows individuals to play a more active role in their own health. Ensuring understanding is one simple way emergency physicians can work toward quality care and health equity for all.

25 UNDER 45

INFLUENCERS IN EMERGENCY MEDICINE

Our **25 Under 45** campaign recognizes 25 young emergency physicians who are making big changes in the world. We are excited to continue celebrating young influencers who shape the future of their communities, hospitals, and our specialty. For full details and nomination instructions, visit us online. **Nominations due July 15.**

EMRA
25 Under 45

EMRA Awards

EMRA AWARDS

Do you have the pleasure of working with a rock star peer in the emergency department? Know someone who has demonstrated exceptional leadership skills? Nominate them for an EMRA Award! Several opportunities are available to recognize outstanding individuals, and self-nominations are encouraged, too!

Nominations due July 15.

Faculty Teaching Excellence Award

Steve Tantama, MD Military Excellence Award

Faculty Mentor of the Year Award

Joseph F. Waeckerle, MD, FACEP Alumni of the Year Award

Augustine D'Orta Humanism Award

Clinical Excellence Award

FOAM(er) of the Year

CORD Academic Assembly Travel Scholarship

LAC Travel Scholarship

EMRA / ACEP EDDA Travel Scholarship

EMRA / EDPMA Scholarship

EMRA / EDPMA Fellowship

Be the Change Project Grant

EMRA Simulation Research Grant

EMRA / ACEP Resident Fellow Health Policy Elective in Washington, DC

EMRA / ACEP Medical Student Elective in Health Policy

https://www.emra.org/be-involved/awards

EMRA at ACEP21

EMRA programming will be held at the Omni Seaport, across the street from the Boston Convention & Exhibition Center. Registration opens in early Summer 2021; registration for ACEP21 is **not required** to attend EMRA programming, but it is encouraged.

Sunday, October 24

11:00 am – 12:30 pm	EMRA Resolution Review & Public Hearing
5:00 pm – 7:30 pm	EMRA/ACEP Leadership Academy (by invitation only)
7:30 pm – 8:30 pm	EMRA Awards Ceremony (virtual)

Monday, October 25

1:30 pm – 6:30 pm	Case-Con Residents
1:30 pm – 6:30 pm	Case-Con Medical Students
1:30 pm – 3:00 pm	EMRA Committee Programming
3:15 pm – 4:45 pm	EMRA Committee Programming
5:00 pm – 6:30 pm	EMRA Committee Programming
6:00 pm – 8:00 pm	EMRA Job & Fellowship Fair (Boston Convention & Exhibition Center)

Tuesday, October 26

8:00 am – 9:00 am	EMRA Rep Council Registration
8:00 am – 9:00 am	EMRA Rep Council Welcome Breakfast & Candidate's Forum
9:00 am – 2:30 pm	EMRA Rep Council and Town Hall Meeting
10:00 am – 4:00 pm	EMRA Resident SIMWars Competition
6:00 pm – 7:30 pm	EMRA 25u45, VIP & Board Alumni Reception

Wednesday, October 27

2:00 pm – 4:00 pm	EMRA 20 in 6 Resident Lecture Competition
6:00 pm – 8:00 pm	EMRA Airway Stories

Thursday, October 28

8:00 am – 6:00 pm	EMRA MedWAR (site to be announced)

Visit **https://www.emra.org/be-involved/events--activities/acep** for registration updates.
All times listed are Eastern.

EMRA 20in6 RESIDENT LECTURE COMPETITION

#EMRA20in6 #EMRAatACEP21

We seek out the best resident speakers in the country to compete for the title "**Best Resident Lecturer.**" Residents are given up to 6 minutes and exactly 20 slides to lecture on any topic relevant to emergency medicine. It's designed to be a fast-paced, intellectually stimulating event in a fun, EMRA-style atmosphere.

Entries are now being accepted at emra.org/20in6.

Proposal Deadline
July 31, 2021

Selection Notice
Aug. 1, 2021

WEDNESDAY, OCT. 27 @ ACEP21
LIVE FROM BOSTON, MA

MONDAY, OCT. 25 @ ACEP21 | BOSTON, MA

Case-con

Medical Student and Resident Case Study Contest

EMRA Case-Con is a poster presentation contest featuring fascinating emergency medicine cases. The event includes a 5-minute presentation followed by 2 minutes of group discussion. Presentations will be judged by a panel of EM residents and faculty.

