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This Holiday Season, We're Thankful for Clinicians Like You.

Our stories are tied to each other, and together, we're champions for healthier communities. We turn our appreciation of your efforts into action every day by **investing in clinician wellness, ensuring our teammates have everything they need to thrive** and continue to focus on what matters most: caring for patients.

EDITOR'S FORUM

Running Toward the Fire: Learning to Lead in the Moments That Terrify Us

AUTHORS

Elizabeth Nisper, MD
Editor-in-Chief, EM Resident
Secretary, EMRA Board of Directors
Corewell Health Grand Rapids/
Michigan State University

Every trainee knows the mixed reactions we get when we tell people we're going into emergency medicine. "Wow, good for you! It takes a special kind of person," is something I've heard more than once. I've never asked what they meant, but I like to think they're referring to adaptability, decisiveness, resilience, and justice — the traits that make our field so unique. More often, though, I suspect they mean we're the ones who run toward the very things others run from. The thrill seekers. The people who are calm under pressure and command a room just by our steady presence.

This stereotype made me proud to be entering EM but it also fueled a feeling of imposter syndrome when I started residency and realized that that confidence wasn't always there. In fact, I felt like I was holding my breath most shifts and stepping over the line of fear and discomfort on a daily basis.

An early conversation with my program director helped me see this wasn't just normal — it was expected. Growth in emergency medicine doesn't wait for confidence. It happens when we act anyway: afraid but determined. The weight of responsibility in training at times is almost physical — pressing down like lead, magnifying every doubt. Yet it's at this edge, where fear meets action, that something remarkable happens. We discover that our tools — our knowledge, instincts, and team — are enough to carry us through.

We all have moments in training that we can recall with startling clarity: the first intubation, the first resuscitation, the conversation with a grieving family. For me, it was the first time I had to tell a conference room full of family that their loved one had died. Fear, in that moment, was unavoidable. But so was action. Emergency medicine is defined by these moments — times when we are asked to step into the uncomfortable, to lean into what feels intimidating, and to act despite the flood of self-doubt. Fear exists because we care—about the patient, about doing the right thing, about honoring the trust placed in us. It sharpens our senses and forces us to focus.

I used to believe that confidence was the prerequisite for competence — that one day I'd wake up ready, my nerves finally silenced. Residency has taught me the opposite. Competence is forged in the fire of uncertainty. Confidence grows only after we push through the fear, perform the procedure, run the code, or navigate the difficult conversation. It's only in hindsight, looking back at what we thought we couldn't handle, that we recognize we've become stronger.

The confidence I've gained from those moments in the ED has shaped me not just as a physician, but as a leader, advocate, and friend. When I ran for election to the EMRA board this Fall, I couldn't help but compare my fear and imposter syndrome to what I felt at the start of residency. Standing at the podium before giving my speech, I thought back to that first family conversation in my intern year. I realized I may never feel fully "ready" for moments like these. But by speaking up, even with shaky words, we discover our voice. Fear doesn't disappear when we start talking; it shifts. Now, instead of questioning whether I'm capable, I acknowledge the fear, breathe through it, and then I act anyway. Over time, I've come to see fear less as a barrier and more as a signal: this is where change begins.

That thin space between panic and presence is where we uncover resilience, humility, and courage. It's also where we become the physicians our patients need most. I'll never forget the moment I learned I'd been elected to the EMRA board. It wasn't relief I felt, but transformation. Fear hadn't disappeared — it had carried me across a threshold. It reminded me that the only way to become the physician I aspire to be is to keep stepping into the spaces that scare me most.

The beauty of this specialty is that those moments are plentiful. As long as we're a part of this field, the work — and the growth — are never finished. For me, it's only just beginning, and I intend to embrace every terrifying day. ✷

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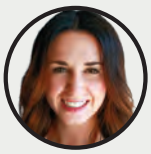

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Interested in serving on the EMRA Board of Directors?

Elections are held each fall. See which positions are up for election this year:
https://www.emra.org/board-election-guide

TABLE OF CONTENTS

References available online.

EM Resident (ISSN 2377-438X) is the quarterly 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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EMRA FY25 Annual Report

Membership: 20,075

90%+
EM residents are EMRA members

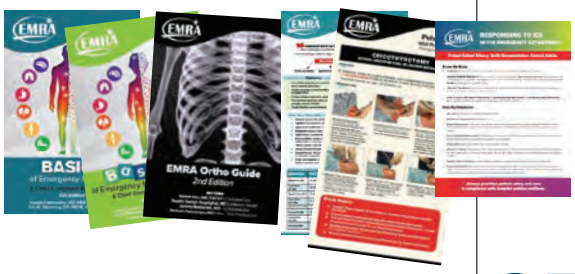
$2.5M+
Annually reinvested into EMRA members

247
EMRAfied residency programs

BEST *DOCTOR*

6 new/newly updated clinical resources

36 *books, reference cards, digital, and mobile resources*



260K+
downloads of EMRA*Cast episodes

18K+ *EM Resident* print distribution

EMRA Match

85K+
monthly page visits to EMRA Match

BEST *LEADER*

22 committees, including NEW groups for Pain Management and Women's Health

38 EMRA Leadership Academy students

25 EMRA Medical Student Council members

24 *representing medical schools from 24 states*

6 EMRA Health Policy Academy fellows

BEST *SPECIALTY*

75+ funded leader travel to ACEP Leadership & Advocacy Conference

6 *Drop the Mic speakers at the EMRA & ACEP YPS Health Policy Primer*

7K+
page visits to review proposed ACGME program requirements

14 resolutions and policy initiatives by the EMRA Representative Council

10+
ongoing collaborations to strengthen EM

EMRA is the voice of emergency medicine physicians-in-training and the future of our specialty.

References available online.

EMRA PRESIDENT

Pauline (Polly) Wiltz, DO

Dr. Polly Wiltz took office as EMRA President during the EMRA Representative Council Fall 2025 meeting. Newly graduated from residency at Case Western Reserve University/University Hospital, Dr. Wiltz brings the power of perspective, experience, and positivity to the office.

WHAT'S YOUR FIRST PRIORITY AS EMRA PRESIDENT?

My first priority as EMRA President is to increase member engagement by creating more accessible, meaningful opportunities for residents and medical students to get involved. EMRA is built on the passion and ideas of its members, and I want to ensure that everyone — regardless of where they train or what stage they're in — feels empowered to contribute. That means breaking down barriers to leadership, promoting mentorship, and making it easier for members to connect with committees, projects, and each other. If we can foster a culture where every member sees a clear path to participation and impact, we'll not only strengthen EMRA today, but also build a stronger foundation for the future leaders of emergency medicine.

WHERE DO YOU WANT EMRA TO BE AT THIS TIME NEXT YEAR?

I want EMRA to be leading the way in redefining how we support residents through education, advocacy, and innovation. On the education front, I hope we've expanded access to high-impact, learner-centered resources that adapt to the real-world needs of emergency medicine trainees — content that is not just informative but truly transformative. In advocacy, I want EMRA to have a stronger presence at both the institutional and national levels, actively shaping policies that protect residents' well-being, fair compensation, and the sustainability of our specialty. And in terms of innovation, I hope we're not just keeping up with change, but driving it — leveraging technology, collaboration, and creative problem-solving to address both the everyday challenges and future threats facing emergency medicine. Ultimately, I want EMRA to be the go-to hub for ideas, support, and progress in the field.

HOW CAN EMRA MAKE A DIFFERENCE TO THE SPECIALTY?

EMRA members can make a difference by recognizing the power they hold — not just as trainees, but as the future leaders of emergency medicine. Whether it's contributing to policy change through advocacy, creating innovative educational tools, or simply supporting fellow residents, every member has something unique to offer. By getting involved in committees, writing for EMRA publications, attending events, or launching new initiatives, members can shape the direction of our specialty. Even small actions — like mentoring a student, speaking up about workplace challenges, or sharing resources with peers — can have a ripple effect. The strength of EMRA lies in its members, and when we come together with purpose and passion, we have the ability to drive real, lasting change in emergency medicine.

HOW WILL YOU EVALUATE YOUR SUCCESS AS EMRA PRESIDENT?

Success as EMRA President will be determined by the impact we make as an organization — both for individual members and the specialty as a whole. There are a couple of different areas where I think we will be able to measure success. First, by how well we've advanced our core priorities: delivering high-quality, accessible education, strengthening resident advocacy at every level, and fostering a culture of innovation and inclusion through leadership. Second, how engaged and empowered our members feel — whether more residents are stepping into leadership roles, contributing ideas, and seeing EMRA as a resource that truly reflects their needs. Success will also mean leaving the organization stronger, more connected, and more forward-thinking than when I started — with

> ***"EMRA is built on the passion and ideas of its members, and I want to ensure that everyone — regardless of where they train or what stage they're in — feels empowered to contribute."***

sustainable initiatives in place that continue to grow beyond my term. Ultimately, if I've helped amplify the voices of residents, improved their experience, and prepared the next generation of leaders to carry the torch, I'll consider that a success.

HOW DO YOU RECHARGE AFTER A TOUGH SHIFT?

After a tough shift, I recharge by giving myself space to decompress — both mentally and physically. Sometimes that means doing something simple like walking my dog, reading a book, or jumping on the phone to debrief what happened with a friend or mentor. Other times, it's about disconnecting completely — spending time with friends or family, hiking through the mountains of my home state, or just getting some much-needed sleep. I do consider naps part of my extra-curricular activities. I also try to reflect on small wins from the shift, even if the day was hard. It helps me stay grounded and remind myself why I chose this path. Ultimately, recharging is about protecting my well-being so I can keep showing up for my patients, my team, and myself.

WHAT IS SOMETHING PEOPLE DON'T KNOW ABOUT YOU?

I used to be a park ranger. If I hadn't pursued medicine, I'd definitely be back to Ranger Polly leading guided hikes in the backcountry of Washington's volcanos.

1 SKILL YOU WANT BUT DON'T HAVE YET:

One of my dreams it to climb all the volcanoes in the Pacific Northwest. I've been an avid hiker for most of my life, but mountain climbing is a whole different beast.

1 SKILL YOU COULD DO WITHOUT:

One skill I could definitely do without is my ability to remember way too many random trivia facts — especially the completely useless ones that pop into my head at the worst times. It's great at trivia nights, but maybe less so when I'm trying to focus on patient care or actually get some sleep. You ever try to fall asleep and "Who Let the Dogs Out" is stuck on repeat, or why the six wives of Henry the 8th are suddenly a top priority in my brain?

References available online.

EMRA PRESIDENT-ELECT

Alexandra Sappington, DO, MS

THE BEST ADVICE ALEXANDRA HAS EVER RECEIVED...

Everything happens for a reason, and you are exactly where you are meant to be in this moment.

Dr. Alexandra Sappington (LSU-New Orleans) has served in leadership roles for ACOEP-RSO, AAEM-RSA, CORD Application Process Improvement Committee, the EMRA Leadership Academy, the AMA-MSS, and a multitude of state and local initiatives. As EMRA President-elect, she has specific goals to support the specialty. In her own words:

"I am passionate about emergency medicine and energized about our future. We have experienced numerous challenges in recent years, and after advocating on Capitol Hill for LAC, I can confirm we are not finished. My experiences with EMRA and ACOEP on the national level allowed me to create mentorship series for medical students, hone my leadership skills, and network with prominent figures in our field. I plan to use the knowledge and insights gained from these experiences to elevate EMRA as it continues to empower our residents to become the best physicians possible and advance emergency medicine as the leading specialty.

"I envision a future where EMRA continues to lead with purpose, and where our members feel connected, supported, and empowered."

WHAT'S YOUR FIRST PRIORITY AS AN EMRA BOARD MEMBER?

Bonding with my fellow board members.

HOW DO YOU RECHARGE AFTER A TOUGH SHIFT?

Working out (I have been a cycle instructor for 7 years now) or going on walks with my dog and my husband.

WHAT IS SOMETHING PEOPLE DON'T KNOW ABOUT YOU?

I went to a performing arts high school in New York City that is well known for its celebrity output.

1 SKILL YOU WANT BUT DON'T HAVE YET:

Fluency in Spanish.

1 SKILL YOU COULD DO WITHOUT:

Being the master of the aux — I bring a speaker to every shift, and if I forget to play music my co-residents let it be known!

DOES PINEAPPLE GO ON PIZZA?

HECK.NO.

FAVORITE COMFORT FOOD/ DRINK:

My Mom's mac and cheese and an old fashioned!

SECRETARY OF THE BOARD
EM RESIDENT EDITOR-IN-CHIEF

Elizabeth Nisper, MD

If you skipped the Editor's Forum, stop right now and read the highly relatable, beautifully encouraging message from our new EM Resident editor-in-chief, Dr. Elizabeth Nisper of Corewell Health in West Michigan. Then come back and get to know her here.

WHAT'S YOUR FIRST PRIORITY AS AN EMRA BOARD MEMBER?

My first priority is to strengthen the diverse voices of EM residents across the country. I hope to provide quick turnaround mentorship to residents interested in publishing, recruit diverse perspectives to write opinion pieces, and feature different programs and all they have to offer.

HOW DO YOU RECHARGE AFTER A TOUGH SHIFT?

Balance, for me, looks different every day. Some days this means decompressing with friends or family, sometimes it means going on a long run, and other times it means re-watching *Gilmore Girls* for the 10th time.

WHAT IS SOMETHING PEOPLE DON'T KNOW ABOUT YOU?

I was a match for a patient on the Be the Match registry during my first year of medical school and got to donate my stem cells!

1 SKILL YOU WANT BUT DON'T HAVE YET:

My husband would say I need to work on my disc golf game...

1 SKILL YOU COULD DO WITHOUT:

Learning to sing my ABCs backwards in elementary school hasn't won me many awards.

DOES PINEAPPLE GO ON PIZZA?

Absolutely!

FAVORITE COMFORT FOOD/ DRINK:

Box mac and cheese, specifically Annie's shells & white cheddar.

THE BEST ADVICE ELIZABETH HAS EVER RECEIVED...

Do it afraid. This is such a simple statement, but it has given me the courage to get through every hard day and shown me that I am capable of more than I think.

VICE SPEAKER

Kailey Jacobson, DO

During medical school, Dr. Kailey Jacobson represented the student voice at the American Medical Association, serving as an EMRA representative to the AMA MSS. Now in residency at Emory University School of Medicine, she will help guide the governing body of EMRA after being elected Vice Speaker of the Council.

WHAT'S YOUR FIRST PRIORITY AS AN EMRA BOARD MEMBER?

My first priority as an EMRA Board member is to work to increase the understanding of and participation in RepCo (Representative Council, comprising one voting delegate from each residency program and the Medical Student Council). It is an incredible way to make change in our profession, and what we do at RepCo launches the priorities of the board throughout the year.

HOW DO YOU RECHARGE AFTER A TOUGH SHIFT?

Usually lay low with my husband, go for a walk with my dog, or grab dinner with friends!

WHAT IS SOMETHING PEOPLE DON'T KNOW ABOUT YOU?

I used to be a professional ballet dancer before going to medical school and spent time across the country and in Romania!

1 SKILL YOU WANT BUT DON'T HAVE YET:

To be proficient in another language.

1 SKILL YOU COULD DO WITHOUT:

Being able to do a cartwheel.

THE BEST ADVICE KAILEY HAS EVER RECEIVED...

If you want to be an effective leader, listen to and accept with humility the feedback that comes from your team.

DIRECTOR OF EDUCATION

Louisa (Lulu) Weindruch, DO

Many of you know Dr. Lulu Weindruch (Baylor University Medical Center) from her extensive service to the EMRA Ultrasound Committee, the Leadership Academy, the Texas College of Emergency Physicians, or one of the many ways she has supported the profession during training. Now let's get to know her as the Director of Education for the EMRA Board.

WHAT'S YOUR FIRST PRIORITY AS AN EMRA BOARD MEMBER?

Help facilitate an open line of communication between the board and our members, especially when it comes to issues that impact our educational experience.

HOW DO YOU RECHARGE AFTER A TOUGH SHIFT?

By hanging out on a patio with my coworkers.

WHAT IS SOMETHING PEOPLE DON'T KNOW ABOUT YOU?

One of my proudest moments was meeting the Jonas Brothers in 2008.

1 SKILL YOU WANT BUT DON'T HAVE YET:

I would love to be an expert gardener one day.

1 SKILL YOU COULD DO WITHOUT:

I can fall asleep anywhere — it's a blessing and a curse.

DOES PINEAPPLE GO ON PIZZA?

Yes, I love salty/sweet combos!

FAVORITE COMFORT FOOD/ DRINK:

Chips and queso with a fountain Diet Coke

THE BEST ADVICE LULU HAS EVER RECEIVED...

Attitude is everything.

Case Report: Arrhythmogenic Right Ventricular Cardiomyopathy Presenting with Inappropriate ICD Discharges

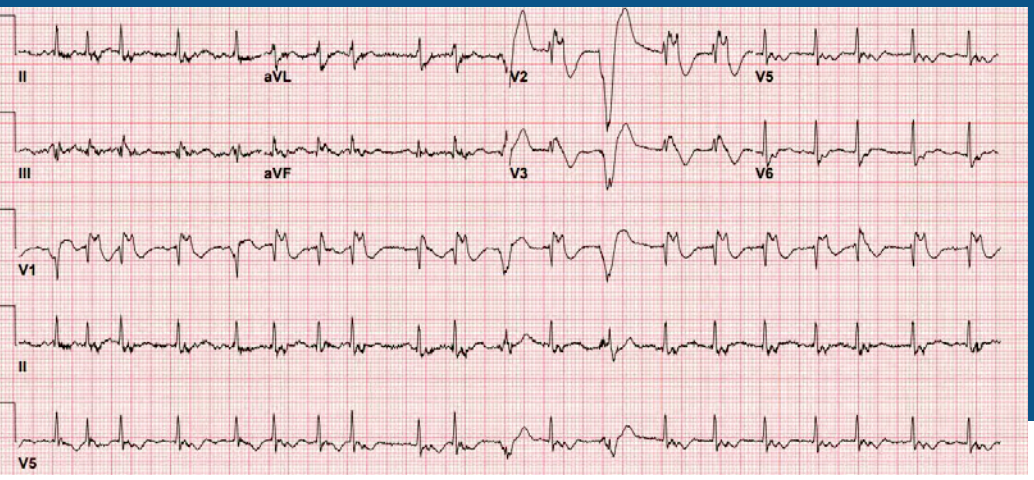


Figure 1. Initial ECG showed atrial fibrillation with RVR and a RBBB.

AUTHORS

Eytan Mendelow, DO
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Daniel Patino-Calle, MD
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We present a case of arrhythmogenic right ventricular cardiomyopathy (ARVC) with inappropriate implantable cardioverter defibrillator (ICD) discharges triggered by atrial fibrillation (AF) with rapid ventricular response (RVR). This case underscores the importance of socioeconomic factors in ARVC management and highlights the role of bedside ultrasound, electrocardiographic findings, and multidisciplinary care in guiding treatment.

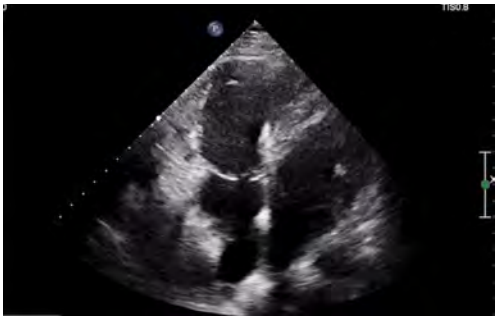
CLINICAL HISTORY

A 38-year-old male with a history of ARVC s/p dual-chamber ICD placement and paroxysmal atrial fibrillation presented to the emergency department (ED) after three ICD discharges. The patient reported acute left-sided chest pain without syncope, shortness of breath, or systemic symptoms beyond occasional cough and palpitations. He acknowledged poor compliance with his antidysrhythmic medication, sotalol, and anticoagulant, apixaban, due to cost and lack of health insurance. On exam, he was normotensive, afebrile, saturating 97% on room air, and with irregularly irregular heart rates in the 120-130s. Initial ECG showed atrial fibrillation with RVR and a RBBB (see Fig 1).

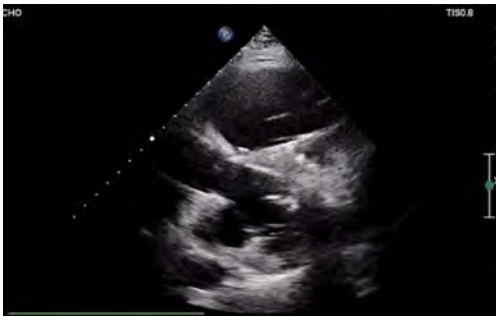
CTA was obtained and showed ground-glass opacities in the right upper lobe suggestive of pneumonia with no pulmonary embolism. POCUS echo showed grossly normal LV systolic function with right ventricular dilation (see Fig 2), and collapsible inferior vena cava (not pictured).

DISCUSSION

ARVC is an inherited cardiomyopathy characterized by replacement of RV myocardium with fibro-fatty tissue which predisposes patients to ventricular dysrhythmias, progressive RV dysfunction, and sudden cardiac death.[1] The ED approach to ARVC necessitates a high index of suspicion, particularly in patients presenting with unexplained syncope, palpitations, or ventricular dysrhythmias. Understanding the underlying pathophysiology, clinical features, and relevant diagnostic tools is critical for timely intervention.



A4C



PSL

Figure 2. Point-of-Care Ultrasound: Apical 4 chamber and parasternal long views show grossly normal left ventricular systolic function and right ventricular (RV) dilation (collapsible inferior vena cava not pictured).

References available online.

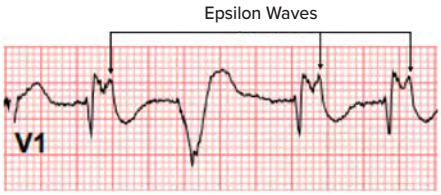


Figure 3. Other EKG features include T wave inversions and QRS complex duration >110 msec in leads V1-V3.[5]

Long-term management involves ICD implantation, antidysrhythmic medications, and lifestyle modifications, all of which can be complicated by socioeconomic barriers such as financial constraints and limited access to healthcare.[2]

DIAGNOSTIC PEARLS

Electrocardiogram: Epsilon wave is the most specific EKG finding in ARVC but is only seen in ~30% of cases. It is characterized by a small deflection at the end of the QRS complex, best seen in leads V1-V2, but may be present in V1-V4. It is caused by pathognomonic myocyte fibrosis leading to delayed depolarization of the RV free wall.[4] The most common ECG finding is a prolonged S-wave upstroke ≥ 55 msec in leads V1-V3. Other EKG features include T wave inversions and QRS complex duration >110 msec in leads V1-V3 (see Fig 3).[5]

Bedside Ultrasound: Ultrasound findings include disproportionate RV dilation, right atrial enlargement, tricuspid regurgitation, and paradoxical septal bowing of the LV (D-sign).[3] In advanced cases, decreased tricuspid annular plane systolic excursion (TAPSE <17mm) suggests RV dysfunction. Regional wall motion abnormalities, particularly akinesia or dyskinesia of the RV free wall due to fibro-fatty infiltration, are a hallmark feature.[4] These findings, when present in the appropriate clinical context, strongly support the suspicion of ARVC.[1]

LABORATORY STUDIES

Laboratory evaluations may be helpful in guiding initial management. An elevated troponin level can indicate myocardial injury resulting from dysrhythmia-induced demand ischemia. Assessing electrolyte levels is crucial, as imbalances such as hypokalemia and hypomagnesemia can exacerbate arrhythmic events. Increased brain natriuretic peptide (BNP) levels, although non-specific, can provide insight into the severity of cardiac impairment. Finally, an elevated lactate concentration may signal decreased systemic perfusion and tissue hypoxia, emphasizing the hemodynamic consequences of ARVC.

RATE AND RHYTHM CONTROL

Patients with ARVC are often adrenergic dependent and are most commonly treated with beta-blockers, in particular sotalol, or amiodarone. Sotalol is a beta-blocker with class III properties that is frequently used in ARVC due to its efficacy in suppressing ventricular dysrhythmias. Other antidysrhythmic medications, including amiodarone and procainamide, can be considered in acute presentations, however, it is important to note that these agents carry a risk of exacerbating dysrhythmias in the fibrofatty RV myocardium. Electrical cardioversion remains first-line for sustained ventricular tachycardia or ventricular fibrillation, while ICD interrogation is critical to deactivate inappropriate shock triggers, optimize device programming, and reduce myocardial injury.[4]

HEMODYNAMIC SUPPORT AND RV FAILURE MANAGEMENT

ARVC-related cardiac dysfunction can precipitate significant hemodynamic instability. Inotropic agents, such as dobutamine or milrinone, may improve RV contractility and reduce afterload, but should be carefully tailored to avoid systemic hypotension and arrhythmogenicity.[1] For those with profound instability, vasopressor support with norepinephrine is recommended. Advanced cases may necessitate mechanical circulatory support, including VA-ECMO (Veno-Arterial Extracorporeal Membrane Oxygenation) or pRVADs (percutaneous Right Ventricular Assist Devices), to provide systemic perfusion and unload the RV. Intravenous fluid administration must be judicious in order to optimize RV preload without causing volume overload which leads to further decompensation. Pulmonary vasodilators, including inhaled nitric oxide, may reduce pulmonary vascular resistance, alleviating right sided heart pressures.

BARRIERS TO CARE IN THE ED

Managing ARVC in the emergency department can be fraught with challenges. The rarity of this condition and its overlap with other arrhythmogenic syndromes can delay diagnosis, especially if key findings like epsilon waves are not present.[4] Definitive care often requires electrophysiology and cardiology input, which may be limited in some settings. Emergency physicians must quickly differentiate ARVC-related dysrhythmias, ensure proper rate control, select appropriate antidysrhythmic medications, and treat underlying conditions under time constraints. Access to inpatient resources can facilitate timely multidisciplinary care and lead to improved patient-centered outcomes.[2]

CASE RESOLUTION

ED management began with assessment of the patient's airway, breathing, and circulation, followed by electrolyte repletion, optimization of volume status, and treatment of his atrial fibrillation with RVR with sotalol.[1] A chest X-ray showed a left lower lobe infiltrate, so treatment with broad-spectrum antibiotics was initiated for pneumonia which was the likely trigger for his uncontrolled atrial fibrillation.

During hospitalization, the patient underwent a multidisciplinary evaluation to address several critical issues. Electrophysiology confirmed inappropriate ICD discharges, leading to device reprogramming and medication adjustment with an emphasis on cost-effectiveness. Cardiology identified RV dysfunction on echocardiography consistent with ARVC, highlighting the importance of strict rate control and anticoagulation.[3] Social work addressed financial barriers by enrolling the patient in an anticoagulation assistance program and coordinating affordable medication access.[2] The patient was discharged on day three in stable condition with plans for follow-up at a safety-net clinic. ✷

References available online.

A Case of Severe Lactic Acidosis Due to Metformin Toxicity

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Metformin toxicity causes a profound lactic acidosis and can be concerning in patients with existing kidney impairment.

CLINICAL HISTORY

A 70-year-old man presented to the emergency department with significant chest and upper abdominal pain. He described the pain as intense pressure and reported he felt short of breath as well. He denied any diarrhea, no rash, and no changes in medication. He reported he had been feeling unwell for some time with some nausea and vomiting, but it worsened on the day of presentation with the new chest pressure and upper abdominal pain. His past medical history is significant for prostate cancer, coronary artery disease requiring stents, diabetes, and prior strokes.

On examination, the patient appeared ill and in acute distress. His skin was pale and capillary refill was delayed. He was tachypneic, but without wheezing or rhonchi. His abdominal exam revealed a soft but diffusely tender abdomen. He had no focal neurologic deficits, and his cardiac exam was benign.

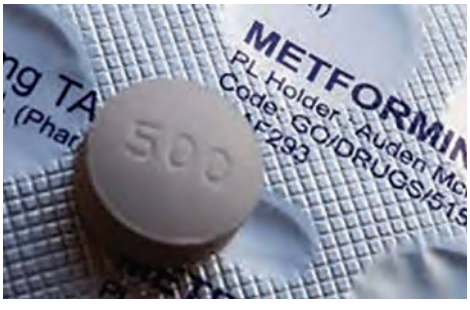
CTA dissection was negative. Initial ED lab work-up revealed a significant metabolic acidosis with an anion gap of 29 and lactate of 7.7, severe acute kidney injury with Creatinine 5.38, beta-hydroxybutyrate >8.0, and elevated lipase of 136. While in the ED, the patient had worsening respiratory distress, and intubation was completed due to respiratory failure. Prior to intubation, he was initiated on bicarb and D5W drips and given 2 cc of push dose epi for lower-than-normal blood pressure as well as 3 amps of bicarb. Following intubation, the patient was admitted to the ICU on a propofol drip for severe metabolic acidosis and to initiate dialysis. While admitted, the patient was found to have an initial presentation due to metformin toxicity.

DISCUSSION

Metformin rarely causes hypoglycemia, but it can cause a profound lactic acidosis in overdose, especially in patients with preexisting renal failure. In healthy individuals, the kidneys can excrete excess lactate in the urine. However, in patients with kidney failure, there is an impaired ability for this function to occur, thus lactate builds up in the body and causes a significant metabolic acidosis.

Acutely worsening kidney function can be due to an underlying cause, such as sepsis, and must be evaluated further. Those with chronic kidney disease are at risk of shock and death in as little as a few hours if the acidosis is not addressed appropriately.

Metabolic acidosis can either fall into the category of non-anion gap or



References available online.

high anion gap metabolic acidosis. This is calculated by subtracting the chloride and bicarb from the serum sodium. An elevated anion gap is defined as AG > 22 and is concerning because many causes are immediately life-threatening, whereas most causes of non-anion gap metabolic acidosis are non-life threatening.

Causes of an elevated anion gap metabolic acidosis (AGMA) can be easily remembered using the mnemonic **MUDPILES CAT:**

- **M**ethanol
- **U**remia
- **D**KA
- **P**araldehyde
- **I**ron/Isoniazid
- **L**actic acidosis
- **E**thanol/Ethylene glycol
- **S**alicylate/ASA
- **C**arbon monoxide
- **A**minoglycosides
- **T**heophylline

Metformin toxicity causes a profound lactic acidosis, placing it among the causes of MUDPILES. Once it has been established that an AGMA exists, further calculations regarding the bicarbonate, delta delta, and if there is appropriate respiratory compensation can be determined as well to aid in management of the condition.

The human body will initially attempt to compensate for the high levels of lactic acid by blowing off excess amounts of carbon dioxide via hyperventilating. As time goes on, the body will tire, causing normal to elevated levels of CO_2 to accumulate and ultimately worsen the acidosis. Poor respiratory effort is a pertinent physical exam finding to be aware of and can be reason itself for intubation. Mechanical ventilation can then be used as an additional tool for metabolic acidosis therapy. Optimal ventilator settings for treating AGMA include a high respiratory rate (RR) and low tidal volumes (TV). The high RR will aid in excretion of CO_2 while the low TV will decrease the potential CO_2 stores within the lungs themselves.

Supportive care and correcting the acidosis are key components to treating the shock and metabolic acidosis caused by metformin toxicity. If a patient presents within 2 hours of ingesting >10 grams of metformin, charcoal can be considered as therapy. The mainstay of treatment for this is vasopressors and, in severe cases, hemodialysis or continuous renal replacement therapy (CRRT) may be required to remove the lactate and metformin from circulation.

Although uncommon, shock in the setting of metformin toxicity can cause refractory shock, in which the severity of vasodilation is unresponsive to fluid resuscitation, high-dose vasopressors, oxygenation, and ventilation. In such cases, AV ECMO has been used as a modality of treatment for extracorporeal removal of toxins.

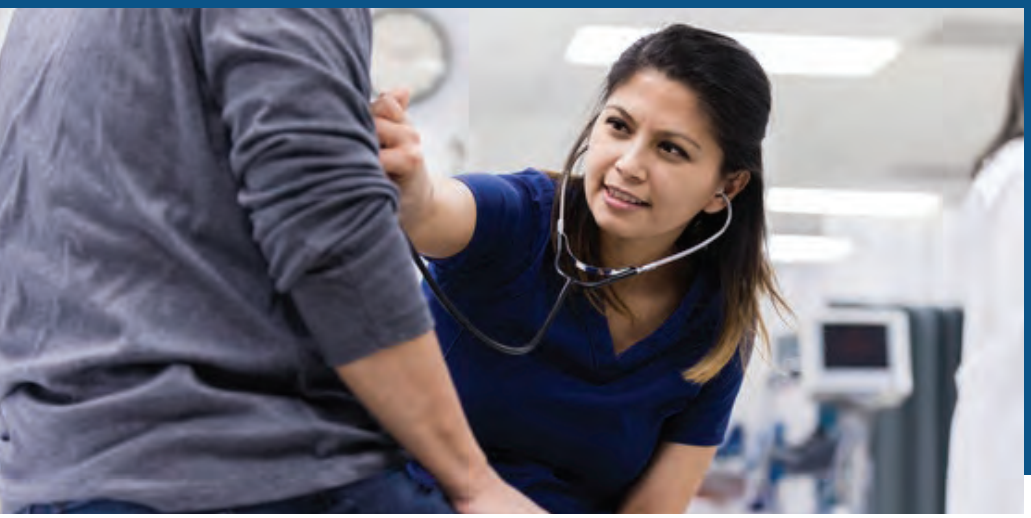
In contrast, in very mild cases of metformin toxicity, it would be appropriate for patients to be observed for at least 8 hours and recheck of labs including lactate, blood gas, and CMP. In most cases, however, admission to the ICU for enhanced elimination of lactate and metformin with optimal ventilatory settings and treatment of shock is required.

CASE CONCLUSION

The patient was diagnosed with severe metabolic acidosis secondary to metformin toxicity in the setting of acute renal failure. He received renal replacement therapy (RRT) and dialysis while admitted with improvement in his condition and was extubated two days after admission. A tunneled dialysis catheter was placed on hospital day 13 and the patient was discharged with outpatient dialysis in place. His twice daily metformin was discontinued, and daily sitagliptin was substituted in its place. ✶

References available online.

Substance Use Harm Reduction 101 for the Emergency Clinician



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WHAT IS HARM REDUCTION IN SUBSTANCE USE?

Harm reduction for substance use is an evidence-based approach to engaging with people who take mind-altering substances with the goal of meeting them where they're at. It aims to be non-judgmental and patient-driven, to lead with respect, and to focus on improving quality of life rather than abstinence. Importantly, the aim of harm reduction is not to get patients to quit taking drugs (which as you probably know from your high school health class doesn't actually work), it's to help them find ways to interact with drugs safely. Most people with whom we talk about substance use in the emergency department won't stop taking drugs just because a clinician has advised them to, and an abstinence-based approach implies that we know better than our patients do regarding their own relationship with substance use. Quitting cold turkey is also extremely difficult and often unrealistic. Instead, we can encourage and facilitate taking incremental behavioral steps to move our patients' relationships with mind-altering substances closer to where they want it to be. That being said, there are times when it's important to emphasize the necessity of complete cessation, for example, in later-stage liver failure or COPD.

WHAT COUNTS AS A "MIND-ALTERING SUBSTANCE"?

We typically think of alcohol and "drugs" (marijuana, cocaine, MDMA, meth) but I personally include tobacco and caffeine in this category. Your pre-shift Cherry Limeade Celsius makes you feel alert, maybe even anxious; nicotine can make people feel more focused or calm. I also make sure to include non-street medications under this umbrella (prescription or OTC), as people can take things like benzodiazepines, antihistamines, antipsychotics, cough medication, etc., in order to change how they feel, be that formally sanctioned by a clinician or not.

DOESN'T A HARM REDUCTION APPROACH ENCOURAGE PEOPLE TO USE MIND-ALTERING SUBSTANCES?

No; this has been demonstrated in many different settings over the last several decades, but perhaps nowhere more convincingly than in Vancouver, Canada. The city was a national epicenter for HIV and intravenous drug use-associated overdose in the late 1990s, which prompted the establishment of a large-scale public health effort, including syringe distribution, community programming, methadone therapy centers, and creation of the first sanctioned Supervised Injection Facility in North America. Rates of HIV, HCV, and drug-related harm — including overdose — have since plummeted.[1] In July 2021, Rhode Island opened the United States' first legal supervised consumption site, and later that year, two sites opened in New York City.[2]

An approach to taking a substance use history: this certainly isn't the "right" way to do this, it's just a system I've found works for me and is aligned with my values and practice.

1. What do you take? I prefer this language over "What do you use?" as that can sometimes sound pejorative, and discounts the fact that many people take mind-altering substances in a way that borders on or is medicinal. If you've ever come home after a long shift and thought "I could use a glass of wine," then

References available online.

you've thought about alcohol medicinally. Many people take more than one thing, so be sure to ask about co-ingestion to evaluate risk of interaction. The free harm reduction website Tripsit has a useful interactive tool that describes the effects of combining many popular substances.

2. How do you take it? Most people will know what you mean, but when a patient asks for clarification, I'll give examples. This means drug delivery method: ingestion (like taking a drink or having a gummy), smoking (including bongs), vaping, injecting (into vasculature or subdermally, called "skin popping"), or insufflating. Substances can be insufflated intranasally (snorting or sniffing) as well as rectally or vaginally — "boofing" aka "plugging" or "booty bumping" is when a substance is placed into a body cavity so that it gets distributed to the rest of the body via mucosal blood vessels. People can also use drugs transdermally (like nicotine patches) or sublingually (like buprenorphine). Note that asking a patient if they take a substance in a way people don't or can't use it — for example, asking a patient if they smoke LSD, which is pretty much only taken sublingually — may expose your lack of knowledge about that substance. However, I empower you to ask patients to educate you whenever appropriate. Regardless of the method, be sure to react non-judgmentally. If they endorse injecting, ask where they inject so you know where to look for signs of infection.

3. Ask safety-related questions related to the method. If the patient is injecting, ask if they use clean needles, if they clean their skin with alcohol swabs before injecting, if they ever share equipment like needles or spoons. If they take edibles, ask if they take store-bought or homemade. For almost all drug delivery methods, I ask if patients measure out their dose. For injection drugs, this might mean using a graduated syringe, like we use for giving medications. For edibles, this might mean checking the package of store-bought material for the milligrams of THC or psilocybin, for instance, in one serving. For powders or crystals, I suggest investing in a small and sensitive scale such as a jeweler's scale to measure out individual doses. Not only does this help to prevent overdose, but it may help people find out how much of a substance makes them feel good compared to how much makes them feel uncomfortable. Some drugs, like pressed pills, are harder to know the dosage — I encourage people to ask whoever they're getting the drug from for dosing information whenever possible. I also ask if they take drugs alone. As you might imagine, this is a safety issue when it comes to adverse drug reactions including overdose, but it also helps give me an idea of the relationship with the substance. While taking drugs alone isn't always indicative of an unhealthy relationship with that drug, solo use might point toward a relationship with that drug that may feel out of that person's control — think of the patient with alcohol use disorder who has an eye-opener or drinks secretly, versus someone who has a beer while hanging out with their friends. I ask about test strips and naloxone with every substance (including marijuana) other than alcohol and nicotine. I encourage you to take the time to learn how to use both, both for patient education and for civilian safety (including your own) outside of the hospital. Many city and state websites have succinct instructional guides — as a New Yorker, I give the following printable materials to my patients with their discharge paperwork: here's the one on test strips, and here's the one on naloxone.

"I recommend meeting them where they are and providing resources according to what they're looking for, or maybe just a step past that."

4. Ask how they feel about their relationship with what they're taking. Sometimes, when patients end up in the ED related to their substance use, it can be a bit of a wake-up call for them to change or reduce their use. However, some people end up in the ED for reasons unrelated to their substance use, or have been brought in involuntarily, or have been in the ED several times related to drugs and/or alcohol. You've likely practiced motivational interviewing before, and it's been well-studied to be effective in encouraging change regarding substance use. However, not all substance use is dangerous, and not all substance use needs to be changed or stopped. (For example: do you have the occasional drink at brunch with your friends, and feel like you have a healthy relationship with alcohol? Great! You probably don't need motivational interviewing on your alcohol consumption.) If a patient endorses a challenging relationship with their substance use, I recommend meeting them where they are and providing resources according to what they're looking for, or maybe just a step past that. This might mean individual or group therapy, support groups, detox centers, methadone clinics... My rule of thumb is: it's not a disorder unless it's causing distress. For example, you might experience anxiety, but it's not Generalized Anxiety Disorder until it's making it difficult for you to live your life — this is built into the diagnostic criteria of many mental illnesses.

Ask about community and loneliness. Unhealthy relationships with drugs and alcohol have been noted

References available online.

to be related to a lack of supportive community. That's why programs like Alcoholics Anonymous focus so much on coming to meetings and spending time with sponsors. AA's Big Book (a worthwhile read and available for free in its entirety on the AA website) has a chapter dedicated to the importance of developing community, and professionals working in substance use recovery will often emphasize forming meaningful relationships outside of the substance. This is because an unhealthy relationship with a substance is thought to be standing in as a surrogate for relationships with people.

This is by no means meant to be an exhaustive resource and certainly isn't the only correct way to approach talking about substance use in the ED. My hope is that this guide may help reframe substance use as something familiar rather than foreign, so that we can better understand our patients. As providers who work at the front door of the healthcare system, it's essential that we take the time to figure out how each of us wants to interact with this crucial topic in contemporary public health, and to let that change as we continue to practice. ✶

References available online.