Students and residents are invited to compete! 3 winners will be selected from each category.

#EMRAcasecon #EMRAatACEP21

Entries are now being accepted at emra.org/case-con.

Abstract Submission Deadline
July 5, 2021

Selection Notice
Aug. 3, 2021

Poster Design Deadline
Oct. 4, 2021

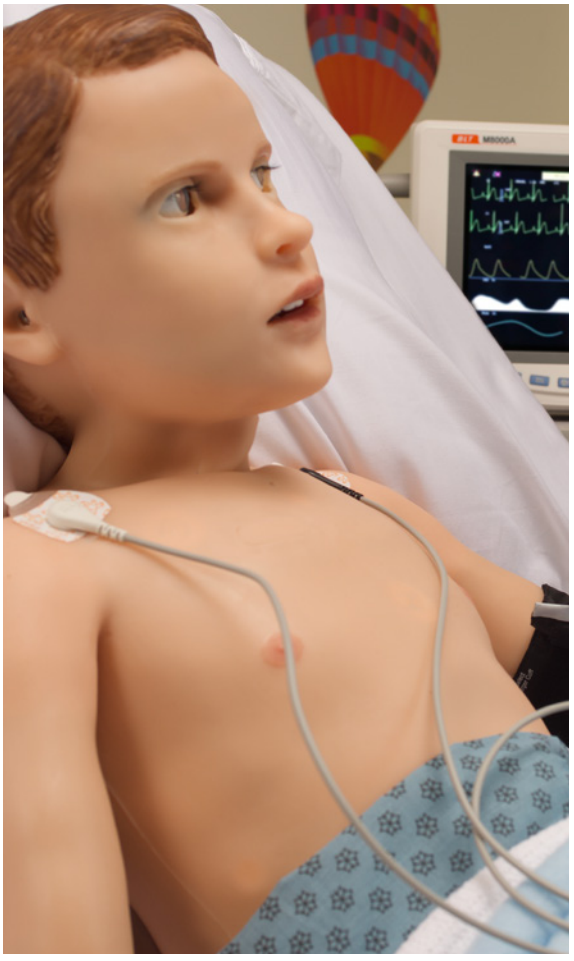
THURSDAY, OCT. 28 @ ACEP21

LIVE FROM BOSTON, MA | SITE TBA

EMRA MEDWAR

Get details and deadlines at **emra.org/medwar**.

#EMRAmedwar #EMRAatACEP21



TUESDAY, OCT. 26 @ ACEP21

LIVE FROM BOSTON, MA

Team entries are now being accepted at emra.org/simwars.

Team Submission Deadline
July 9, 2021

Selection Notice
July 15, 2021

EMRA Resident SIMWARS

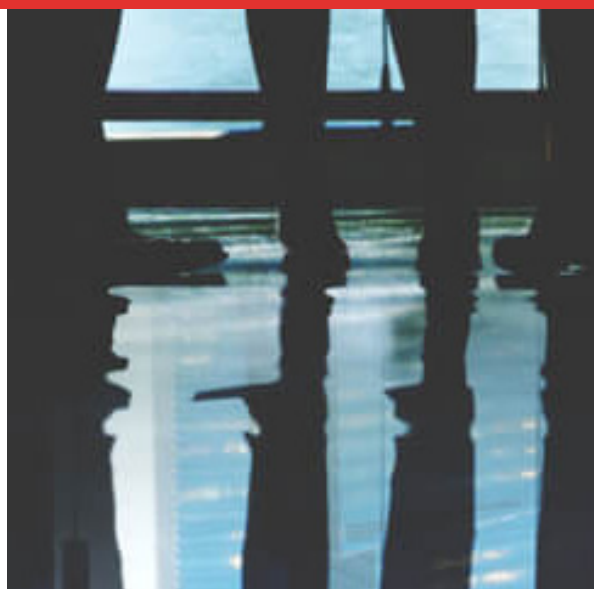
#EMRAsimwars #EMRAatACEP21

SAVE THE DATE!