From Heart Stop to Foot Drop: Lisfranc Injury after Cardiac Arrest

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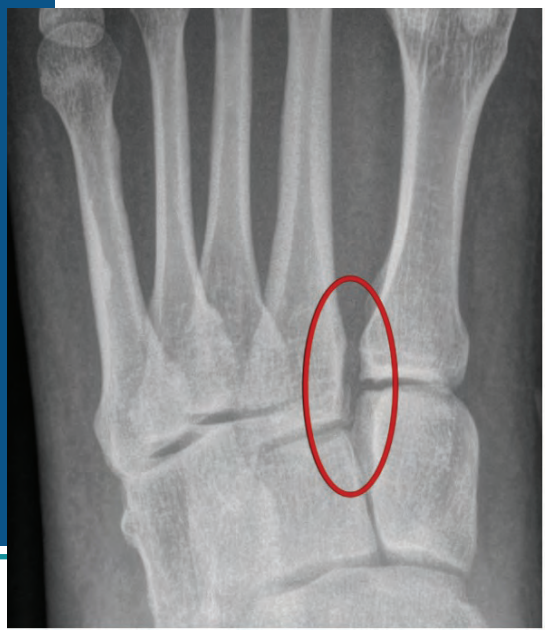

Figure 1. Initial foot X-ray read as normal, cortical irregularity seen at base of second metatarsal

INTRODUCTION

The Lisfranc joint complex, also known as the tarsometatarsal (TMT) joint complex, is essential for maintaining the arch of the midfoot.[1] The TMT joint complex functions as a pivotal joint for midfoot function and stability, particularly with weight-bearing activities. The main osseous architecture comprising the TMT joint complex is the articulation in the midfoot between all three cuneiforms, the cuboid, and the metatarsal bones.

Lisfranc injuries are typically categorized into high-energy and low-energy mechanisms. High-energy mechanisms involve significant axial loading or rotational forces that cause either ligamentous injury or fracture dislocation at the TMT joint. Low-energy mechanisms are most commonly caused by axial loading or twisting while the foot is planted.[2] Direct trauma to the midfoot can also result in Lisfranc injuries.[3]

Injuries to the Lisfranc complex may be misdiagnosed or go untreated, which is associated with substantial morbidity due to its intricate role in biomechanics and maintaining the arch of the foot. When the joint is injured, the injury results in abnormal weight distribution and biomechanical changes in the foot arch.[3] The location and severity of a Lisfranc injury will determine if surgery is recommended.[1]

CASE PRESENTATION

A 49-year-old male patient presents to the emergency department due to 1 week of left foot pain and swelling. The patient suffered from a cardiac arrest following a witnessed syncopal collapse while carrying groceries. Since he was discharged from the hospital, despite a negative foot X-ray completed as an inpatient, he has continued to have difficulty ambulating due to significant pain, and noticed progressive bruising of the foot. Physical exam was notable for midfoot and forefoot swelling, significant ecchymosis of the plantar midfoot and lateral foot, and dorsal and plantar midfoot tenderness to palpation overlying the Lisfranc complex. Metatarsal squeeze test was positive. Remainder of the left lower extremity exam was unremarkable.

Recent AICD placement precluded the patient from obtaining an outpatient MRI. A CT scan of the foot was performed in the ED, which demonstrated an acute non-displaced intra-articular fracture at the insertion of the Lisfranc ligament at the base of the 2nd metatarsal with otherwise normal tarsal-metatarsal alignment.

CLINICAL EXAMINATION FINDINGS

The clinical exam findings in patients with Lisfranc injuries vary based on the type and severity of the injury. There will likely be pain and tenderness overlying the TMT joint complex, swelling of the midfoot region, or plantar ecchymosis.[2] Stress testing for instability of the TMT joint may show dorsal subluxation suggestive of instability.[4] Neurologic deficits or compromised blood flow due to compartment syndrome are relatively rare but can occur, and may accompany severe dislocations or fractures.

X-rays of the foot may be unremarkable, demonstrate subtle diastasis at the base of the first and second metatarsals, or reveal a fracture such as a "fleck sign" at the base of the first or second metatarsal.[5] CT or MRI may help to confirm diagnosis, with MRI being a preferred modality to evaluate Lisfranc joint complex in addition to bony structures.[6]

DISCUSSION AND MANAGEMENT

Alternative diagnoses that may be considered when evaluating for Lisfranc injuries include midfoot and metatarsal shaft fractures, soft tissue injury, and vascular etiologies. However, XR imaging and vascular studies will show different patterns of injury and pathology that are not typically seen with Lisfranc injuries.

References available online.

Management of Lisfranc injuries may be conservative or surgical based on the severity of the injury. Non-operative management may be considered for non-displaced injuries or those with minimal disruption of the TMT joint complex.[2] For patients evaluated in the ED with concern for Lisfranc injury, it is reasonable to immobilize with a boot or short leg splint/cast, encourage non-weight-bearing status, and provide crutches to assist ambulation and mitigate risk of worsening injury. It is imperative for patients to follow up closely as an outpatient with a sports medicine provider, podiatrist, or orthopedic foot/ankle specialist within 1-2 weeks for continued management to ensure that there is no injury progression or in case surgical fixation is indicated.

Early diagnosis and appropriate management favor a positive outcome.[6] Surgical intervention generally leads to better functional outcomes in cases of displaced fractures or joint instability. However, this can come with long-term complications such as arthritis, stiffness, and post-traumatic deformities such as midfoot arthritis and arch collapse.[7,8]

CASE RESOLUTION

At discharge, the patient was provided with a walking boot and advised to limit weight bearing on the affected extremity with use of cane (unfortunately for the patient, broken ribs suffered from CPR made use of crutches difficult). He was able to follow up with a podiatrist the following week, and treatment options including surgical fixation and non-weight bearing in a short leg cast were suggested, but the patient declined. ✷

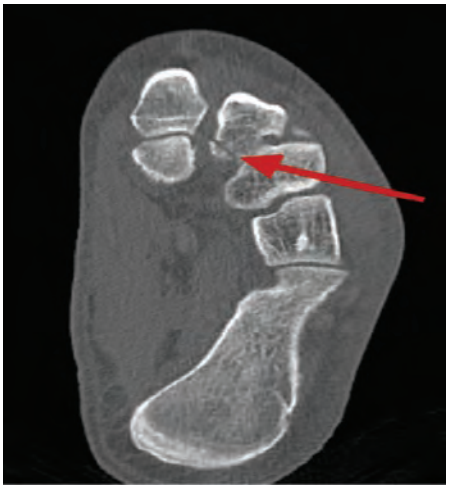

Figure 2. CT of left foot demonstrating fracture at base of second metatarsal

References available online.

Thoracic Outlet Syndrome as a Cause of Arm Swelling in Hemodialysis Patients: A Case Report

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Upper extremity swelling in the hemodialysis (HD) dependent patient may be the result of multiple conditions. The evaluation of a vascular fistula or graft placed for hemodialysis is commonly initiated early on to identify potential abnormality or emergency.

Ultrasound is often sufficient to evaluate peripheral causes of arm swelling; however, it may fail to identify more proximal etiologies, such as thoracic outlet syndrome (TOS). Familiarity with differentials for upper extremity swelling in the HD dependent patient may be helpful in the ED, as the prevalence of end-stage renal disease (ESRD) continues to rise in the United States and now approaches 786,000 individuals.[1]

CASE REPORT

A 60-year-old male with a history of ESRD on HD presented to the ED with a complaint of one month of right upper extremity (RUE) swelling. Initial vital signs were heart rate 72 beats per minute; respiratory rate 18 breaths per minute; blood pressure 168/98 millimeters of mercury; temperature 37.3° C; and oxygen saturation 100% on room air. The patient had a mature arterio-venous fistula (AVF) in the right arm and was sent to the ED from the dialysis center as the arm swelling appeared to worsen with each session of HD. He was unable to receive dialysis for the past several weeks as the dialysis center was unwilling to access the fistula in fear of worsening a possible AVF complication. Prior to this visit, the patient had been evaluated twice at an outside emergency department with an upper extremity ultrasound showing no abnormality within the arm or fistula. The patient's swelling had progressively increased, resulting in severe pain and +2 pitting edema involving the digits up to the axilla.

A repeat RUE venous Doppler study in our ED again demonstrated a patent AVF without thrombus. The decision to obtain a computed tomography (CT) of the chest with intravenous (IV) contrast was made for additional diagnostic information.

The CT chest demonstrated "venous stent overlying the proximal right subclavian and distal right brachiocephalic vein with severe narrowing at the area of the first rib, which appears to be causing impingement of the stent." These findings were relayed to the patient, which prompted him to recall that he had previously undergone venous stenting 3 months earlier at a nearby facility.

The patient was admitted to the medicine service, with the nephrology and vascular surgery teams following for the primary diagnosis of vascular TOS. During his hospital stay, he received HD and interventional radiology guided stenting with improvement in his right subclavian vein stenosis and a significant reduction in his RUE swelling. Vascular surgery recommended treatment with aspirin and clopidogrel and transfer to a hospital with cardiothoracic surgery capabilities for first rib resection as a definitive treatment for venous TOS.

DISCUSSION

TOS is a relatively rare condition that can lead to severe swelling of the extremity distal to the site of stenosis. Diagnosis of this condition may be difficult as the site of stenosis can be located much more proximally. This often leaves ultrasound studies inadequate and necessitates the use of IV contrast, which may be contraindicated specifically in oliguric patients such as ours. TOS encompasses a group of disorders that occur when structures running between the first

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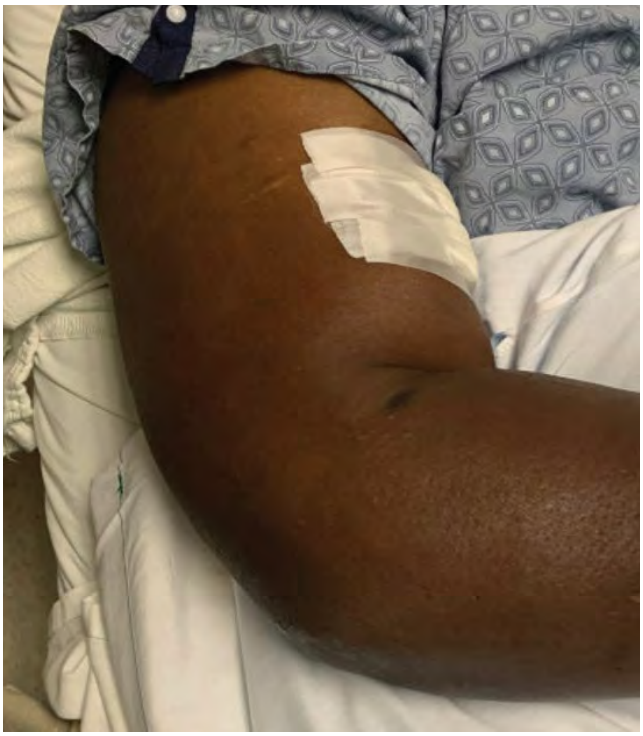


Figure 1. Right upper extremity swelling with ipsilateral HD fistula

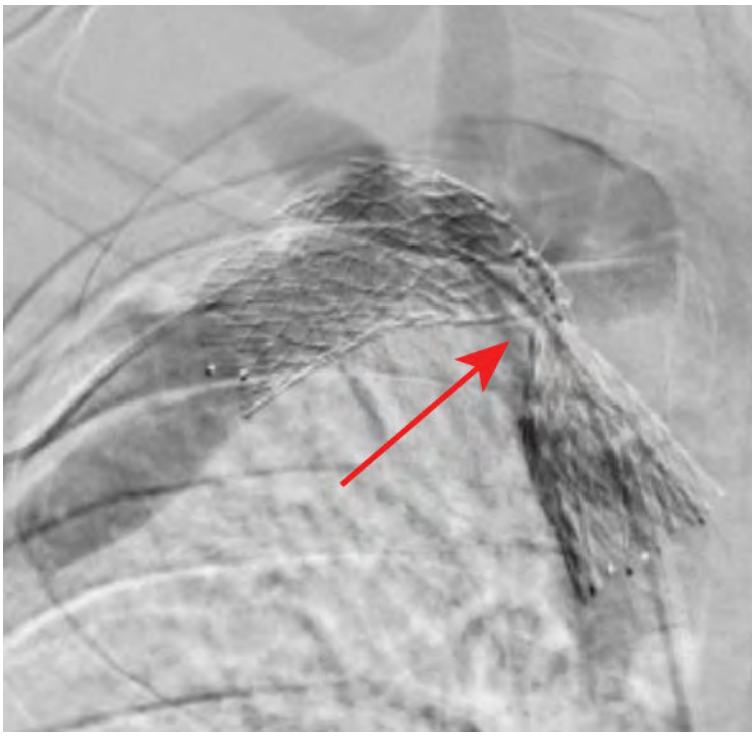


Figure 2. Venous thoracic outlet syndrome with stenting at site of severe stenosis

rib and collarbone (thoracic outlet) are compressed. It can be subcategorized into neurogenic, venous, or arterial, with neurogenic TOS being the most common, representing about 95% of cases. Venous TOS accounts for 3-5% of cases, and arterial TOS accounts for the final 1-2%.[2]

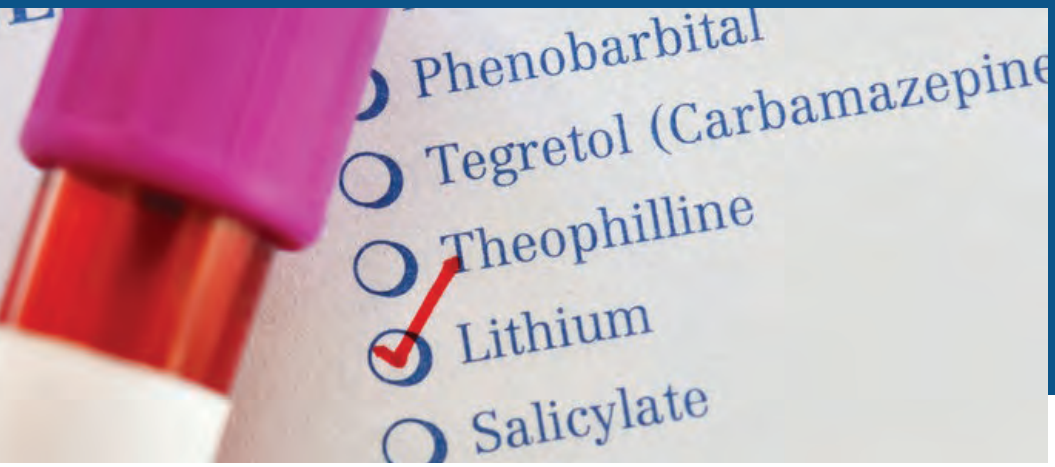
TOS can be caused by a variety of mechanisms, including trauma, repetitive motions, and anatomic variations. Venous TOS is recognized as a potential complication associated with AVF formation and is exacerbated by conditions unique to HD.[3] Vascular grafts are associated with neointimal hyperplasia, marked by increased smooth muscle cell proliferation and increased luminal stenosis, additional factors such turbulent flow, and endothelial dysfunction are also contributory.[4,5]

Treatment of venous TOS is often necessary for the salvage of the AVF and requires venous stenting followed by decompression of the thoracic outlet through first rib resection.

CONCLUSION

TOS is an important cause of arm swelling and fistula failure among ESRD and should be considered in ED settings. Due to the increased risk of coagulopathy with a high-flow fistula, patients on hemodialysis are more likely to experience thrombosis and stenosis due to endothelial dysfunction and neointimal hyperplasia.[6] Although limited data are present, there have been several clinical incidents in which patients have required thoracic outlet decompression for venous TOS associated with hemodialysis.[7] In the workup of a painful and swollen arm with AV-fistula, differentials should include both peripheral and central sources of swelling. In cases such as these, an upper extremity ultrasound is helpful in the diagnosis of pathology within the arm — but may have limited efficacy if the disease process extends proximally into the torso. This case demonstrates the importance of broadening our differentials, using high-quality clinical judgment with respect to history taking and examination, and involving our consultants early in order to accelerate the timing in which patients are treated since prior studies have suggested that a prolonged duration of symptoms is associated with poorer outcomes.[8] ✷

A Case of Lithium Toxicity Masked by Behavioral Symptoms in a Psychiatric Patient



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CASE REPORT

A 14-year-old female with a history of bipolar disorder, depression, and hypothyroidism presented to the emergency department with her mother for a psychiatric evaluation. Her mother expressed concern that the patient had been "pocketing" her medications—lithium, quetiapine, and levothyroxine—by hiding them in her cheek and spitting them out over the past two weeks. On some of these occasions, the mother found her hiding the medication and was able to flush them down the toilet. Otherwise, medications were kept locked away to prevent independent access by the patient.

Additionally, the mother reported the patient had recently been eating poorly, often hiding her food. The mother was concerned this behavior might indicate a suicide attempt, as the patient had previously attempted to starve herself.

Over the past few days, the patient had exhibited increasingly erratic behavior, including episodes of violence followed by withdrawal. Family members noted this pattern had occurred multiple times before, and it was assumed that much of the patient's altered mental state was due to behavioral issues.

On physical examination in the ED, the patient was awake and alert, though her motor movements were slowed. She demonstrated diminished responsiveness to external stimuli and would not give verbal responses. Her oral mucous membranes were dry. She exhibited mild hyperreflexia in addition to inducible clonus in her lower extremities. Her vital signs were within normal limits.

Laboratory tests, including glucose, CBC, TSH, free T4, salicylate level, acetaminophen level, urine drug screen, urine pregnancy test, and ECG, all came back within normal limits. However, CMP revealed an elevated creatinine level of 1.30 mg/dL and hyponatremia with a sodium of 128 mEq/L. A lithium level was also obtained, which was elevated at 6.7 mEq/L.

Consultations with pediatric nephrology and toxicology were promptly initiated, and the patient was admitted to the pediatric ICU for fluid resuscitation and hemodialysis (HD).

DISCUSSION

Distinguishing between medical and psychiatric causes of altered mental status (AMS) can be challenging, as the two may present with similar clinical manifestations.[1] As a result, patients with underlying psychiatric conditions who present with AMS are at heightened risk for misdiagnosis, though data on the frequency of such misdiagnoses remains limited.

Emergency physicians must maintain a high level of suspicion for potential organic or alternative causes of AMS in psychiatric patients, even when the clinical history strongly suggests a psychiatric etiology. This caution is especially critical in pediatric populations, where symptoms may be less apparent or misattributed to behavioral issues. Particular attention should be paid to physical exam findings, such as hyperreflexia and clonus in this case. These findings may help expand the differential diagnosis.

References available online.

Physicians must also be vigilant when managing patients on high-risk medications, such as lithium, which has a narrow therapeutic index of 0.8–1.2 mEq/L.[2] Lithium toxicity, if not promptly recognized and addressed, can lead to catastrophic consequences. Severe intoxication may present with seizures, coma, hemodynamic instability, and even death.[3] Additionally, physicians should be acutely aware of lithium's renal excretion, as any degree of kidney impairment in a patient on lithium therapy can precipitate toxicity.

In any patient presenting with AMS who is prescribed lithium, obtaining a serum lithium level should be a part of the workup in the ED if available.

It is important to note that serum lithium levels do not fully reflect intracellular concentrations, where lithium exerts its pharmacologic effects. Consequently, treatment decisions should not be based solely on lithium levels but must also consider the patient's clinical symptoms.[4]

TREATMENT

Hemodialysis (HD) is the preferred treatment for severe lithium toxicity. Guidance on indications for HD is provided by the Extracorporeal Treatments in Poisoning (EXTRIP) Workgroup.

Per EXTRIP guidelines, extracorporeal treatment (ECTR) for lithium poisoning is recommended:

- If kidney function is impaired and the lithium is greater than 4.0 mEq/L
- In the presence of a decreased level of consciousness, seizures, or life-threatening dysrhythmias irrespective of lithium levels

Additionally, ECTR is suggested:

- If the lithium level is greater than 5.0 mEq/L
- If confusion is present
- If the expected time to obtain a lithium level less than 1.0 mEq/L with optimal management is greater than 36 hours[5]

CASE CONCLUSION

The patient underwent intermittent HD until her lithium levels declined and stabilized. Gradually, her mental status returned to baseline. Psychiatry was consulted and patient did admit to intentional lithium ingestion with suicidal intent. She described becoming physically ill in the days following ingestion, likely leading to her dehydration and worsening of lithium toxicity.

The initial differential diagnosis for this patient was broad. Dehydration raised the possibility of metabolic disturbances, while medication nonadherence suggested a potential exacerbation of hypothyroidism. The presence of lithium use raised concerns for lithium toxicity or serotonin syndrome, the latter of which can be precipitated by lithium.[3] Although a psychiatric etiology for the AMS remained on the differential, it was ultimately considered a diagnosis of exclusion.