EMRA's **Job & Fellowship Fair** will be held in-person at ACEP21 on Monday, Oct. 25. Meet with recruiters and programs from across the country, and get ready to network with leading emergency medicine employers.

EMRA JOB & FELLOWSHIP FAIR

Monday, Oct. 25 at ACEP21 | 6:00 - 8:00 pm Eastern
Boston Convention & Exhibition Center



https://www.emra.org/be-involved/events--activities/acep/

EMRA RESIDENCY FAIR

Aug. 14 — 19 | VIRTUAL

We are excited to provide you with a virtual experience that personally connects you to 160+ emergency medicine residency programs across the country.

To take care of members preparing for the 2022 cycle, MSIVs will have first access. As space allows, we will open registration to all medical student members.

Our virtual platform provides:

- Customizable virtual profile
- Residency program booths organized in geographical regions
- Ability to chat with up to 5 program representatives from each program
- Opportunity to schedule appointments

EMRA
MEDICAL STUDENT
COUNCIL

VIRTUAL MEDICAL STUDENT FORUM

Saturday, Aug . 14 | 9:30 am - 12:30 pm Central

The EMRA Medical Student Forum brings together program directors and faculty to answer questions specific to transitioning to residency. General sessions answered big-picture topics, and breakout sessions per year to target exactly where you are in your training.

EMRA Virtual Residency Fair

EMRA Medical Student Forum

Note: EMRA Releases 6 New Publications

MRA has released a bevy of updated clinical guides and brand-new publications, all designed to help you become the best doctor you can be.

These new publications will be incorporated into the appropriate EMRA member kits this summer, with app versions to follow in MobilEM later this year.

None of these valuable guides would be possible without the care and attention of the content teams. Hundreds of emergency physicians have dedicated countless hours to produce evidence-based guidance you can count on at the bedside. ✶

Special Thanks to the Editors of these New EMRA Guides

Emergency ECGs: Jeremy Berberian, MD; William J. Brady, MD; and Amal Mattu, MD

Basics of EM, 4th ed.: Joseph Habboushe, MD, MBA, and Eric Steinberg, DO, MEHP

Basics of EM: Pediatrics, 3rd ed: Joseph Habboushe, MD, MBA, and Eric Steinberg, DO, MEHP, with C. Anthoney Lim, MD, MS, and Jeranil Nunez, MD

EM Fundamentals, 2nd ed: Laura Welsh, MD, and the Boston Medical Center EM Residency

PEM Fundamentals: Cindy D. Chang, Patricia Padlipsky, MD, and Kelly D. Young, MD, MS, with Los Angeles County Harbor-UCLA Medical Center

Urgent Care Guide: Brian J. Levine, MD, FACEP, and Lori Felker, DHSc, PA-C. ✶

ABEM Sets Virtual Oral Exam Dates for Fall 2021

The American Board of Emergency Medicine has announced the 2021 oral exam dates, which will be held virtually:

Fall 2021 Exam Dates*

September 10-13, 2021

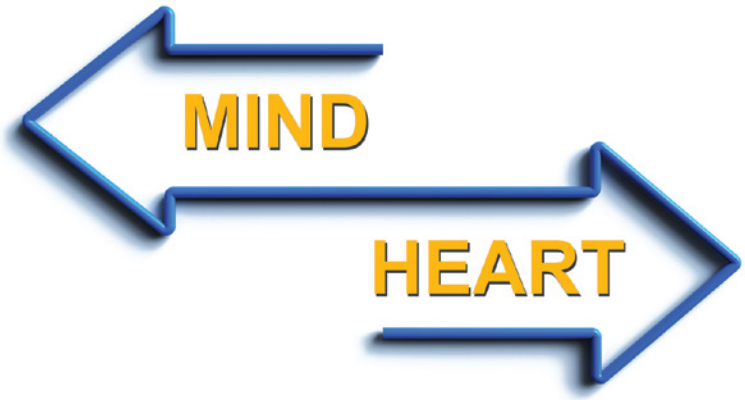
October 5-10, 2021 December 8-11, 2021

**Exam dates are tentative and subject to change*

Candidates who were scheduled for the postponed 2020 exams will be the first assigned to the 2021 administrations. ABEM is working to confirm logistics for these exams.

ABEM values the Oral Exam as part of the process to become ABEM certified and has invested in pivoting our delivery to meet physician needs. We recognize the frustration that EM physicians are experiencing due to COVID-19. Please know we are focused on providing all eligible physicians the opportunity to become certified as soon as possible.

The ABEM Board is committed to providing the opportunity to take the Oral Certification Exam in 2021 to all who were affected by the postponed exams in 2020. ✶



The Hidden Curriculum

Joy McLaughlin
Florida Atlantic University Charles E. Schmidt College of Medicine
Class of 2022

The school talks to us about "the hidden curriculum;"
The only subject they can't teach and only act as adminiculum.
When some of us realize that the shiny apple we have bitten is rotting at the core
And what we learn about ourselves is more than we've bargained for.

A patient with a still born (still waiting to be born)
Tests COVID positive and I'm not permitted to scrub and see this rare case through
I'm not the same person that I thought I knew
I'm more concerned with the opportunity cost
Than the whole new life that this patient has lost.

Another patient, much younger than me
Comes in with a tender, swollen knee
She's a champion athlete
Finally able to compete
After nearly a year since her MCL repair,
But we both worry it's another tear.
While I'm evaluated, I palpate patella,
Tendons, pulses, and lamella,
She's gracious as I fumble through
But that was hours before she knew—
"A thorough history, a pertinent exam"
Writes my attending, but I feel a sham,
Imaging reveals a shattered bone
The next time she'll compete is now unknown
We give her time to process this result
And lamely wait for the ortho consult.
And yet I'm determined to find a way
To more often be the person that I was that day,
And twice a day 'till she was d/c'd
Every shift I'd check, every note I'd read
She wasn't my patient after her admission
I'm an EM student; she was in stable condition.
But I know what it's like to want something so much
And how tightly to our dreams we clutch.

We are all heroes but occasionally villains
Armed with our penems and penicillins
But exhausted by the journey that it took to get here
And tackling our own doubt, debt, and fear.
The hidden curriculum reveals the chiasm between
What is ideal and real in the medical machine
But I was told "the more urgent the condition, the slower you should go"
Leave room for the human among everything you try to know. ✶

Lessons in Vulnerability

Diana Halloran, MD
Northwestern University

It is hard enough to be an intern, let alone an intern in the middle of a global pandemic. In addition to learning how to diagnose, treat, perform procedures, and identify critically ill patients, we learned how to cope with constantly witnessing the most brutal and honest human experiences: emotional trauma, sadness, pain. And while I might have anticipated this emotional burden, what I didn't anticipate — or train for — was the resultant feelings of futility.

I was an intern resident physician in Chicago in 2020. My last few months of medical school were full of uncertainty — my canceled ICU rotation, weekly university-wide coronavirus updates, watching the numbers of available hospital beds dwindle nationwide. Like thousands of 2020 graduates, I celebrated 4 years of medical training over Zoom.

Everything changed on July 1, 2020, when I walked into the emergency department for the first time as a physician and immediately entered the fray: My very first patient was COVID positive. The nurse and I donned our yellow disposable gowns, goggles, gloves, orange N95s, and we entered the room. I stumbled through introducing myself — saying "*My name is Dr. Halloran and I'll be one of the people taking care of you today*" for the first time, barely convincing myself or the patient.

Every day since then I have seen the heart-wrenching emotional cost of this pandemic and medicine as a whole. A patient with COVID having difficulty getting enough oxygen into her body, listening to her husband tell her he loves her right before I insert a tube into her lungs to help her breathe. The wife of another patient with COVID pulls me aside outside her husband's room – he has a weak immune system, and she is devastated that her work as a home health nurse might have exposed him to the virus. There are stories of hospital staff treating their own critically ill co-workers. These were daily occurrences, and they have taken their toll on us.

Along with seeing so much sadness these past months, every day I wonder — did I matter? Did I make a difference? Did my work help this patient and their family?