Obtaining a lithium level was paramount to the patient's clinical course. Despite a history that did not strongly suggest lithium overdose and minimal physical exam findings indicative of lithium toxicity, the lithium level was crucial for guiding appropriate treatment. Without this diagnostic step, the patient may not have received timely HD, potentially leading to devastating consequences. ✶

Clinical Pearls

- Maintain a high index of suspicion for medical causes of AMS in psychiatric patients.
- Psychiatric symptoms may mask underlying medical causes of AMS. Always consider and rule out non-psychiatric causes such as toxicologic, metabolic disturbances, or medication-related issues. Look for subtle physical exam findings that can aid in the diagnosis.
- Obtain a lithium level in patients with AMS who are on lithium therapy.
- Lithium toxicity can go unrecognized in patients with AMS. A reassuring history or vague symptoms should not deter the clinician from obtaining a lithium level when AMS is present in a patient taking lithium.
- Consider HD in patients with lithium toxicity.
- In cases of lithium toxicity, HD is recommended (1) if kidney function is impaired and the lithium is greater than 4.0 mEq/L or (2) in the presence of a decreased level of consciousness, seizures, or life-threatening dysrhythmias irrespective of lithium levels.

References available online.

I've Got Herpes on the BRAIN! A Case of HSV Encephalitis

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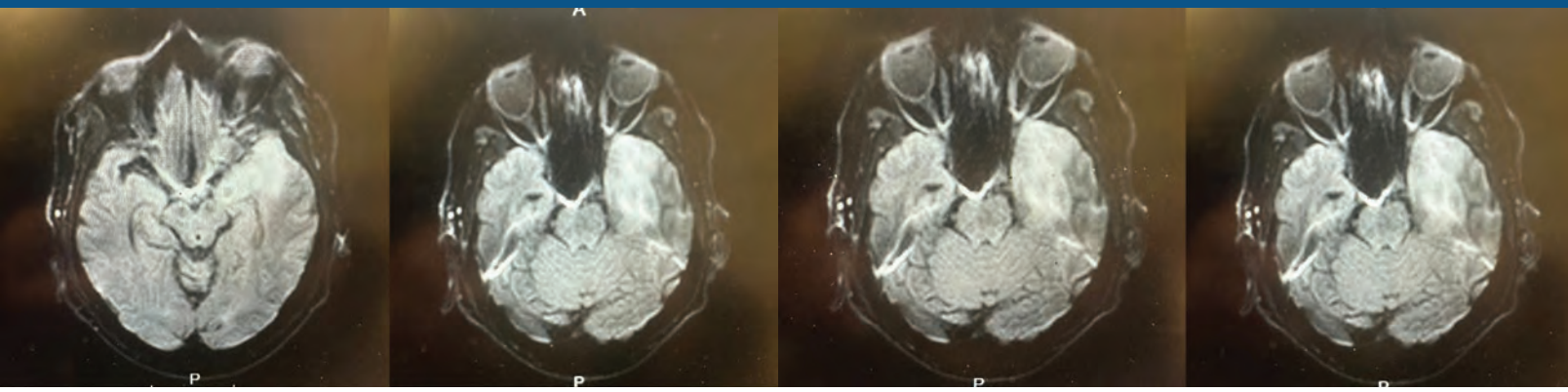

A 56-year-old male with a history of Type 2 diabetes, hypertension, and hyperlipidemia presented to the emergency department for altered mental status, fever, and headache for the past few days.

The patient was initially seen at an outside clinic, where he was found to have mild leukocytosis but otherwise unremarkable workup. Per the patient's wife, the patient had become increasingly altered. During ED visit, the patient was found to be febrile at 102°F despite administration of antipyretics, ondansetron, and broad spectrum antibiotics (ceftriaxone 2g IV and ampicillin 2g IV and vancomycin 2g IV). Altered mental status labs were ordered including CBC, CMP, TSH, ammonia level, salicylate level, alcohol level, acetaminophen level, urinalysis, urine drug screen, blood cultures, lactic acid, creatinine kinase, COVID/flu/RSV, EKG, chest X-ray, CT head without contrast. Labs were notable for hypokalemia at 2.9 and a creatinine kinase of 640, but otherwise showing no acute abnormalities contributing to his presentation. A lumbar puncture was attempted unsuccessfully, and CT Head showed mild biparietal cerebral atrophy with no acute intracranial findings. The patient was admitted to the medicine floor.

On admission, the patient was found to be somnolent, oriented only to person, with expressive aphasia. A lumbar puncture was ordered, with a meningitis panel and HSV serology. HSV-1 serology returned positive and CSF showed increased cell count, increased glucose, and increased CSF protein. MRI was performed at this time, which showed mild abnormal FLAIR hyperintensity and restricted diffusion within the left medial temporal lobe cortex. Given high suspicion for HSV encephalitis, an acyclovir regimen was started.

The patient continued to have waxing and waning presentation of altered mental status and expressive aphasia. Headaches were continuously treated with IV acetaminophen and dexamethasone.

EPIDEMIOLOGY/CLINICAL FEATURES

Herpes simplex virus type 1 (HSV-1) encephalitis is the most common cause of sporadic fatal encephalitis worldwide. The clinical syndrome is often characterized by the rapid onset of fever, headache, seizures, focal neurologic signs, and impaired consciousness.[1] This patient had presented with fever, headache, and altered mental status for the days prior to his Emergency Department admission. HSV-1 encephalitis is a devastating disease with significant morbidity and mortality, despite available antiviral therapy.

Other clinical features can be marked by acute (<1 week in duration) and include focal cranial nerve deficits, hemiparesis, dysphasia, aphasia, or ataxia.[2] Over 90% of patients will have one of the above symptoms plus fever.[2] Other associated neurologic symptoms include urinary and fecal incontinence, aseptic meningitis, localized dermatomal rashes, and Guillain-Barré syndrome.[3] During the patient's ICU stay, he did require an external male catheter after a few days as he had become incontinent. The patient also had diminished comprehension, paraphasic spontaneous speech, impaired memory, and loss of emotional control — which are all common sequelae of the disease process.[4] The patient would repeatedly

References available online.

forget the names of the members of his healthcare team despite their continuity throughout his hospital stay. The patient exhibited alternating periods of sadness and extreme happiness, suggesting a potential loss of emotional regulation.

EVALUATION/DIAGNOSIS

Lumbar puncture is indicated for cerebrospinal fluid analysis and polymerase chain reaction (PCR) testing for HSV in any patient with encephalitis. The detection of herpes simplex virus DNA in the cerebrospinal fluid (CSF) by polymerase chain reaction (PCR) testing is considered the gold standard for establishing the diagnosis. Examination of the CSF typically shows a lymphocytic pleocytosis with counts ranging from 10 to 400 cells/microL, an elevated protein, and an increased number of erythrocytes (in 84% of patients).[5] This was consistent with this patient's laboratory findings; however, this patient did not have a high number of erythrocytes. Nucleated cells including lymphocytes were significantly elevated as was the CSF protein. ✶

> ***"Herpes simplex virus encephalitis is the most common cause of sporadic fatal encephalitis worldwide."***

References available online.

Orbital Cellulit*US*: Utilizing Ultrasound for Evaluation of Orbital Cellulitis

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Orbital cellulitis is an ophthalmologic emergency that requires immediate diagnosis and management; delayed diagnosis can pose a risk for vision loss and intracranial infectious extension. This case supports the use of bedside ultrasound in providing a timely and accurate diagnosis of orbital cellulitis.

CASE PRESENTATION

A 53-year-old-male presented to the ED for 7 days of right eye pain, blurry vision, and diplopia. The pain started while on a recent vacation. He denies any trauma, submersion injuries, or foreign body sensation. He had seen an ophthalmologist in an outside state and was prescribed a course of prednisone eye drops, which initially improved his symptoms. Several days later, the patient reported worsening of his symptoms with escalating pain, irritation, and swelling. Additionally, he progressed to developing diplopia and blurry vision, despite medication compliance. His symptoms were not associated with fevers, headaches, or changes in vision in the left eye.

PHYSICAL EXAM

Physical exam was significant for right eye proptosis with restriction of primarily abduction and elevation. He experienced pain with movement in all directions and resistance to retropulsion. Evidence of mild lid edema and erythema without scleritis was also present. His visual acuity was 20/20 OS and 20/25 OD and his pupils were equal and reactive to light. Tonometer pressure in the right eye was 22 mmHg, compared to the left eye which was 12 mmHg. There were no rashes present on the face or body, mastoid tenderness, or inflammation of the tympanic membranes.

LAB/IMAGING

Lab work revealed a white blood cell count of 6.4 [3.7-10.1 K/mm3], Lactic acid of 1.0 [0.7-2.0 mmol/L], ESR of 12 [0-15 mm/Hour], TSH of 0.91 [0.465-4.68 uIU/mL], Free T4 of 1.04 [0.78-2.19 ng/mL], and Total T3 of 1.60 [0.97-1.69 ng/mL].

Bedside ultrasonography was performed by the emergency physician and was notable for fluid collection around the posterior globe itself without discernible abscess. CT scan of the orbit with IV contrast (Figure 1) demonstrated acute right orbital cellulitis with superimposed posterior scleritis, and optic perineuritis. Recommendations per ophthalmology were to start intravenous steroids along with broad spectrum antibiotics. After antibiotics and steroids had been started, the patient was admitted to the hospital for continuation of medications as well as observation.

POCUS

Bedside ultrasonography was significant for free fluid posterior to the globe without discernible abscess. The linear probe was utilized on an ophthalmologic preset to obtain longitudinal and transverse images of the orbit. Longitudinal views of the right eye revealed a substantial volume of free fluid posterior to the globe with evidence of optic nerve enlargement and ill-defined margins (Figures 2 & 3).

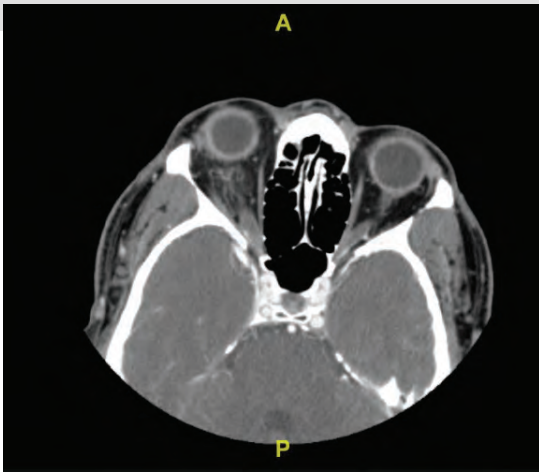


Figure 1. Initial CT of the orbit with IV contrast demonstrated acute right orbital cellulitis with likely superimposed posterior scleritis and optic perineuritis.

References available online.

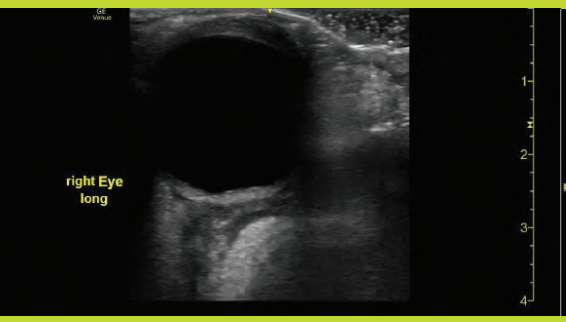


Figure 2. Hospital day 1 longitudinal view of the right eye using linear probe showed fluid surrounding the globe. Measurements of the optic nerve were 0.31 cm and 0.49 cm.

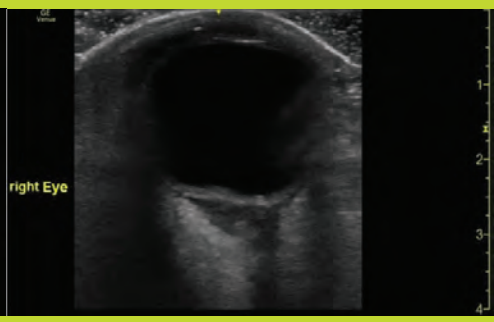


Figure 3. Hospital day 2 longitudinal view of the right eye using linear probe showed improvement of surrounding fluid around the globe.

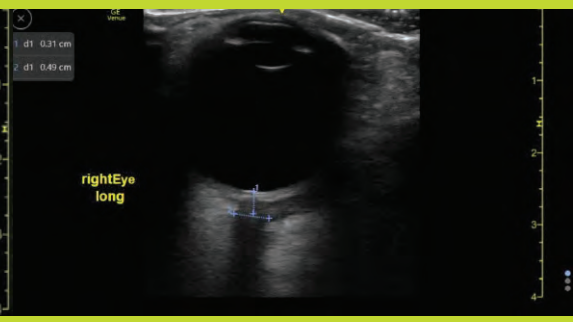


Figure 4. Right eye longitudinal view showed greater improvement of fluid collection compared to the previous day, although a small collection of fluid remained.

Repeat bedside scans were performed during the patient's stay in the hospital to monitor progression of disease and response to antibiotic therapy. Day 3 of admission showed a smaller volume of fluid consistent with radiographic improvement (Figure 4).

DISCUSSION

Orbital cellulitis is an ophthalmologic emergency that requires immediate diagnosis and management; delayed diagnosis can pose a risk for vision loss and intracranial infectious extension. Point-of-care ultrasound proved to be useful in the initial diagnosis of orbital cellulitis and for assessing response to treatment.

Although ultrasound can be used for risk stratification of patients with non-specific eye complaints, it is unlikely to negate the need for advanced imaging such as CT and magnetic resonance imaging in patients with symptoms highly suggestive of orbital cellulitis (i.e., ophthalmoplegia, proptosis, and impaired vision).[2] However, ultrasound may prove to be the preferred imaging modality in pediatric and uncooperative patients who may not tolerate a contrast-enhanced CT scan in the ED setting.

This case supports the use of bedside ultrasound in providing a timely and accurate diagnosis of orbital cellulitis.

CASE CONCLUSION

The patient received IV methylprednisolone 125 mg for acute inflammation, as well as vancomycin 3,072 mg, piperacillin/tazobactam 4.5 g, and ceftriaxone 2 g for broad spectrum antibiotic coverage.

Ultrasound re-evaluation on hospital day 2 showed improvement of periorbital swelling and the posterior orbital fluid.

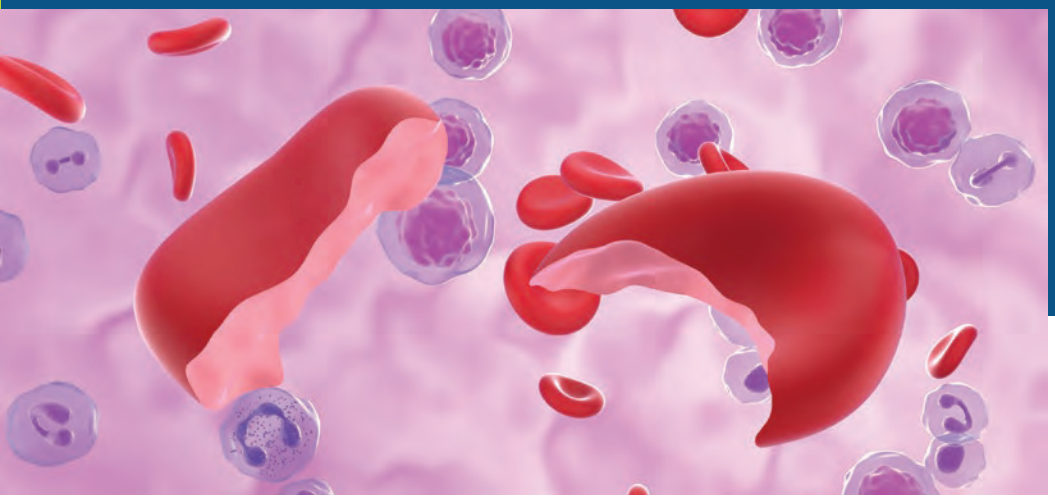
By hospital day 3, there was almost complete resolution of symptoms as well as a significant decrease in the fluid surrounding the globe. Physical exam showed improvement of ocular movement in all directions, with reduced pain and proptosis.

Following antibiotic completion in the hospital, the patient was continued on amoxicillin/clavulanic acid for an additional 7 days and prednisone 80 mg for 28 days with a taper. ✶

Disclaimer: This research was supported (in whole or in part) by HCA Healthcare and/or an HCA Healthcare affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA Healthcare or any of its affiliated entities.

References available online.

Cold Agglutinin Syndrome: A Case Report



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CASE REPORT

A 20-year-old female with no remarkable past medical history presented to the ED with a chief concern of 3 days of dark urine. The patient reported previously noted dark urine a few weeks prior to this presentation with full spontaneous resolution. In both episodes the patient had not experienced dysuria, vaginal discharge, urinary urgency or frequency, and these episodes occurred outside the patient's normal menstrual cycle. The patient also endorsed an approximately 1-week history of concomitant subjective warmth, fatigue, dry cough, tension-type headaches (similar to prior headaches and without concerning features), xerostomia, and reduced oral fluid intake. Further, the patient reported 2 episodes of urticaria each lasting less than 1 hour occurring in the 3 days prior to her ED visit. The first episode was noted on her bilateral upper extremities while the second presented over her bilateral lower extremities. Both episodes resolved spontaneously and the patient denied any use of novel cleansing products, fabrics, or awareness of other potential exposures.

On ED arrival, the patient's vital signs were recorded as a pulse 150, blood pressure 122/71, temperature 37.5°C, and oxygen saturation 99% on room air. Overall, the patient was well-appearing and the physical examination was unremarkable apart from the presence of dry mucous membranes without notable hepatosplenomegaly, lymphadenopathy, or other concerning findings. Urinalysis was notable for the presence of amber colored urine with 1+ protein, 2+ urobilinogen, 41 white blood cells per high powered field, and many bacteria. The sample was contaminated by 58 squamous epithelial cells, positive for nitrites, and negative for red blood cells (RBCs). Complete metabolic panel was remarkable for a transaminitis with alanine aminotransferase (ALT) elevated to 169 and aspartate aminotransferase (AST) elevated to 185, alkaline phosphatase (ALP) elevated to 129, and direct bilirubin elevated to 0.4 with a total bilirubin of 1.2. Complete blood count demonstrated a normocytic anemia with hemoglobin (Hb) 8.9, hematocrit 24, RBC count 2.54, and mean corpuscular volume (MCV) 93. White blood cell count was within normal limits at 8.3k/mm^3 and absolute lymphocyte count elevated to 3,840/mm^3 cells. Electrocardiography revealed sinus tachycardia with ventricular rate 124.

With no history of anemia, liver disease, and in the absence of hematuria, these results were concerning for an active hemolytic process potentially related to an underlying infection. Further testing revealed elevated lactate dehydrogenase (LDH) 1,108 U/L, haptoglobin <10 mg/dL, and a reticulocyte count 5.4% and 198.4 k/mm^3 cells. Peripheral blood smear was positive for polychromasia, target cells, and RBC agglutination. Epstein-Barr heterophile antibody testing was positive and additional viral testing for influenza A/B, COVID-19, and respiratory syncytial virus was otherwise negative. Ferritin was elevated to 192 ng/mL — however, iron level, iron percent saturation, total iron binding capacity, and transferrin were all within normal limits. A direct antiglobulin test was performed and was positive for complement C3 and negative for immunoglobulin G (IgG). Cold agglutinin titer testing returned positive at 1:128. Findings consistent with a diagnosis of cold agglutinin autoimmune hemolytic anemia (AIHA), believed secondary to infectious mononucleosis.

References available online.

In accordance with hematology recommendations, the patient was covered in warmed blankets with special care to acral areas. A repeat Hb was obtained decreasing to 7.8 from 8.9 g/dL. The patient was subsequently admitted for observation to the hospitalist service. Transfusion was not indicated during the admission as the patient's Hb levels ranged from a low of 7.2 g/dL before uptrending to 8.6 g/dL prior to discharge. Further testing demonstrated broad reactivity of the patient's plasma with cord blood cells and less so with her own RBCs at 1-6°C and weak reactivity at ambient room temperatures. Due to continued improvement, the patient was ultimately discharged in stable and improved condition on hospital day 3.

DISCUSSION

Cold agglutinin syndrome is an AIHA mediated by cold agglutinin, typically monoclonal, proteins. Primarily, these proteins consist of IgM autoantibodies, although IgA and IgG proteins have been reported in the literature.[1-3] These autoantibodies are invariably directed against specific RBC surface antigens of the *I* and *i* types. Binding across multiple antigen binding sites on each autoantibody leads to the agglutination characteristic of the disease process. Anti-*I* antibodies are usually associated with primary cold agglutinin disease and *Mycoplasma* infection, whereas anti-*i* antibodies are more commonly associated with Epstein-Barr virus (EBV) infections.[4] As *i* antigens are vastly more prevalent on infantile RBCs, the patient's plasma being more strongly reactive with cord blood than with her own RBCs is further evidence of the diagnosis.