I spend my days providing care, helping where I can, listening to fears and concerns. I become more confident with procedures I never thought I could be as a medical student. I learn so much every day yet still feel that I will never know enough to be competent and confident. During my intensive care unit night shifts I have to pronounce a death for the first time for my patient who had listed her code status as DNR. Her nurse sat with her and held her hand while she died alone in her room as my senior resident and I attended a code across the unit. The nurse and my senior resident help me go through the steps, listening to her now silent heart and lungs, shining a light into her open, unseeing eyes, until it is time to pronounce. We leave her room, and my senior asks how I am doing as I swallow back tears. I go down to the emergency department to see my co-residents, who tell me their stories of their first death exams. I feel understood — they know exactly how I'm feeling, they've been here before. I cry in the elevator, then tamp down my sadness and go back to the ICU to call the patient's husband.

Months later, after work I continue to see people walking outside without masks, I hear my neighbors having another party. During my commute to the hospital one day I catch faint snippets of a stranger's conversation about how "we sure did overreact to this COVID business, didn't we?" A flash of anger stops me in my tracks. I think of our full ICUs and my COVID patient mere hours earlier who needed a ventilator. I think about all of the death I have seen, of the families I have called to tell them their loved one has died. Of the many death exams I have now performed. For another moment I wonder if what I am doing matters.

But I get to the hospital, put on my N95, and I am reminded of the difference we *do* make. Of the connections that we forge with patients and their families, not only during times of their greatest struggles but also in the quiet moments. It is easy for me to overlook the small differences we make day to day and of the impact we have. Every time I start to lose sight of this meaning, another patient brings it back up to the foreground. An older woman comes to the emergency department for abdominal pain. Our imaging reveals a high likelihood of cancer, so I discuss the findings with her. As we sit together in her room, a small moment of stillness in an otherwise busy emergency department, she tells me about her faith, her husband, and the inner strength that she has.

These moments show me how I feel the most connected to my patients during times of quiet and emotional honesty.

I had been trying to block the ever-growing internal wave of sadness from tragedies witnessed, to bury my feelings in an effort to protect myself, but that effort to protect nearly imprisoned — it left me alone behind an impenetrable wall. How can I connect with patients, empathize with them and their struggles, if I have effectively numbed myself and my emotions?

Through my patients I have learned how to be more open, more empathetic, more understanding. Through these moments of vulnerability, I am reminded that our actions do matter, we do make a difference, and we do not go unnoticed. ✶

Regarding "Failure of Follow-Up: Scrotal Hernia Case Illustrates Healthcare Disparities"

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Dear Editor,

We read with interest the article by Dr. Jason David et al. in the November edition, titled "Failure of Follow-Up: Scrotal Hernia Case Illustrates Healthcare Disparities." We commend the authors for highlighting this unusual case and emphasizing the need for interdisciplinary management in patients who face healthcare disparities. We would like to address the proposed surgical treatment and estimated costs, with insight from the surgeon's perspective.

A large hernia (>10 cm width) with loss of domain (LOD) is categorically a "complex abdominal hernia." Treatment considerations differ substantially when comparing a simple hernia to those with extensive LOD. LOD can generally be understood to describe the significantly abnormal relationship between a hernia sac and the existing abdominopelvic volume.[1] Different sources have expressed the concept of LOD as a ratio of hernia sac volume to abdominal cavity volume or total peritoneal volume. Some surgeons use a cutoff value of 20%, while other authors use up to 30%. It serves as a predictor of operative difficulty and success.[2] This case was a large, though patent, hernia containing several loops of bowel with significant LOD.

Treatment is not simple. In returning the hernia contents to the abdominopelvic cavity and restoring the integrity of the abdominal wall, surgical treatment must be highly individualized and carefully planned. A complex abdominal wall reconstruction (CAWR), consisting often of an abdominal component separation combined with placement of mesh, is typically warranted. This often requires collaboration between general surgery and plastics/reconstructive teams; additionally, postoperative care may require an experienced ICU.[3] The surgical resources for complex mesh repairs are not available at all hospitals.