This pathology is termed "cold" as the agglutinin proteins bind to their target antigens between 0-4°C and are most active between 3-4°C.[5] If the patient's blood is cooled, often occurring organically in acral regions of the body, cold agglutinin will bind to the RBCs and the resultant agglutination may precipitate circulatory symptoms such as Raynaud-like phenomenon or acrocyanosis. Additionally, complement-mediated hemolysis results as the now bound IgM recruits complement C1 leading to classical pathway activation and ultimately to macrophage phagocytosis.[6] This hemolysis is principally extravascular and occurs in hepatic Kupffer cells.[4]

While most cases comprise a primary autoimmune process and are more properly termed cold agglutinin disease (CAD), cold agglutinin AIHA may complicate other disease processes such as EBV infection, as occurred in this case, as well as *Mycoplasma pneumoniae* pneumonia and certain lymphomas, and in such cases is then generally referred to as secondary cold agglutinin syndrome (CAS).[6,7] *Mycoplasma* or primary atypical pneumonia has been identified as the likely underlying cause in 8% of AIHA cases and EBV rarer still and implicated in ~1% of cases, although the overall frequency of clinically significant hemolysis in both of these disease processes is unknown.[8] Believed quite rare overall, CAD has an incidence of 1/million, though this is likely an underestimation due to lack of clinical recognition.[9]

Diagnosis of CAD/CAS should begin with testing including a CBC with peripheral blood smear, LDH, haptoglobin, and indirect bilirubin. A direct antiglobulin (Coombs) test should be performed if workup is concerning for AIHA. Finally, a cold agglutinin titer should be obtained. Should testing concomitantly demonstrate evidence of hemolysis with a positive Coombs test and cold agglutinin titer >1:64 (higher number indicating greater presence of autoantibodies) at 4°C, the diagnosis is made.[1]

Generally, secondary CAS does not warrant specific treatment beyond supportive care and management of the underlying disease process as in this case.[7] Supportive care should most prominently consist of cold temperature avoidance to reduce further agglutination and hemolysis. Should the patient's Hb levels enter a transfusable range, this may be safely performed. More emergent and temporizing measures such as plasmapheresis, intravenous immunoglobulin (IVIG), and complement inhibitors such as eculizumab or sutimlimab may be warranted in individuals experiencing symptomatic or severe anemia.[7,10] In contrast, glucocorticoids are ineffective as they fail to inhibit IgM antibody production and should not be administered.[11] Should the EM physician suspect primary CAD, referral to, or consultation with, hematology is advised for consideration of management with rituximab-bendamustine or rituximab monotherapy.[12]

TAKE-HOME POINTS

Cold agglutinin syndrome (CAS) represents an autoimmune process resulting in complement-dependent hemolytic anemia usually mediated by IgM autoantibodies known to complicate various infections and malignancies. Although most cases secondary to infection are mild, transient, and resolve with the underlying infection, ~1/3 of patients develop anemia with hemoglobin levels <8 g/dL. In these instances, treatment beyond supportive measures such as cold avoidance, active rewarming, blood transfusion, and the initiation of additional pharmacotherapies may be warranted.

As infectious mononucleosis is a common diagnosis made in the emergency department (ED) setting and CAS is a relatively under-discussed complication, it is quite likely that many cases go undiagnosed. Though uncommonly requiring interventions beyond supportive care, it is nonetheless important for the EM physician to be familiar with this potentiality given an increased risk of morbidity and mortality in those diagnosed with EBV infectious mononucleosis. A high index of suspicion is indicated when patients testing positive for EBV present with associated findings such as acrocyanosis, dark urine, fatigue, pallor, or jaundice, and otherwise unexplained anemia as further management may be indicated. ✷

References available online.

Toxic Twist: Valacyclovir and the Mystery of Altered Mental Status

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A 46-year-old male with a history of end-stage renal disease (ESRD) on dialysis was brought to the emergency department by his family for intense, new-onset visual hallucinations. Just prior to ED arrival, he visited his ophthalmologist for a routine evaluation. Shortly after leaving this appointment, he started experiencing vivid hallucinations of being on a farm and seeing children run past him.

He denied any infectious symptoms, trauma, recreational drug use, or pain. His vital signs were unremarkable. On exam, the patient was alert, oriented, and spoke in full sentences. He maintained insight and knew the hallucinations were not real, but continued to see things that were not there. His neurological exam was unremarkable.

Family members at the bedside reported he seemed different compared to his usual self, although they were unable to describe specific new behaviors other than the visual symptoms.

DIAGNOSIS / MANAGEMENT

An ophthalmology note from the patient's routine visit earlier in the day noted baseline visual acuity and did not reveal any clues to the patient's acute change in presentation. Further chart review was significant for a recent admission due to a malfunctioning AV fistula requiring a fistulogram. The patient was discharged a few days prior to ED presentation with his home medications refilled. While reviewing this discharge summary, the ED resident noticed a prescription for valacyclovir: 1g three times per day.

As there was no documented indication for antiviral therapy, this medication may have been inadvertently prescribed or refilled at discharge. Nevertheless, the patient had diligently taken several doses.

The plasma elimination half-life of the delivered acyclovir by valacyclovir in those with normal renal function is averaged at 2.5-3.3 hours, while the half-life for those with ESRD is averaged at approximately 14 hours.[1] Thus, the recommended dosing of valacyclovir for patients with renal disease differs depending on the indication and the level of renal impairment. Some dosing regimens are as low as 500mg every two days.[1] The patient's prescription had not been adjusted for his renal impairment, and as a result, he ingested approximately 6 times the recommended dose prior to presenting to the ED.

Symptoms from valacyclovir toxicity range widely in severity, from general malaise to coma. As seen in this patient, toxicity can cause central nervous system (CNS) effects including agitation, hallucinations, confusion, and encephalopathy.[1] Non-neurological sequelae such as acute kidney injury and thrombotic microangiopathies such as thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS) have also been reported.[1]

The Poison Control Center was consulted. Given the patient's relatively mild encephalopathy, they encouraged regularly scheduled dialysis to assist with valacyclovir clearance. The patient was noted to grow progressively more agitated and delirious over the next day, but his mental status rapidly returned to baseline after dialysis without recurrence of visual hallucinations. He continued to do well and was discharged after a brief hospital stay.

TAKE-HOME POINTS

- Medications often require patient-specific dosing adjustments to avoid adverse effects. Even medications that we view as relatively low-risk can cause severe toxicity.

- A thorough medication reconciliation is required for all patients with altered mental status, especially for patients who have recently interacted with the healthcare system. Errors in medication prescription can lead to serious consequences and even toxicity, as demonstrated in this case.

- Symptoms of valacyclovir toxicity can range in severity. CNS effects range from general malaise to hallucinations, agitation, and coma. They are most often seen in patients with renal impairment.[2] Early identification and intervention, such as dialysis in severe cases to assist with metabolite clearance, can lead to favorable outcomes.[3] ✶

References available online.

The Tales of (Cardiac) TNK

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INTRODUCTION

Coronary artery disease (CAD) affects 16.8 million people worldwide annually, and about half are noted to have a known history of CAD.[4]

Inferior wall myocardial infarctions (MI) are most commonly caused by an occlusion of the posterior descending artery via the right coronary artery, which is seen in 80% of the population. In the other 20%, it is seen as an occlusion in the circumflex artery.[1] MIs affect about 3 million people worldwide, and cause about 1 million deaths in the U.S. annually.[2] In 2013, about 40-50% of all MIs were inferior in nature, and it was noted that inferior MIs have a better prognosis compared to other MIs; 2% vs. 9% mortality.[1] Recent studies have shown this number to increase to about 12%, especially with right ventricular involvement.[3]

The main treatment for any coronary occlusion is immediate reperfusion; the preferred treatment involves Percutaneous Coronary Intervention (PCI). According to the American Heart Association (AHA), the gold standard is to allow for door to balloon time within 90 minutes. In the cases of not being in a PCI capable facility, thrombolytics can be administered within 30 minutes of contact with the patient. This standard has not changed in over 20 years. Outside of reaching metrics, when else should thrombolytics be considered? In both scenarios below, acute decompensation was a key driver that pushed providers to give thrombolytics, even while being in a PCI capable center. Although outcomes were different, this may be something to consider in our acute occlusive myocardial infarction (OMI) patients if their courses change while still under our care.

CASE PRESENTATION 1

A 69-year-old female with a past medical history of hypertension (HTN), diabetes (DM), questionable history of congestive heart failure (daily lasix and metoprolol), presenting with worsening shortness of breath at night. Per family, the patient has been having trouble breathing for the last week, worse when lying down at nighttime. Denies any new symptoms that made them come to the ED that night. The patient reported no other associated symptoms of dizziness, chest pain, nausea, vomiting, changes in urinary habits. Vitals on arrival remarkable for hypotension (80s systolic) but on repeat normotensive, respiratory rate of 35, saturating 90% on room air. On examination, the patient appeared in mild respiratory distress, but clear in all lung fields. EKG on arrival show normal sinus, questionable depressions in the lateral leads. Initial management included placing the patient on Bi-Level Positive Airway Pressure (BIPAP). Initial bedside echocardiogram demonstrated grossly preserved ejection fraction with no concern for myocardial hypokinesis, mild B lines were seen with a plump IVC.

Labs collected on arrival demonstrated: BUN/Creatinine 85/5.6, glucose 320, potassium 5.2, CO2 9, Anion Gap 23, BHB 0.4. Cardiac labs noted with a trop of 353, BNP >70000. Venous blood gas, pH 7.2, pCO2 27, lactate 4.4, HCO3 11. Patient appeared to be in an Anion Gap Metabolic Acidosis likely secondary to impending DKA vs. Uremia vs. Lactic acidosis vs. AKI/CKD with no known history of renal disease, no history of hemodialysis discussion or use in the past.

One hour into the visit, the patient began to have chest pain and was

References available online.

noted to be hypotensive on one read with systolic in the 80s, MAPs ranging between 50-60s. The patient's blood pressure was varying during this time, at times normotensive, but majority of hypotensive with MAPs in the 50s. A repeat EKG was conducted showing mild ST elevations in leads II, III, aVL with reciprocal ST depressions in aVL, V4-V5. Serial EKGs confirmed these findings, and STEMI code was called. The patient at this time was consented for emergent PCI, loaded with antiplatelet therapy, heparin, and aspirin, but continued to be hypotensive. At this time, the patient was started on vasopressor and inotropic support with epinephrine infusion, later escalated to Norepinephrine to stabilize prior to catheterization.

The Cardiology team notified the ED team that cardiac cath lab was down, and the patient required transfer to a tertiary center to allow for catheterization. Cardiology initiated transfer and an ambulance would be there shortly to pick up the patient.

At this time, the patient continued to decompensate, with worsening hypotension, worsening tachypnea, diaphoresis, and vomiting.

In light of the peri-code inferior MI, now unstable for transfer, about 1.5 hours into the patient's ER visit, a decision was made to give thrombolytics; 50 mg of tenecteplase was administered. The decision to intubate for airway protection was made after pushing thrombolytics, as the patient was not hemodynamically stable prior to this intervention and there was hope of some clinical improvement after thrombolysis. The patient did not immediately worsen after thrombolytics and was intubated in one attempt successfully. Repeat bedside echocardiograms demonstrated worsening global hypokinesis. Patient's course continued to worsen, hypotension continued, leading to additional vasopressin, phenylephrine, and milrinone infusions. Sodium bicarbonate infusion was added to address the metabolic acidemia, along with treatment for questionable sepsis vs. other etiologies.

Ultimately, the patient became bradycardic, pulseless prior to transfer, and ROSC was not achieved.

CASE PRESENTATION 2

A 70-year-old Punjabi-speaking male with past medical history significant for CAD and NSTEMI (10/2017) s/p PCI of the dRCA, HTN, HLD, and DM who presented to the emergency department with complaints of 1 hour of sudden onset substernal, crushing chest pain, and shortness of breath that awoke him from sleep. Patient's son was present and helped translate and provide history. He noted the patient felt well during the day and had a few beers during a Super Bowl party. He denies vomiting and vision changes since the onset of symptoms, but the patient was too uncomfortable to provide further details.

Per EMS, the patient was given aspirin 325 mg en route to the hospital. On presentation, the patient was initially hypertensive at 191/94 and tachycardic to 134, but on repeat blood pressure 140/82, afebrile, saturating 100% on room air with no respiratory distress. Initial examination showed an uncomfortable appearing, mildly diaphoretic patient. Lung fields were clear to auscultation and cardiac auscultation showed tachycardia, but was otherwise unremarkable with no lower extremity edema. Initial bedside echocardiogram demonstrated preserved EF with no large global hypokinesis, lungs had no b lines and the IVC had respiratory variation.

Initial EKG showed normal sinus rhythm notable for ST elevation in lead AVR and ST depressions in anterior leads V2, V3, V4 and lateral leads V5 and V6.

Cardiology was consulted immediately upon this ECG, and recommended to initiate treatment with plavix loading dose, heparin bolus and drip, admission to Cardiac Critical Unit (CCU). Before medication could be initiated, the patient was found to be standing at bedside with difficulty breathing, rales bilaterally, and B lines on ultrasound. Repeat blood pressure was again elevated with systolics in the 190s. Patient was helped back into bed and given versed for increasing agitation likely secondary to difficulty breathing, oxygen saturations were in the 70s. Started on BIPAP and high dose nitroglycerin therapy at 300 mcg/min for concern of Sympathetic Crashing Pulmonary Edema (SCAPE) in the setting of possible OMI. After 5 minutes, the patient's vitals started slowly improving with oxygen into the low 80s and blood pressure 174/95; he remained diaphoretic and the ECG demonstrated worsening ischemia with evolving STEMI concerns.

Cardiology bedside activated Cath Lab but would not be available for one hour, later to find out the lab was not functioning. Family at bedside agreed for thrombolysis for STEMI without emergent PCI capability. The patient was given 50 mg TNKase, remained on BIPAP and high dose nitroglycerin infusion. The patient was further optimized prior to intubation with oxygen saturations consistently 100%, blood pressure remained elevated but improving slowly. The decision again was made to intubate after thrombolytics for the concern of further deterioration if the patient was not optimized prior to intubation.

Patient was intubated on one single attempt with notably frothy secretions, and placed on propofol for sedation. Nitroglycerin drip was continued, lasix given, and the patient later had resolution of hypertension and SCAPE concerns and nitroglycerin was discontinued. Serial EKGs showed improvement in AVR ST elevation and ST depressions, though persistent, much improved upon transport to CCU.

The patient went for non-emergent cardiac catheterization later in the day and was found to have severe triple vessel disease: 95% stenosis of the proximal LAD, 90% stenosis of the proximal RCA, and 100% stenosis of the RPDA with left to right collaterals. The patient was then transferred to another facility for surgical evaluation for Coronary Artery Bypass Graft vs. high-risk PCI management.

The patient went for high-risk PCI, LAD was stented, and he followed up with his primary care doctor 3 weeks after this initial emergency visit.

References available online.

INDICATIONS OF THROMBOLYTICS

Thrombolytic therapy is indicated for ST-segment elevation myocardial infarction (STEMI) when primary percutaneous coronary intervention (PCI) is unavailable within 120 minutes of first medical contact. This is considered most effective when administered within the first 3 hours of symptom onset. Even if PCI is not feasible, thrombolytics can be considered up to 12 hours of contact. Candidates for thrombolysis must have ST-segment elevation in at least two contiguous leads or a new left bundle branch block (LBBB) with a high suspicion of infarction. Prompt administration, ideally within 30 minutes of hospital arrival, improves outcomes by restoring coronary perfusion.[9]

However, thrombolytics are contraindicated in patients with high bleeding risks, such as those with a history of hemorrhagic stroke, active internal bleeding, recent major trauma or surgery, or severe uncontrolled hypertension (>180/110 mmHg). While thrombolysis is an important alternative to PCI, patients should be transferred to a PCI-capable center for further evaluation and possible rescue PCI if thrombolysis fails.[9]

MANAGEMENT OF INFERIOR MI/ CARDIOGENIC SHOCK

Cardiogenic shock is a life-threatening condition, causing decreased end organ perfusion due to lack of cardiac output. The main cause of cardiogenic shock is cardiac dysfunction secondary to acute myocardial infarction. Patients may be noted to have hypotension; however, recent studies have even noted that hypotension may not always be noted. Decreased cardiac output is the main driver for this type of shock.[5]

Inotropic agents may be required to increase cardiac contractility, or vasoactive support for hypotension. First line of vasopressors may be norepinephrine, followed by vasopressin. Epinephrine also has some utility in cases when the above fails, and systemic resistance continues to be low as well as having an added inotropic effect. Phenylephrine is alternatively preferred by some cardiologists for hypotension.

Dobutamine is another common agent that can be used as an adjunct. Dobutamine is known as an inodilator; acting as a beta agonist, it increases heart rate but also inhibits cAMP breakdown leading to vasodilation. The idea of vasodilation is important, as it counteracts the compensatory mechanism of vasoconstriction seen with decreased cardiac output; this increases afterload, leading to worsening cardiac output. Adding this agent can help maintain cardiac output, which is essential in cardiogenic shock while knowing these side effects. In similar fashion, milrinone can also be added with the known side effect of hypotension as well. Milrinone works via inhibiting phosphodiesterase-3, which is found specifically at the cardiac myocytes. This in turn increases cAMP, leading to increased cardiac contractility and lusitropy; the perfect inotropic agent adding in both systole and diastole. And even works distally to help with vasodilation.[7] Studies have shown both these agents have no different effects on morbidity and mortality when used in the setting of cardiogenic shock.[8]

> **“However, thrombolytics are contraindicated in patients with high bleeding risks, such as those with a history of hemorrhagic stroke, active internal bleeding, recent major trauma or surgery, or severe uncontrolled hypertension.”**

If all fails, these patients may require ECMO or further mechanical circulatory support including an intra-aortic balloon pump to help further decompress cardiac function and improve coronary perfusion. Patients in cardiogenic shock are associated with a mortality rate of 50%. Emergency physicians need to recognize this disease process as early intervention is crucial for patient outcomes.

CONCLUSION

Managing cardiogenic shock in the emergency setting requires a rapid, systematic approach to stabilize hemodynamics and address the underlying cause. Emergency medicine physicians play a crucial role in early recognition, initiation of vasopressor and inotropic support, and coordination of advanced therapies such as mechanical circulatory support and percutaneous coronary intervention (PCI). When PCI is unavailable, thrombolytic therapy serves as a critical alternative in cases of cardiogenic shock secondary to acute myocardial infarction, though its risks must be carefully weighed against its benefits. The decision to administer thrombolytics requires thorough patient evaluation, considering contraindications and the urgency of reperfusion. Ultimately, a multidisciplinary approach, integrating emergency, cardiology, and critical care teams, is essential to optimizing survival and improving patient outcomes in cardiogenic shock. ✶

References available online.

POCUS FOR THE WIN

Point-of-Care Ultrasound for the Diagnosis of Acute Achilles Tendon Rupture

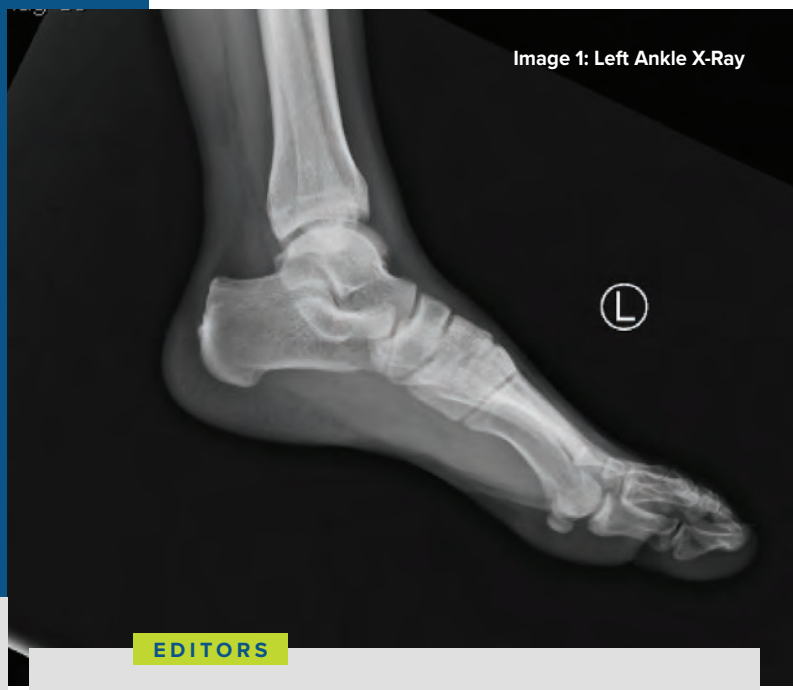
Image 1: Left Ankle X-Ray

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HISTORY OF PRESENT ILLNESS

A 35-year-old male with no significant past medical history presented to the emergency department (ED) with left calf pain that started while playing basketball that evening. The patient described feeling a sudden "pop" in his distal left calf, immediately followed by sharp pain and inability to bear weight. He denied any other injuries, falls, loss of consciousness, or head trauma.

PHYSICAL EXAM

His vital signs were a blood pressure of 118/66 mmHg, heart rate of 111 bpm, temperature of 99.2°F, respiratory rate of 18 breaths/minute, and SpO2 96% on room air. He appeared in no acute distress and had no obvious external signs of traumatic injury. His exam was notable for tenderness to palpation at the distal posterior left gastrocnemius along the Achilles tendon. He was unable to actively dorsiflex his foot, however sensation was intact throughout the lower extremity. Dorsalis pedis and posterior tibial pulses were palpable and 2+ bilaterally. The Thompson's test was positive, with no plantarflexion observed upon squeezing the left calf while lying prone, suggesting an acute Achilles tendon rupture.

LABS AND IMAGING

X-rays were performed of the left foot and ankle (Image 1). Both were negative for acute fracture, dislocation, or foreign body.

POINT OF CARE ULTRASOUND

Point-of-care ultrasound (POCUS) was performed to evaluate the integrity of the Achilles tendon. Using the linear probe in transverse and sagittal orientations, a disruption of the tendon was visualized with a surrounding anechoic fluid collection consistent with a hematoma, indicative of acute tendon tear. (Image 2,3)

DISCUSSION

The Achilles tendon is the most commonly ruptured tendon, with 40 cases per 100,000 annually.[1] This case highlights the utility of bedside point-of-care ultrasound in the emergency department for diagnosing musculoskeletal injuries. Historically, Thompson's test alone has been used to diagnose Achilles tendon injuries in the ED. It has an estimated sensitivity of 96% and a specificity of 93%.[2] Despite these metrics, Achilles tendon ruptures are

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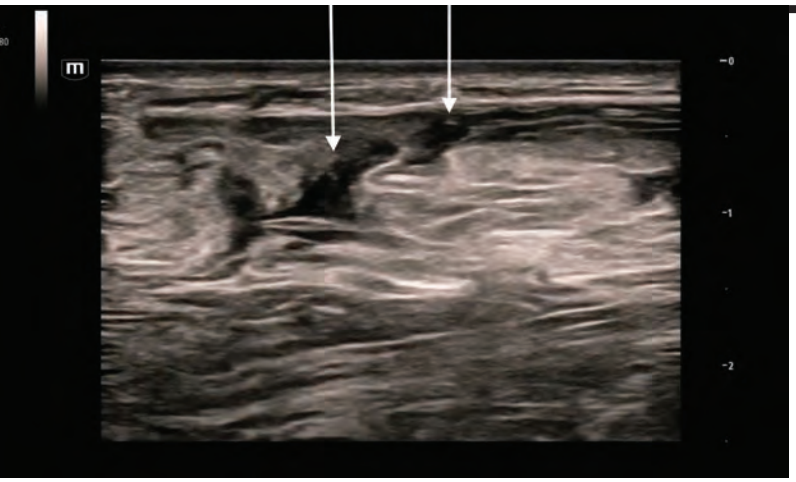

Image 2: Sagittal View—Disruption of the Tendon with a Surrounding Fluid Collection

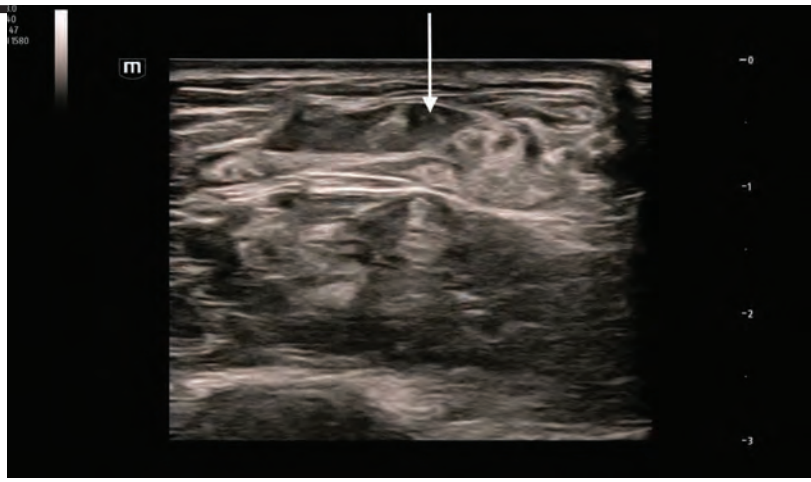

Image 3: Transverse View—Disruption of the Tendon with a Surrounding Fluid Collection

misdiagnosed as ankle sprains in up to 20–25% of cases.[3] Misdiagnosis of these injuries may result in poor functional outcomes and increased risk of re-rupture, as untreated patients continue full ankle motion, exacerbating injury severity.[4] Proper diagnosis of Achilles tendon rupture significantly impacts patient management compared to the common misdiagnosis of ankle sprain. Management options include both operative and nonoperative approaches.[5] Conventional non-operative care involves immobilization in a plantarflexed splint or 3D boot.[5]

We propose that implementing both bedside clinical exams, such as Thompson's test, and POCUS can significantly reduce the misdiagnosis of Achilles tendon ruptures. Ultrasound has been shown to have high sensitivity (94.8%) and specificity (98.7%) for diagnosing complete Achilles tendon ruptures, providing both patient and provider with a confirmed diagnosis in the ED.[4] On ultrasound, a normal Achilles tendon should appear organized with the parallel, tightly packed fibers. Injury to the tendon disrupts this organization, manifesting as loosening of the fibers, or appearing like the end of a frayed rope. Edema and bleeding appear as heterogeneous, hypoechoic changes or anechoic fluid pockets. The disruption and edema may both contribute to abnormal tendon thickening greater than 1cm in diameter. Partial ruptures may have some or all of these changes but with some fibers still intact. Complete ruptures demonstrate full loss of normal tendon structure with a visible gap, while partial ruptures typically exhibit areas of disrupted fibers interspersed with intact fibers and focal hypoechoic changes. The unaffected side should be used for comparison.[4] Although MRI is considered the gold standard for diagnosing tendon injuries and is important for operative planning, it is costly, time-consuming, and less accessible in emergency settings. Ultrasound serves as a practical and effective diagnostic tool in the emergency department, eliminating barriers to treatment and evaluation.

HOSPITAL COURSE AND CASE RESOLUTION

The patient was immobilized in a plantarflexed short leg splint. Orthopedic surgery was consulted and recommended outpatient follow up. Subsequent Orthopedic evaluation led to surgical Achilles tendon repair. The patient has been doing well post-operatively. ✶

Subacute Combined Degeneration Secondary to Nitrous Oxide Use: A Case Report

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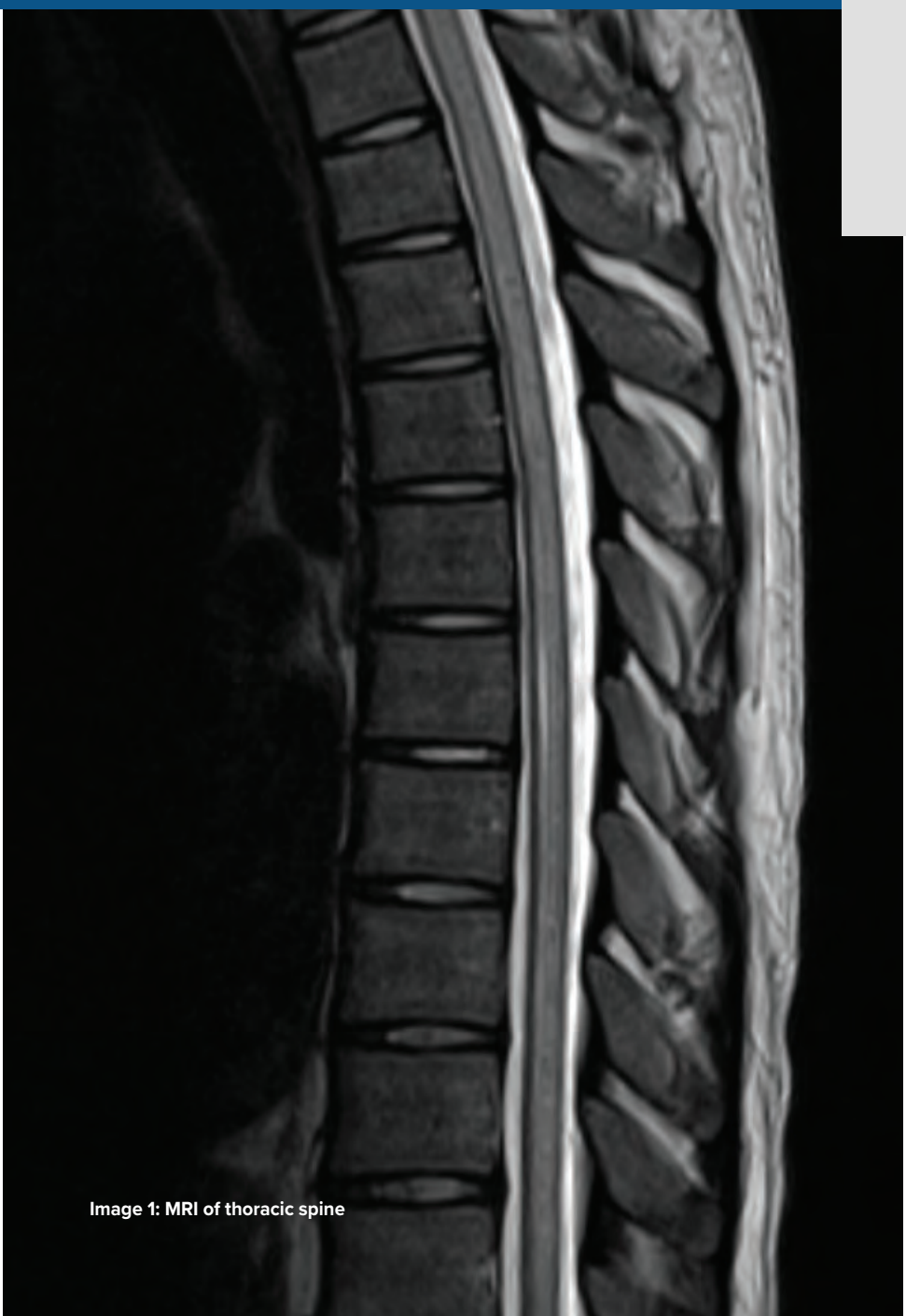


Image 1: MRI of thoracic spine

A 24-year-old male with a past medical history of cannabis use presented to the emergency department 4 times in a short period with progressive neurological symptoms.

On his first presentation, the patient reported a 4-day history of intermittent and migratory numbness of the feet and hands. He endorsed recovering from a brief respiratory illness a few weeks prior, as well as extensive recreational cannabis use. He denied a history of intravenous drug use. Serious neurologic disease, namely Guillain-Barré, was considered in the differential diagnosis. However, his physical exam was largely reassuring. He was noted to have mild patellar hyperreflexia bilaterally and a normal (downgoing) Babinski sign. Screening labs (CBC, BMP, hepatic panel) were normal. His presentation was ultimately attributed to his cannabis use, and the patient was advised to reduce his cannabis use and to follow up with Neurology for nerve conduction studies.

Two weeks later, the patient returned to the ED with a complaint of worsening numbness in his bilateral hands and feet, now described as constant. He also reported significant anxiety and requested medication for this. He described continued

marijuana use as well as newly reported recreational nitrous oxide use. He admitted this had transpired prior to his first visit, but he had not felt comfortable disclosing that information. On this visit, the patient's neurologic exam was noted to be normal, although reflexes were not specifically documented. Labs were again unremarkable. He was encouraged to utilize his Neurology referral. He was discharged with a short course of diazepam for anxiety and instructions on mindfulness and breathing exercises.

The patient re-presented 5 days later with a chief complaint of numbness and weakness. Unfortunately, due to high patient volumes and boarding, he did not receive a full evaluation before leaving the emergency department, but he did undergo a medical screening exam. He reported that he was now having weakness and difficulty walking, which he attributed to complete loss of sensation in his feet. The patient was concerned for vitamin B12 deficiency based on his internet searching and requested a B12 shot. The patient did not have a physical exam documented on this visit (the facility's medical screening note template did not include physical exam findings). The patient was given a vitamin B12 shot, per his request.

On the patient's fourth emergency department encounter, 23 days after his initial presentation, he returned in a wheelchair with chief complaints of dizziness, decreased sensation, and muscle tightness. He stated that he "didn't know where his feet were" and had been utilizing a wheelchair for a few days due to his inability to ambulate. The patient was more forthcoming on this visit and stated that his symptoms actually began 1 month prior, after a night of heavy nitrous oxide use. Physical exam was remarkable for increased tone in the distal extremities, loss of proprioception and vibratory sense from C8 downward, positive Hoffman sign, 10-12 beat clonus at the ankles bilaterally, upper and lower extremity hyperreflexia, and somewhat diminished pinprick sensation on the face. A broad workup was initiated, including MRIs of the cervical and thoracic spine. These demonstrated findings of subacute combined degeneration of the dorsal column through a significant portion of both the cervical and thoracic spinal cord, raising concern for acute myelopathy related to nitrous oxide toxicity. Neurology was consulted from the emergency department and recommended admission to the hospital for further workup.

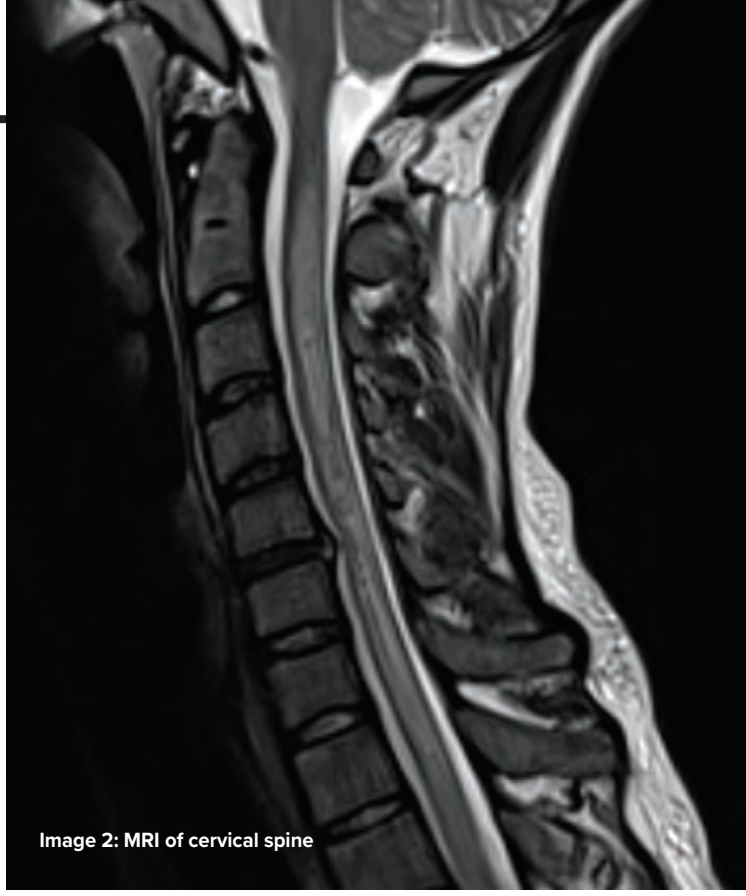


Image 2: MRI of cervical spine

CASE MANAGEMENT AND RESOLUTION

During the inpatient course of our patient, additional history regarding his nitrous oxide use was obtained. He reported using nitrous oxide on a daily basis for approximately 2 weeks prior to symptom onset, and stopped his use upon noticing his initial symptoms of extremity numbness.

Neurology documented even more extensive neurologic damage on their exam, including diminished sensation distal to the level of the clavicles, spastic tone in upper and lower extremities, spastic and wide-based gait, and intermittent urinary retention requiring catheterization.

During his hospital stay, the patient received daily cyanocobalamin injections but saw no significant neurological improvement. He was ultimately discharged from the hospital to inpatient rehabilitation for treatment of substance use disorder as well as for extensive physical/occupational therapy. The patient was subsequently lost to follow up.

DIFFERENTIAL DIAGNOSIS DISCUSSION

The differential diagnosis for diffuse peripheral numbness, an outline for which is depicted in Table 1, is quite broad. If an underlying neuropathy is suspected, one of the most important distinctions to make is whether symptoms originate from the central or peripheral nervous system.

When motor symptoms are present, findings on physical exam are particularly useful. The presence of hyperreflexia, spasticity, and an upgoing (abnormal) Babinski, which were present in this patient, imply involvement of the brain and/or spinal cord. Hyporeflexia, flaccidity, and a downgoing (normal) Babinski are typical of a peripheral nervous system issue.[1] Both central and peripheral causes can be life-threatening. However, making the proper diagnosis will often rely on different modalities. When a central cause (MS, ADEM, transverse myelitis, subacute combined degeneration, etc.) is suspected, imaging, particularly MRI of the brain and/or spinal cord, is useful and can often be diagnostic.

References available online.

NEUROLOGIC	INFECTIOUS	TOXIC/METABOLIC	AUTOIMMUNE/ PARANEOPLASTIC	OTHER
Guillain-Barré Syndrome	Lyme Disease	Electrolyte Derangements	Myasthenia Gravis	Charcot-Marie-Tooth Disease
Multiple Sclerosis (MS)	Viral Myelitis	Vitamin Deficiencies	Lambert-Eaton Syndrome	Functional Neurological Disorder
Spinal Cord Compression	HIV Neuropathy	Medication Side-effect	Acute Disseminated Encephalomyelitis (ADEM)	
Transverse Myelitis	Neurosyphilis	Heavy Metal Toxicity		
Stroke		Diabetic Neuropathy		
		Alcoholic Neuropathy		

Table 1: Differential Diagnosis for Diffuse Peripheral Numbness

For this patient, the differential diagnosis initially focused on marijuana intoxication and Guillain-Barré syndrome.

Acute marijuana intoxication is associated with incoordination and chronic use has been shown to cause anxiety and hyperreflexia, though the extent of his physical exam findings, especially on later presentations, would be inconsistent with this.[2] Although the time-course of his neurological symptoms after respiratory infection would have been consistent Guillain-Barré syndrome, the hyperreflexia would have been unusual.

Guillain-Barré typically presents with areflexia or hyporeflexia; only rare cases of axonal Guillain-Barré syndrome are known to manifest with hyperreflexia, and these cases are typically preceded by a gastrointestinal infection.[3] After the second visit, when the patient admitted to using recreational nitrous oxide, the differential shifted toward this toxicity. However, the patient did not receive a complete workup for the suspected cause of symptoms until his neurologic deficits had progressed to significant dysfunction and inability to perform activities of daily living.

NITROUS OXIDE TOXICITY

Recreational abuse of nitrous oxide, colloquially known as laughing gas or whippets, is increasing in prevalence due to its widespread availability, in both retail stores and online, and is most often seen in adolescent or young adult males.[4] There are multiple mechanisms by which large doses or chronic exposure to nitrous oxide can produce toxicity. One that is well understood and potentially treatable is functional vitamin B12 deficiency.[4] Nitrous oxide inactivates cobalamin (vitamin B12), which leads to downstream irreversible blocking of methionine synthase and consequential lack of production of metabolites needed for DNA synthesis and myelin production.[5] This results in demyelination within the central and peripheral nervous systems. Clinically, this often manifests as severe neurologic complications including subacute combined degeneration (SCAD) and myelopathy. These often present just as this patient did—with paresthesia, gait instability, and weakness.[4] Laboratory work-up may elucidate low serum levels of cyanocobalamin, though it is important to note that patients can have normal levels.[6,7] If left untreated, or if not treated in a timely manner, neurologic deficits are often irreversible.[4]

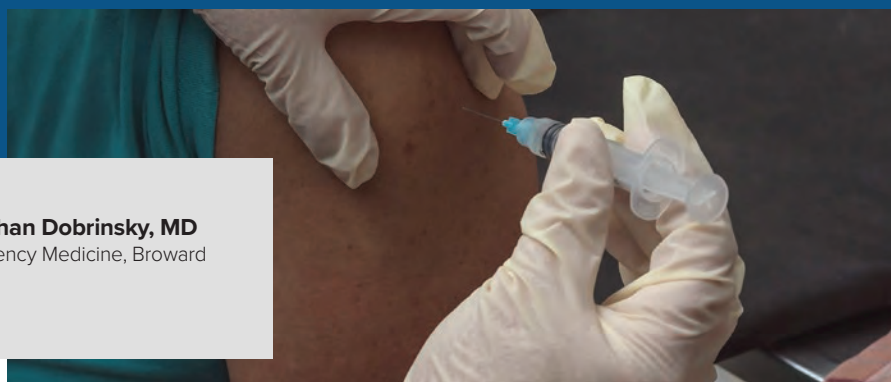
Currently, there is no defined treatment recommendation for individuals with nitrous toxicity, but the generally accepted plan is scheduled cyanocobalamin injections, as were administered to this patient.[4,6–8] Although this patient did not show improvement in an inpatient setting, other cases in the literature have demonstrated significant decrease in symptoms and occasionally full recovery with early identification, cessation of use, and aggressive B12 treatment.[7,8] Therefore, obtaining a thorough patient history and maintaining suspicion for nitrous oxide abuse is crucial for improved patient outcomes.