Potential complications are significant. Our colleagues rightfully bring up the well-known complications of general hernia intervention, including poor wound healing and hernia recurrence. With complex LOD hernias requiring abdominal wall reconstruction, the adverse sequalae can be substantially life-threatening. Reduction of hernia contents into the peritoneal cavity can lead to an abrupt increase in intra-abdominal pressure, reduction of venous return, and decreased diaphragmatic excursion. Reduction in tidal volume, postoperative ileus, and abdominal compartment syndrome are potential postoperative complications. Intensive management is essential, including bladder pressure monitoring, serial abdominal examinations, and consideration of elective mechanical ventilation to monitor peak airway pressure.[1] The overall morbidity and mortality of an elective CAWR can range as high as 66% and 6.7% respectively.[4]

These complications become more prominent with underlying comorbidities. This patient had several conditions that compromised his ability to heal and served as a positive predictor of morbidity and mortality for CAWR:[5] untreated Hepatitis C, HIV, poor nutrition, IVDU, and untreated psychiatric illness.

Arguably, the most important preoperative consideration for patients who present for urgent evaluation of a hernia is the delineation of elective vs emergent/urgent categorization. It is imperative that complicated hernias be repaired in the elective setting if feasible, as the morbidity and mortality rise with emergent interventions**. Prospective studies have shown that the risk for 30-day mortality, reoperation, and readmission increased up to 15-fold after emergency repairs rather than elective repairs.** Mesh repair is widely accepted as superior to tissue repair in large hernias as the recurrence rate decreases by 50%-75%.[6] In comparison to elective surgery, the option of using mesh is less likely in emergency surgery. Bowel resections are significantly more common in emergency hernia operations compared to elective repair.[7,8] This patient's hernia was widely patent, and he had no clinical or laboratory abnormalities to suggest bowel compromise or systemic signs of sepsis. Given this patient had no indication for emergent surgery and the aforementioned factors made him a poor surgical candidate, the safest course of action was treatment of medical comorbidities and repair of his hernia in the elective setting.

The overall predicted costs for surgical intervention for hernias with LOD range between $24,000-$64,000. Increased hospital length of stay, ICU admission, preoperative testing, and treatment of any complications with possible need for reoperation can add $54,000-$100,000+.[8,9] Postoperative reconditioning must also be factored, as frail patients may require physical rehabilitation services to maximize mobility after a CAWR. These expenses represent a major barrier to a patient who is uninsured or underinsured.

Our EM colleagues rightfully bring up concerns that extend beyond this case; namely, the growing use of the ED as the sole source of primary care. The social determinants of health often play a major role in precluding patients from obtaining outpatient surgical evaluation. Delay in management can progress disease from elective to urgent or emergent in nature - while emergency hernia repair typically leads to higher costs and worse outcomes.

We agree that a continuous series of ED visits for chronic, non-emergent surgical illness is not productive for the patient. It is only through the interdisciplinary effort of surgery, social work, primary care, and patients themselves that barriers can be overcome, and meaningful progress made in delivering elective surgery. ✶

References available online

ECG Challenge

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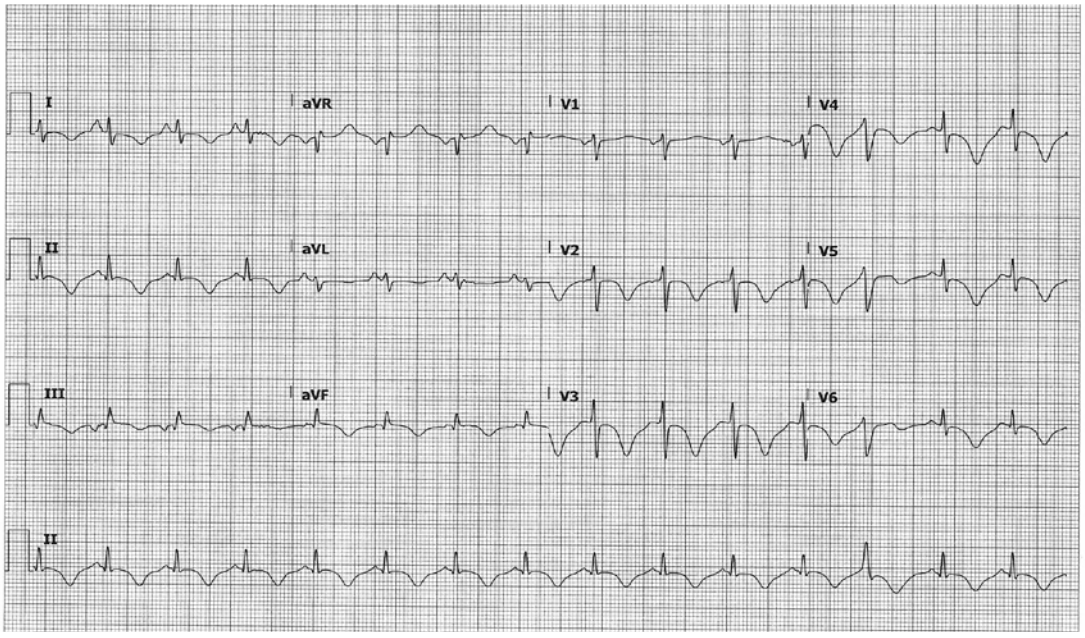
CASE.