Nitrous oxide toxicity is difficult to diagnose due to variable reliability of patient histories and ambiguity of presentation, so this condition often goes unrecognized. Half of individuals in the United States aged 12 and older have used illicit drugs at least once.[9] Despite the significant prevalence of substance use in our population, healthcare workers have been found to exhibit implicit biases or negative attitudes toward caring for individuals who endorse substance use, as well as a lack of trust in the reliability of this patient population.[10,11] In a setting that is designed to care for emergently ill individuals, it is possible that providers may have approached this patient's encounters with some implicit biases regarding his reported marijuana use, and subsequently easily dismissed his concerns especially given his reassuring physical exam on the first three visits.

Another salient aspect of this case is the number of ED presentations this patient had before receiving an accurate diagnosis and proper treatment. A large analysis of short-term return visits to the emergency department has outlined multiple categories that drive these visits, including factors related to the patient, provider errors in diagnosis or management, healthcare system issues, and factors related to the underlying illness.[12] All of these factors were at play in this patient's case. Non-disclosure of nitrous use at his initial visit, cognitive bias of providers, high boarding resulting in the patient leaving before a complete evaluation was performed on his third visit, and ultimate progression of his symptoms during this timeframe likely all contributed to the number of ED visits preceding his diagnosis and admission. Other studies have demonstrated that patients who return to the emergency department for the same complaint are at increased risk of adverse outcomes, suggesting that a broadened work-up and closer consideration of admission may have been warranted on earlier return visits.[13] ✷

References available online.

Rethinking B52



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Acute agitation is a common presentation in the emergency department. Since the 1980s, medications used to manage agitation in the ED include first-generation antipsychotics (FGAs) such as haloperidol and droperidol.[1]

One common pharmacological practice has been to administer a drug cocktail known as a B52, named for "B" Benadryl, "5" mg haloperidol, and "2" mg of lorazepam, but the cocktail since evolved to simply "52," consisting of 50 mg of an anticholinergic (either haloperidol or droperidol) plus 2 mg of a benzodiazepine.[2] A 2022 study designed to compare the efficacy of B52 vs. 52 concluded that both combinations infrequently required repeat agitation medication, while the B52 combination resulted in more oxygen desaturation, hypotension, physical restraint use, and longer length of stay.[3] Other controlled studies to examine this protocol have been limited, with inconclusive results, likely due to the complexity of the evidence. These studies have compared various combinations of medications, different doses, and different routes of administration.

Absent a compelling body of research, sedating medication continues to be directed by inherited or local practice protocol rather than by evidence, with too many sites persisting with the use of haloperidol and other FGAs.[1,4]

Recent literature has focused on the management of agitation in specific populations, novel pharmacologic agents, and a reconsideration of previously utilized medications (e.g., B52, haloperidol, and droperidol).[1,5] Newer and equally potent pharmaceutical agents and treatment protocols with better side effect profiles that reduce the risk for oversedation, dystonic reactions, and QT prolongation are available. Best practice guidelines have recommended these approaches over the traditional reliance on the B52 cocktail, haloperidol, and droperidol.[1,5] In a 2013 Cochrane Review of 25 haloperidol studies, the authors put forth this recommendation: "Haloperidol is a potent antipsychotic drug with a high propensity to cause adverse effects.... when available, clinicians may wish to prescribe an alternative antipsychotic with less likelihood of adverse effects such as Parkinsonism, akathisia, and acute dystonias."[1]

Concerns about droperidol with regards to QTc interval prolongation and subsequent risk of developing torsades de pointes led to a 2001 FDA Black Box Warning calling into question its widespread use.[6]

Multiple studies published since the FDA warning have confirmed the safety and efficacy of low-dose droperidol for the treatment of nausea, vomiting, migraine headaches, and agitation in the ED.[6,7] Droperidol at 2.5 mg IV was found to be superior to prochlorperazine at 10 mg IV for migraine control, with doses of up to 8.25 mg IM found to be superior to placebo for migraines without inducing any QT prolongation. Higher doses (greater than or equal to 10 mg) of droperidol have been used safely, with no evidence for risk for QT prolongation, for acute undifferentiated agitation in ED patients.[6,8] A 2015 study of 1009 patients supported the use of high-dose droperidol for the treatment of acute behavioral disturbance in the ED with no evidence of increased QT prolongation.[8] The ACEP 2021 policy statement on the basis of numerous randomized controlled trials, prospective observational studies, and a systematic review from 2018 confirmed the safety and efficacy of droperidol for acute psychosis-induced agitation. An intramuscular dose of up to 10 mg of droperidol appears to be as safe and possibly more effective than other medications used for acute agitation. There were no reports of increased cardiac or respiratory events in any of the droperidol trials. It was concluded that droperidol provides effective treatment for acute agitation in the ED.[6]

The FDA has agreed that the current literature does not support the mandate of electrocardiogram prior to administration of droperidol or telemetry monitoring for doses <2.5 mg IV. Additionally, there should be no restrictions for use of higher doses of droperidol in the ED, provided cardiac monitoring is available soon after IV administration for high-risk patients. For agitated psychosis based on the extensive literature supporting the safety of droperidol, ACEP recommends the continued use of droperidol, at even higher doses, starting at 5–10 mg IM or IV, and up to 20 mg, regardless of initial monitoring capability or EKG. It is further recommended that the FDA black box warning be revised to reflect current data regarding the safety and efficacy of droperidol.[6]

Now considered a safe and effective treatment for acute agitation in the ED, droperidol has been studied for its efficacy and safety when compared to

References available online.

haloperidol. A randomized, controlled trial conducted in Australia comparing IM haloperidol and IM droperidol showed the risk for adverse effects was low in both groups, with no cardiac dysrhythmias reported.[9] Another study comparing IM haloperidol and IM droperidol found that IM droperidol decreased combativeness significantly compared with IM haloperidol at 10, 15, and 30 minutes. There was no significant difference between the two drugs when given IV.[10]

Informed by the results of current literature regarding the efficacy and safety of droperidol, further efforts to move forward have been advanced by revised clinical policy from ACEP aiming to clarify the often indirect evidence and seeking to guide clinicians in choosing the most effective parenteral agents for patients with severe agitation.[4,6] The clinical policy gives a Level B recommendation to the use of a combination of droperidol and midazolam (or another atypical antipsychotic plus midazolam) for "rapid and efficacious treatment of severe agitation." The combination of droperidol and midazolam appears to result in rapid sedation, requires fewer additional medications, and has a favorable safety profile in agitated ED patients. A randomized study found that droperidol (5 mg IV) plus midazolam (5 mg IV) resulted in a higher proportion of patients adequately sedated at 10 minutes compared with droperidol (10 mg IV) or olanzapine (10 mg IV) alone. Similarly, another randomized study found that a combination of droperidol (5 mg IV) with midazolam or olanzapine (5 mg IV) resulted in quicker time to adequate sedation than IV midazolam alone. Both droperidol and olanzapine appear to work more quickly than haloperidol. A 2018 study found more patients were adequately sedated at 15 minutes with 5 mg of IM midazolam compared with haloperidol, 5 mg, haloperidol 10 mg, and ziprasidone 20 mg.[4,6]

ACEP policy recommendations provide a Level C consensus recommendation for the use of ketamine (commonly used in procedural sedation and induction setting), as a novel therapy for analgesia and severe agitation and delirium in the ED, in critical circumstances, "in situations where the safety of the patient, bystanders, or staff is a concern."[6,12] Ketamine is a highly dissociative sedative that provides effective low-dose analgesia, procedural sedation, and general sedation.[12] Rapid dissociation, favorable cardiovascular stability, and preservation of respiratory drive suggests that ketamine may be an option for the safe control of agitated and violent ED patients.[12] Studies examining the safety of ketamine used for pain and agitation in the ED have described the efficacy and safety for pain with dosing regimens of 0.1–0.3 mg/kg, (IV), and for dissociative sedation ketamine, dosing is defined as 3–5 mg/kg, (IM). IM ketamine provided significantly shorter time to adequate sedation than a combination of IM midazolam and haloperidol;[13] and when used for dissociative sedation for severe agitation, ketamine led to less endotracheal intubation than reported in the prehospital literature.[12] Currently, ACEP recommends the use of ketamine, at a dose of 3 to 5 mg/kg (IM), achieving sedation in 2-10 minutes with a single dose in situations where patient and staff safety is the overarching concern. It is critical, however, to be cognizant of and prepared for the potential for serious adverse effects including respiratory depression and laryngospasm.[4,6,12,13]

Safer and more efficacious protocols for the management of acute agitation in the ED have also been advanced by the American Association for Emergency Psychiatry's (AAEP) 2012 Project BETA, tasked to set forth best practice guidelines for the evaluation and management of agitation.[5]

In the decade that followed, those guidelines have been expanded with new recommendations, summarized in Table 1.

Despite the assumption that B52 combination therapy is commonly used in the ED, there is little research to support its efficacious use.[2] Concerns about the risk of oversedation, variable patient response, and potential side effects (extrapyramidal symptoms, anticholinergic effects, dependency, and paradoxical reactions) have inhibited the widespread use of the B52 or 52 cocktails in the ED. Alternative pharmacological management protocols endorsed and promulgated by best practice guidelines have been found to be safer and more efficacious in the management of severe agitation than many first-generation antipsychotics (FGAs) for the emergency management of delirium, agitation due to alcohol intoxication, agitation due to a known psychiatric disorder, and undifferentiated presentations with prominent psychotic symptoms. Second-generation antipsychotics (SGAs) such as olanzapine, ziprasidone, and risperidone, are often favored over FGAs such as haloperidol and droperidol. Both classes have similar efficacy in managing agitation, but many FGAs pose a greater risk for adverse events/effects on patient health.[1,5] ✶

SUSPECTED ETIOLOGY	MEDICATION AND DOSE	COMMENTS
Primary psychiatric or undifferentiated with prominent psychosis	Olanzapine, 5 mg-10 mg (PO, ODT or IM); risperidone, 2 mg (PO, ODT or liquid); ziprasidone, 10 mg-20 mg (IM); droperidol, 5 mg-10 mg (IM)	Add lorazepam 1 mg-2 mg if haloperidol or mono therapy ineffective; risk of respiratory depression for IM olanzapine and IM lorazepam within 1 hour
Intoxication with central nervous system depressant (including alcohol)	Haloperidol, 2 mg-10 mg (PO or IM); olanzapine, 5 mg (PO); risperidone, 2 mg (PO, ODT, or liquid)	Avoid benzodiazepines if possible; FGAs still heavily favored, but SGAs likely sage if given PO; IM risks respiratory depression
Stimulant intoxication, alcohol or benzodiazepine withdrawal OR undifferentiated without psychosis	Lorazepam, 1 mg-2 mg (PO or IM); diazepam, 5 mg-10 mg (PO); midazolam, 5 mg-10 mg (IM)	Add SGA if prominent psychotic symptoms

EPS, extrapyramidal symptoms; FGA, first-generation antipsychotic; IM, intramuscular; ODT, oral dissolvable tablet; PO, oral pill; SGA, second-generation antipsychotic

Table 1. AAEP Guidelines for ED Management of Acute Agitation

References available online.

A Self-Contained Hypothermic Rewarming System: A Case Study

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Hypothermia is a serious medical condition that affects urban and rural populations. An estimated 1,500 patients in the United States die from hypothermia each year.[1] Hypothermia (defined by core temperature below 35°C) occurs when heat loss exceeds heat production.[1] Humans can adapt to heat exposure, but compensatory measures are limited in cold exposures.[2] Hypothermia is classified into four categories based on core body temperature: mild (35°C-32°C), moderate (32°C-28°C), severe (<28°C), and profound (<24°C)[1,3] with acuity increasing as temperature decreases.

Mild hypothermia results in dehydration due to diuresis caused by a central volume shift.[2] Moderate hypothermia can precipitate cardiac conduction abnormalities, including bradycardia, atrial fibrillation, premature atrial and ventricular beats, and electrocardiogram changes such as QT prolongation and Osborn waves.[2] Severe hypothermia leads to bradycardia resulting from decreased function of the sinoatrial node, shifts in oxygen and electrolyte concentrations, and pH changes of the tissues.[2] Bradycardia can exacerbate hypotension, which increases the risk of coma, cardiac dysrhythmias, and resultant cardiac arrest.[4] Cardiac arrest secondary to profound hypothermia can be uniquely challenging as guidelines recommend rewarming to a near-normal core body temperature prior to ceasing resuscitative efforts.

Rewarming techniques include active internal warming measures such as warmed and humidified oxygen therapy via endotracheal tube, thoracic lavage via chest tubes, peritoneal and/or bladder lavage, warmed IV fluids, and external warming measures such as heated blankets or external targeted temperature management devices. Resuscitation of the severely hypothermic patient is often prolonged and inefficient, requiring a significant amount of dedicated personnel with time to achieve desired core temperatures of 33°C-34°C.[5,6] Because resuscitations can be quite burdensome on the limited staff in community EDs, the prolonged resuscitation time can lead to poor patient outcomes. This leaves a need for a more efficient rewarming method for these hypothermic cardiac arrest patients in hospitals without extracorporeal membrane oxygenation (ECMO) capability and limited staff.

The most efficacious and rapid rewarming technique for severe or profound hypothermia is ECMO.[7] However, ECMO is resource-draining and limited in availability, requiring trained personnel, blood product transfusion, ICU monitoring, and frequent laboratory monitoring.[8] Due to the limited availability of ECMO-capable centers and challenges associated with transferring a patient in cardiac arrest, ECMO is often not realistic. More commonly utilized methods of resuscitation include modified ACLS protocol with continuous CPR until the patient has been rewarmed to 30°C prior to continuation of ACLS medications and defibrillation.

Studies have shown that using intravascular rewarming catheters could be more effective and less invasive than traditional rewarming measures.[9,10,11] Other studies have shown that rewarming rates are comparable between traditional rewarming measures and endovascular rewarming, with no increased risk of complications in patients with endovascular rewarming.[12] In addition, the use of endovascular rewarming simultaneously provides physicians with the capability to give vasoactive medications and rapidly correct hypovolemia.[12] While this is a novel technique that requires more investigation into its efficacy, preliminary data suggest that these catheters are highly effective in hypothermic rewarming.[11] It has been shown that the more hypothermic a patient is, the faster this modality can rewarm them, but as the patient approaches normal body temperature, the warming rate decreases because of peripheral vasoconstriction on heat distribution.[11] The use of intravascular rewarming catheters is also less invasive than other measures such as peritoneal lavage, thoracic lavage, and ECMO.[9,10,11] This system also has the potential to alleviate the personnel burden.[11] Some concern has been raised

References available online.

about the incidence of thromboembolism when rewarming hypothermic patients with targeted temperature management catheters.[13,14] However, this is a risk of any central venous catheter, and studies have shown that the placement of IVC filters prior to removal of these catheters later in the patient's care can alleviate this risk.[13,14] Other studies have shown that these endovascular rewarming catheters are very safe and do not significantly increase complication rates in patients at all.[12]

CASE PRESENTATION

Between December 2022 and December 2023, two patients presented to affiliated local EDs in hypothermic cardiac arrest, during which intravascular warming devices were utilized in the resuscitation. Patient A was a 30-year-old male who presented via EMS after being found down. He was initially noted to have a weak pulse and agonal respirations, unresponsive to naloxone, but lost pulse when transferred to the EMS stretcher. Cardiac rhythms en route included initial ventricular fibrillation followed by pulseless electrical activity that persisted until arrival at the emergency department. Upon arrival at the ED, the patient had a core body temperature of 26.5°C by temperature-sensing urinary catheter. Patient B was a 38-year-old male who presented via EMS after being found down. He was found outside of a gas station and had requested help from an employee shortly before EMS arrival. Upon EMS arrival, the patient was pulseless, apneic, and unresponsive to naloxone. EMS detected ventricular fibrillation, asystole, and then pulseless electrical activity, which persisted until arrival at the emergency department. Upon arrival, his core temperature was 29.1°C by temperature sensing urinary catheter.

Both patients were "found down" in cold environments with initial resuscitation attempts by EMS, including naloxone and ACLS protocol with CPR, defibrillation, and medications. In both cases, supraglottic airways were placed during transport to the emergency department. Patient A was transported to a single attending coverage community department, while Patient B was transported to a university hospital staffed by double coverage residents and attendings.

Upon arrival, Patient A underwent continuation of CPR and resuscitation. The team replaced the supraglottic airway with an 8.0 endotracheal tube (ETT) and utilized a mechanical chest compression device to provide uninterrupted, effective CPR and to reduce the burden on the limited staff. A temperature-sensing

References available online.

urinary catheter monitored progress while external warming measures, including warm blankets and a forced-air patient warming device, were used. Additionally, warm intravenous fluids were infused. Four 28 French chest tubes were placed in an anterior/posterior configuration bilaterally, with the anterior tubes infused with microwaved bags of warmed saline (in the absence of a fluid warmer) connected via IV tubing and the posterior tubes connected to wall suction, eliminating the need for personnel to manually flush the chest tubes. Despite these measures, the core temperature rose 2.9°C in the first 111 minutes. During the initial rewarming process, the team obtained a ZOLL Quattro Intravascular Temperature Management catheter. They inserted the Quattro into the patient's right femoral vein with a target temperature of 37°C. The patient achieved a core temperature of 34°C, rising an additional 4.6°C 140 minutes after placement of the rewarming catheter. Despite following ACLS protocol (with medications), the patient remained in asystole after two rounds of ACLS and was pronounced dead.

Upon arrival of Patient B, the team continued CPR and resuscitation, noting rhythm changes, including asystole, ventricular fibrillation with two shocks, and pulseless electrical activity, prior to determining the patient's core temperature to be 29.1°C. The patient's supraglottic airway was replaced with a 7.5 ETT, and CPR was continued using a mechanical chest compression device. Warm blankets and a forced-air patient warming device were placed, warm intravenous fluids were transfused, and warm humidified air was provided via ETT. A right femoral arterial line and central venous catheter were placed to aid in management. Four chest tubes were placed with a 28 and 32 French placed in an anterior/posterior configuration bilaterally, with the anterior tubes being infused with warm fluids using a massive transfusion warming device and the posterior tubes connected to low wall suction. In the initial 152 minutes, Patient B's core temperature rose 0.7°C to 29.8°C. At this time, a ZOLL Quattro Intravascular Temperature Management catheter was procured from a neighboring hospital and used to replace the right femoral central line, with a target temperature of 38°C. In the following 133 minutes, the core temperature rose an additional 2.5°C to a temperature of 32.3°C. The team then initiated ACLS protocol for two rounds, but the patient remained in asystole and was pronounced dead.

DISCUSSION

These two cases presented to separate facilities, a single-coverage community hospital and a double-coverage resident and attending academic facility. Neither ED stocked the ZOLL Quattro Intravascular Temperature Management catheter. Both cases had rapid improvement in the rate of rewarming after insertion of the temperature management device. Patient A rose 2.9°C in the first 111 minutes (0.026°C/minute) and an additional 4.6°C in the next 140 minutes (0.033°C/minute) after placement of the rewarming catheter. Patient B's temperature rose 0.7°C in the initial 152 minutes (0.005°C/minute) and an additional 2.5°C in the next 133 minutes after the placement of the catheter (0.019°C/minute).

The use of the targeted temperature management devices reduced the number of hospital personnel required at the bedside throughout the prolonged resuscitations. Also, the device is placed like a central line. As such, no additional training is needed for emergency physicians to incorporate this system into their practice. Once the intravascular catheter was placed in patient A, the resuscitation required one nurse for monitoring and documentation purposes and one technician who assisted in warming fluid to be used in the intrathoracic lavage through bilateral chest tubes. The attending continued to see other patients, allowing for improvement in the patient care and overall ED flow.

Once the intravascular catheter was placed in patient B, one nurse continued warm fluids intravascularly via massive transfusion warming device for intrathoracic lavage as needed. The use of the intravascular rewarming device has major implications for personnel, including physicians, nurses, and technicians. The resource-sparing component of these catheters could be particularly advantageous in smaller facilities with limited staff.

The impact of this system on personnel burden alone has the potential to improve patient care significantly. It allows physicians and other staff to care for other patients while simultaneously rewarming the hypothermic patient. This could decrease ED wait times, which in turn could decrease poor outcomes related to delays in care. This system serves as a viable option that should be considered, especially in resource-limited community EDs. It is highly effective in rewarming and is a less invasive technique than many traditional warming measures.

This two-patient case series on the use of intravascular temperature management devices supports prior research on rewarming critically ill patients with moderate to severe hypothermia to improve their outcomes (REFs). An effective ZOLL or similar catheter could obviate the need for nasogastric and thoracostomy tubes while facilitating more rapid rewarming. Because this system can be used with forced-air patient warming devices, warm fluids, and other external warming measures, it shows considerable promise in maximizing the rewarming potential in moderate to severe hypothermia patients. With more research on its efficacy, this endovascular rewarming system could serve as an equally effective yet less invasive method than ECMO while simultaneously decreasing personnel and resource burden in emergency departments all over the country. ✳

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Alcohol-Induced Narrow Complex Tachycardia

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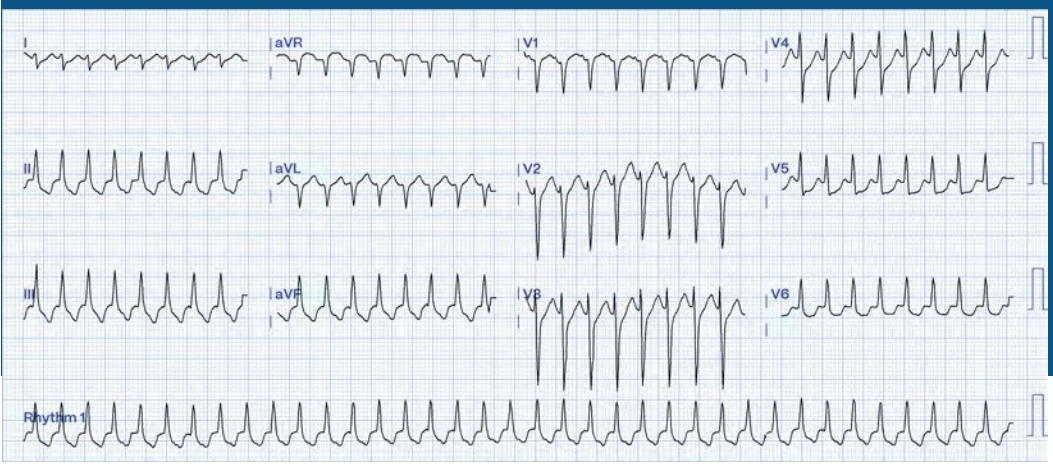

Figure 1. Initial ECG

CASE

A 57-year-old man presented to the emergency department with palpitations and a sensation of nervousness after alcohol consumption. He had no history of angina or myocardial infarction.

On examination, his blood pressure was unmeasurable due to hemodynamic instability. Oxygen saturation was 98% on pulse oximetry, and cardiac monitoring showed a regular tachycardia at 240 beats per minute. (Figure 1)

After the patient underwent synchronized cardioversion, sinus rhythm was immediately restored. (Figure 2)

Further evaluation was performed, revealing hypokalemia with a serum potassium level of 2.9 mmol/L. Other laboratory tests, including troponin, inflammatory markers, and thyroid function were unremarkable.

DISCUSSION

The first ECG showed a regular narrow-complex tachycardia with no obvious P waves and regular R-R intervals.

The differential diagnoses include:

- Atrioventricular nodal reentrant tachycardia (AVNRT)
- Atrioventricular reentrant tachycardia (AVRT)
- Ectopic atrial tachycardia
- Atrial flutter 2:1 conduction
- Sinus tachycardia

Sinus tachycardia:

Defined by steady rhythm with P waves before each QRS complex. This is unlikely in our case. The ECG rate of 240 bpm is faster than the sinoatrial node can deliver an impulse. Moreover, there are no visible P waves preceding each QRS complex.[1]

Atrial tachycardia:

A form of SVT defined by abnormal p-wave morphology and axis, uniform p-waves, an isoelectric baseline, and normal QRS morphology.

Second, the ECG shows no distinguishable P waves, which makes atrial tachycardia less likely.

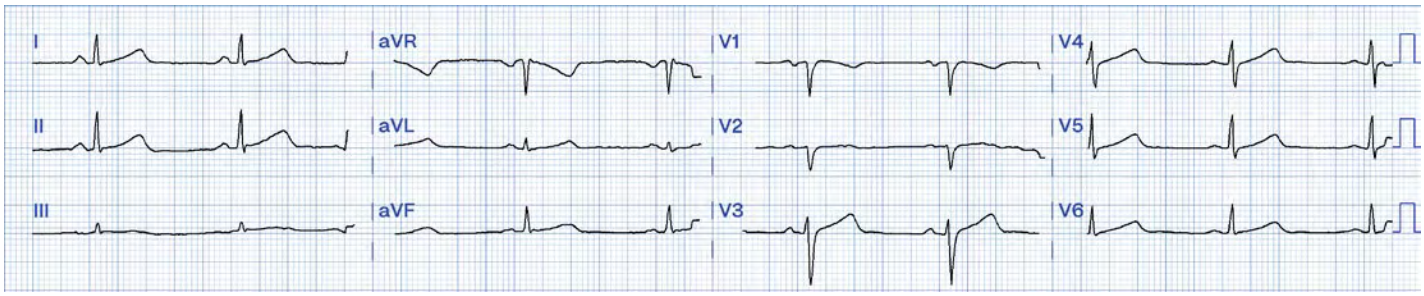

Figure 2. ECG after cardioversion

References available online.

Atrial flutter:

Defined by narrow complex tachycardia with loss of the isoelectric baseline and a "Saw-tooth" pattern of inverted flutter waves in leads II, III, aVF.

The lack of sawtooth-shaped F waves across the inferior leads and a ventricular rate of 240 beats per minute make atrial flutter 2:1 conduction less likely.

Another important consideration in tachycardia is ventricular tachycardia. However, this presents as a wide complex tachycardia, and atrioventricular (AV) dissociation and capture beats may be present. This is also unlikely given the narrow QRS complexes in our case.

The ECG findings — including a regular rhythm, narrow QRS complexes, a heart rate of 240 bpm, and P waves that may be indistinct or appear after the QRS complex—are consistent with the diagnosis of AVNRT or AVRT.

Since P waves are not visible during the tachycardia episode and the pre-hospital electrocardiogram showed no evidence of Wolff-Parkinson-White (WPW) syndrome, AVRT is unlikely.[2] Concurrently, after electrical cardioversion, delta waves characteristic of WPW syndrome remain absent. This ECG study suggests a low-fast variant of AVNRT.[3] Wolff-Parkinson-White (WPW) can alter the morphology of the QRS, or it may conceal some accessory pathways, leading to other conduction abnormalities. Therefore, a delta wave may not always be apparent on the resting ECG.

In our case, we suspect alcohol contributed to the patient's episode of AVNRT. Alcohol is not a direct cause of AVNRT, but it may act as an indirect trigger through several mechanisms, including:[4]

- Increased catecholamine levels and stimulation of the sympathetic nervous system.
- Electrolyte imbalances (hypokalemia, hypomagnesemia).

In a case-control study, numerically more individuals with SVT were observed in those who consumed >6 drinks per day; however, this was of borderline statistical significance.[6]

Individuals who consume excess amounts of alcohol can present with excessive vomiting, leading to dehydration and electrolyte imbalance, thereby increasing the risk of triggering cardiac arrhythmias. Thus, intravascular volume replacement is necessary to ensure electrolyte balance to prevent the recurrence of tachyarrhythmia.

CASE CONCLUSION

A patient presented to the emergency department with regular tachycardia, narrow complex, and no distinct P waves observed. After excluding other causes, two main diagnoses were considered: AVNRT and orthodromic AVRT. Prompt electrical cardioversion is critical in hemodynamically unstable patients, as timely intervention is essential to restore sinus rhythm and prevent further complications.

LEARNING POINTS

Recognizing regular narrow complex tachycardia: QRS complex duration <120 ms.

Differential diagnosis includes: AVNRT, AVRT, ectopic atrial tachycardia, atrial flutter 2:1 conduction, and sinus tachycardia.

Atrioventricular nodal reentrant tachycardia (AVNRT): Regular rhythm, heart rate 180-240 bpm, P waves are unclear, possibly buried within the QRS complex or appearing immediately after QRS, the most common cause of regular narrow-complex tachycardia in adults.

Alcohol as a trigger: Alcohol may act as an indirect trigger through sympathetic nervous system stimulation or electrolyte imbalance (hypokalemia, hypomagnesemia) triggering AVNRT episodes.

Urgency of treatment: Synchronized electrical cardioversion is the safest and most effective measure in hemodynamically unstable patients.

Electrolyte correction: Hypokalemia (2.9 mmol/L in this case) should be corrected immediately to prevent recurrence.

OPINION-EDITORIAL

Significance

AUTHOR

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EM Residency Class of 2025

This began as a research project about abortion. But not officially, because I am a resident physician working in a hospital system that does not perform abortions, and I would never have received IRB approval for that.

Instead, I decided to research miscarriage management, which requires the same medical knowledge as pregnancy termination. I was concerned, like others at the time, that the 2022 *Dobbs v. Jackson Women's Health Organization* decision that overturned *Roe v. Wade* would have an impact on our ability as emergency physicians to care for pregnant patients. After this court decision, my state outlawed all abortions except in cases of medical emergency, though the qualifications for such an event were vague. There were rumors from the OB/GYN residents that their attendings were no longer offering medical management for miscarriage or ectopic pregnancy and instead were taking all patients to the OR out of fear of prescribing medications that were being banned for their use in abortion care.

After a few tries, I got IRB approval and started gathering the data for my project, working on it sporadically during a research elective that doubled as "maternity leave" after the birth of my second child. In 2024, my state passed a constitutional amendment supporting a "fundamental right to reproductive freedom" and some abortion clinics are planning to reopen soon.

I finished the project and ultimately found no statistically significant differences in any of the metrics I analyzed, including ED length of stay, admission rates, and formal ultrasounds, among others. On paper, there was no change in our management of incomplete spontaneous abortion or missed abortion from 2021-2024, despite major changes to the legal landscape during that time.

Which I guess is good news? Even so, it doesn't feel like an accurate representation of the state of women's health care over the past few years. But numbers don't lie, or at least they shouldn't. So, as a trainee in the era of evidence-based medicine, I felt like my project was, at best, a waste of time and, at worst, an indication that I was overly biased in my topic choice.

Slightly disappointed, I started a new project that I hoped would represent my overall residency experience. It was a qualitative survey of women I had come to respect and love during my time in medical school and residency. I asked them to tell me about joy, specifically in the context of work. I chose the topic because it's something I've found to celebrate amid endless bad news and articles about chart-topping ED burnout scores and failed wellness initiatives. Because, quite simply, that has not been my experience.

I love my job.

I'm not naïve (something a naïve person would say, you must be thinking). Our job is emotionally and physically exhausting and getting harder all the time with changes to the healthcare system. We see horrific things daily and walk hand in hand with unfixable social problems. Part of embracing joy, for me, has involved learning to find a place for these experiences that leaves my soul mostly intact and allows me to embrace life at home with my husband and young children.

Buoyed by the project and feedback related to it, joy has helped frame the remainder of the year. In brief quiet moments in the trauma bay and interrupted conversations, I've been considering souls. Not in the metaphysical sense, but in the everyday. As in, does your work make you more or less fulfilled in the rest of your life and your relationships with others? And if work is making things worse, then why is that and how do we do better?

Maybe the answer is burnout and metrics and sleep deprivation and decision fatigue. Or maybe it's something else, something about a fundamental shift in our relationship to why and how we became doctors in the first place. So far, what I've come up with mainly is that thinking about these things tends to be helpful. Searching for and identifying joy and purpose begets more joy and purposefulness. The converse is likely true as well. ✶

References available online.

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1. An ill-appearing 6-year-old boy presents with a high fever, inspiratory stridor, accessory muscle use, and a barky cough that has worsened over the past day. His mother says he had a positive flu test 10 days ago and that he can breathe better when he is flat on his back. Nebulized racemic epinephrine is administered, after which the stridor is unchanged. His SpO2 is 90% on room air. What is the best next step in management?

A. Administer dexamethasone and repeat nebulized racemic epinephrine
B. Order intravenous broad-spectrum antibiotics and prepare for intubation
C. Provide suctioning and supplemental oxygen
D. Start oral antibiotics and order a chest x-ray

2. In addition to aspirin and heparinization, what is the most appropriate intervention for a patient with an acute ST-elevation myocardial infarction pending imminent percutaneous coronary intervention?

A. Alteplase
B. Cangrelor
C. Rivaroxaban
D. Ticagrelor

3. What is the most common presenting symptom in pulmonary embolism?

A. Chest pain
B. Dyspnea
C. Hypotension
D. Hypoxia

4. How do patients with alcoholic ketoacidosis most commonly present?

A. Abdominal pain and vomiting
B. Altered mental status due to intoxication
C. Bradypnea
D. Coma

5. Which structure is best evaluated for traumatic injury using noncontrast CT?

A. Pancreas
B. Small bowel
C. Spine
D. Stomach

PEERprep For Physicians

ANSWERS
1=B; 2=D; 3=B; 4=A; 5=C

References available online.

ECG Challenge

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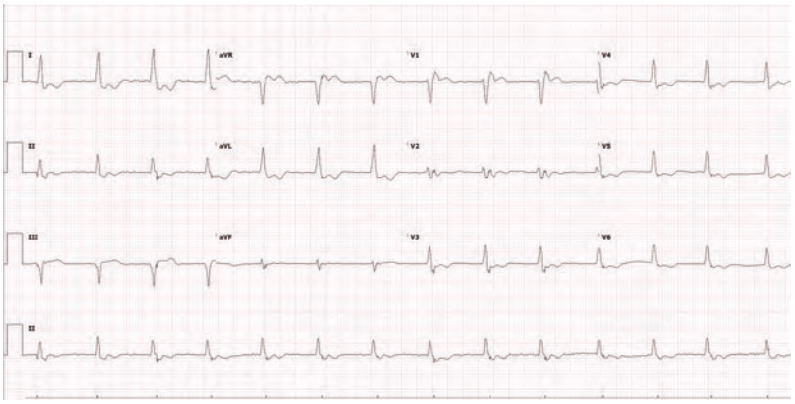
CASE

A 61-year-old male with a past medical history of permanent atrial fibrillation and pacemaker placement s/p AV node ablation presents with palpitations. What is your interpretation of his ECG?



ANSWER ON PAGE 54

References available online.

ECG Challenge

ANSWER

This ECG shows an accelerated junctional vs idioventricular rhythm at 82 bpm with underlying atrial fibrillation, normal axis, prolonged QRS complex duration with an IVCD, STE in leads III, V1, and aVR, and STD in leads I, II, aVL, and V3-V6. There is also RV pacing with failure to sense.

The absence of distinct P waves both indicates a native rhythm of atrial fibrillation and provides a clue to the mode in which the pacemaker is functioning: there are no atrial beats to sense, and so the ventricular pacing spikes cannot be atrial sensed. The pacer spikes seen at the bottom of the ECG occur regularly, suggesting the ventricles are paced at a set rate, consistent with ventricular sensed, ventricular pacing (VVI). In this mode, the pacemaker's firing should be inhibited by intrinsic ventricular activity. In this ECG, there are pacer spikes occurring within or after the native QRS complexes which means that inhibition is not occurring. This is happening because the device does not recognize the native electrical activity of the heart, and its pacing is therefore asynchronous with regard to the QRS, a characteristic finding of failure to sense. In an appropriately functioning VVI pacemaker, all pacer spikes should precede a QRS (see Figure 1) and there should be no pacer spikes if the patient's native ventricular rate exceeds the fixed ventricular rate programmed in the pacemaker.

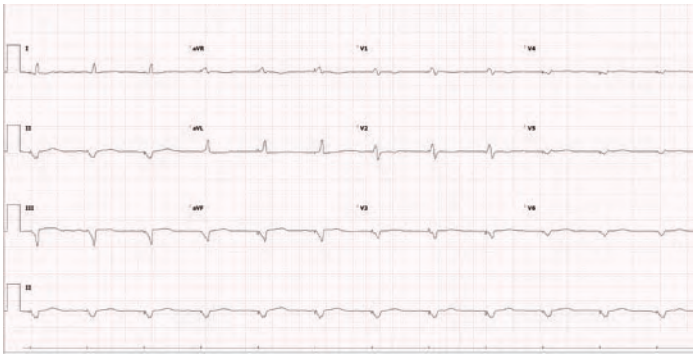


Figure 1. Baseline ECG with appropriately functioning VVI pacemaker

The etiology of failure to sense often involves changes in the physical condition of the device itself, in particular the pacer leads which are susceptible to physical fracture, failure of their insulation, and diminishment of their signal through fibrosis. Alternatively, the threshold for electrical activity may simply be set too high, such that intrinsic ventricular activity produces a signal not reaching the minimum amplitude the device is set to recognize. In other cases, the pacemaker may have been calibrated appropriately initially, but the signal the heart's electrical activity produces has diminished over time, either because of progression of underlying cardiac disease or because of a transient pharmacologic or metabolic insult (e.g., hyperkalemia).

CASE CONCLUSION

This patient was admitted to the cardiology service for interrogation and reprogramming of his pacemaker.

PACEMAKER LEARNING POINTS

Pacemaker Codes

CHAMBER(S) PACED	CHAMBER(S) SENSED	RESPONSE TO SENSING
Atrium	**A**trium	**T**riggered
Ventricle	**V**entricle	**I**nhibited
Dual chamber	**D**ual chamber	**D**ual (triggered and inhibited)
n**O**ne	n**O**ne	n**O**ne

Pacer spikes are usually visible on the EKG, either at the bottom of the EKG and/or preceding the P-wave and/or QRS complex

- **Atrial pacing:** spikes immediately precede P-waves
- **Ventricular pacing:** spikes immediately precede QRS complexes
- **Dual chamber pacing:** spikes immediately precede both P-waves and QRS complexes
- **Biventricular pacing:** 2 spikes immediately precede QRS complexes

Atrial pacing

- Pacemaker lead usually implanted in the right atrial appendage
- Results in P-waves with normal morphology

Single ventricle pacing

- Pacemaker lead usually implanted in the RV apex
- Results in a LBBB pattern in the limb leads and anteroseptal precordial leads
- The major difference between an intrinsic LBBB and a single ventricle paced rhythm is that the QRS complex will almost always be negatively oriented in leads V5-V6 with a single ventricle paced rhythm

References available online.

Biventricular pacing

- Two pacemaker leads usually implanted in the RV apex and the surface of the posterior or lateral LV
- Typically results in a narrower QRS complex than with single ventricular pacing
- Dominant R wave in lead V1 +/- V2 is common
- AICD will have a thick coil that differentiates it from a pacemaker

FAILURE TO SENSE LEARNING POINTS

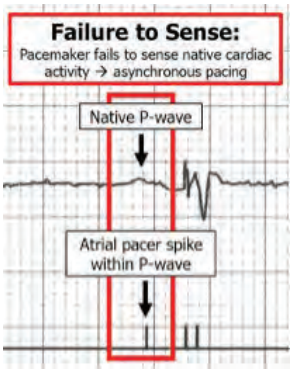
- Pacemaker fails to sense native cardiac activity -> asynchronous pacing (see Figure 2)
- Sensing refers to the pacer's ability to recognize native cardiac beats
- EKG shows pacer spikes before, after, or within P-waves and/or QRS complexes
- Causes include lead insulation break, new intrinsic bundle branch block, electrolyte abnormalities, and Class IC antidysrhythmics (e.g., flecainide)

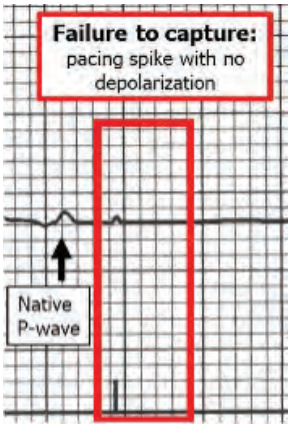
FAILURE TO CAPTURE LEARNING POINTS

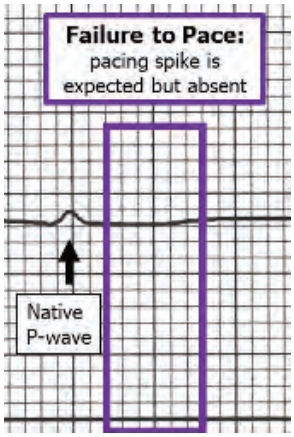
- Delivery of pacing stimulus without subsequent myocardial depolarization (see Figure 3)
- EKG shows absence of depolarization after pacer spikes
- Causes include functional (e.g., electrode displacement, wire fracture) and pathologic (e.g., electrolyte disturbances, AMI)

FAILURE TO PACE LEARNING POINTS

- Paced stimulus is not generated when expected (see Figure 4)
- EKG shows decreased or absent pacemaker function
- Causes include oversensing, lead fracture or insulation defect
- Oversensing- pacing inhibited by non-cardiac activity (e.g., skeletal muscle activity) inappropriately recognized as native cardiac activity





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