A 55-year-old female presents from home due to a brief episode of unresponsiveness and AMS.

What is your interpretation of her ECG?
(Bonus points if you identify the less obvious abnormality.).

See the ANSWER on page 52



ECG Challenge

This ECG shows normal sinus rhythm with a PVC (the 3rd to last beat) at a rate of 89 beats per minute, normal PR interval, normal QRS complex duration, prolonged QTc interval, and deep, wide, symmetric T-wave inversions in leads I, II, aVF and V2-V6. The less obvious abnormality is the presence of left arm-left leg (LA-LL) lead reversal.

Discussion

The T-wave represents ventricular repolarization. In the limb leads, the T-waves should always be upright in leads I and II, and inverted in lead aVR. The T-waves in leads III and aVL can be inverted or upright, and lead aVF will usually have an upright T-wave but may be flat or slightly inverted. In the precordial leads, the T-waves should always be upright in leads V5-V6 and usually upright in leads V1-V4. Inverted T-waves in lead V1 can be a normal variant, as are biphasic T-waves (upright then inverted) in the right precordial leads. In general, the T-wave amplitude is < 6 mm in the limb leads and < 10 mm in the precordial leads.

The differential diagnosis for inverted T-waves includes:

- Acute ischemia (early reciprocal changes)
- Bundle branch blocks
- Cardiomyopathies
- CNS injury
- Digitalis effect
- Intra-abdominal disorders
- Juvenile T-wave pattern
- LVH
- Metabolic abnormalities
- Pericarditis
- Pre-excitation syndromes
- Pulmonary embolism
- Toxicologic abnormalities
- Ventricular paced rhythms
- Wellens' type B

Given this patient's history, it would be reasonable to assume that these T-waves are cerebral T-waves.

This term is used to describe the broad, deep TWI seen with a variety of neurologic pathologies, including stroke, TIA, intracranial hemorrhage, and seizures. Cerebral T-waves are typically seen in the precordial leads and will often have an outward bulge in in the descending limb making them asymmetric. Other common ECG findings seen with CNS etiologies include QTc prolongation, seen in this ECG, and bradycardia.

Not many things cause such broad and deep TWI as seen in this ECG, so it is valuable to review the other possible causes, even if they don't fit the clinical picture.

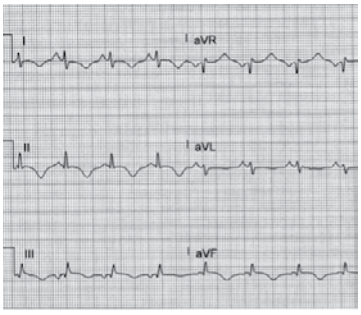
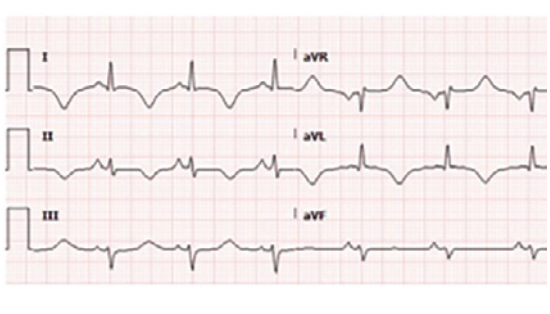
- Takotsubo cardiomyopathy can cause deep TWI and QTc prolongation. It can also present with STE that mimic a STEMI, and it can be difficult to differentiate the two clinically.
- Hypertrophic cardiomyopathy (HCM), more often the apical variant, can cause deep TWI with large R-waves in the precordial leads.
- Wellens' type B will show deep TWI, typically in leads V2-V3, suggestive of a critical stenosis/lesion of the proximal LAD.
- Pulmonary embolism can cause deep TWI in the inferior and right precordial leads.

This ECG shows LA-LL (left arm-left leg) lead reversal. A notable finding in this ECG that should prompt concern for LA-LL lead reversal is the prominent P-wave in lead I when compared to lead II. In general, the P-wave should be more prominent in lead II than lead I in normal sinus rhythm with correct lead placement.

ECG findings seen with LA-LL lead reversal include (see **Figure 1**):

1. Leads I and II "switch places," meaning that the normal findings in lead I are noted in lead II and vice versa
2. Leads aVL and aVF "switch places," so the normal findings in lead aVL are noted in lead aVF and vice versa
3. Lead III is inverted
4. Lead aVR is unchanged

FIGURE 1. Comparison of the limb leads from the case ECG (with LA-RA lead reversal) with a repeat ECG (with correct lead placement)

LA-LL lead reversal (from case ECG)	Correct lead placement (from a repeat ECG)
	

T-WAVE LEARNING POINTS

- Always upright in leads I, II, and V5-V6 and inverted in lead aVR
- TWI can be a normal variant in leads III, V1, and aVL (aVF may be slightly inverted)
- The differential diagnosis for TWI is broad and the workup should be based on clinical picture

Case Conclusion

The patient underwent a broad work up which was notable for a slightly elevated troponin and a normal head CT. A non-emergent cardiac catherization showed clean coronaries and an echocardiogram showed apical hypokinesis diagnostic of Takotsubo cardiomyopathy. It was later discovered that the patient had been undergoing significant emotional stress at home, consistent with her diagnosis of Takotsubo. Note that this was a very atypical presentation for Takotsubo which commonly presents with chest pain. ✶

References available online

Board Review Questions

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1. A 22-year-old man presents with watery, non-bloody diarrhea of 2 days' duration that is associated with cramping, abdominal pain, and nausea. He just returned from a 2-week service project in Mexico. What is the most appropriate management?
   A. Give a single dose of ciprofloxacin 750 mg PO
   B. Prescribe metronidazole 500 mg three times daily for 7 days
   C. Provide reassurance and advice on symptomatic therapy
   D. Send stool for examination for WBCs, ova and parasites, and culture

2. A mother brings in her 30-day-old daughter for difficulty breathing. The baby was born at home at 37 weeks' gestation. The mother received normal prenatal care, and there were no complications during the pregnancy or delivery. The baby only recently returned to her birth weight of 6 pounds. She is formula fed 3 ounces every 2 to 3 hours and is noted to fall asleep during most feeds. The baby is awake and responsive on examination, with a palpable liver edge 2 cm below the costal margin. Her hands and feet are cool and slightly mottled. A 3/4 harsh, holosystolic murmur is noted at the left lower sternal border. Upper and lower extremity blood pressures are symmetric. What is the likely underlying cause of her symptoms?
   A. Coarctation of the aorta
   B. Total anomalous pulmonary venous return
   C. Transposition of the great arteries
   D. Ventricular septal defect

3. Which symptom is reliably found in patients who develop clinical signs of pneumoconiosis?
   A. Acute wheezing
   B. Chest pain
   C. Gradual onset dyspnea
   D. Sputum production

4. Which treatment is contraindicated in the management of an agricultural worker who presents with diaphoresis, fasciculations, hypersalivation, miosis, respiratory distress, and vomiting?
   A. Atropine
   B. Diazepam
   C. Physostigmine
   D. Pralidoxime

5. Which physical examination finding is reassuring when trying to rule out a mandibular fracture in a patient with facial trauma?
   A. The interdental incisor distance at maximal opening is less than 4 cm
   B. The patient can bite down on and break a tongue blade while the examiner twists it
   C. The patient has a sublingual hematoma on the affected side
   D. The patient's chin is deviated toward the affected side ✶

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

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