EM Resident
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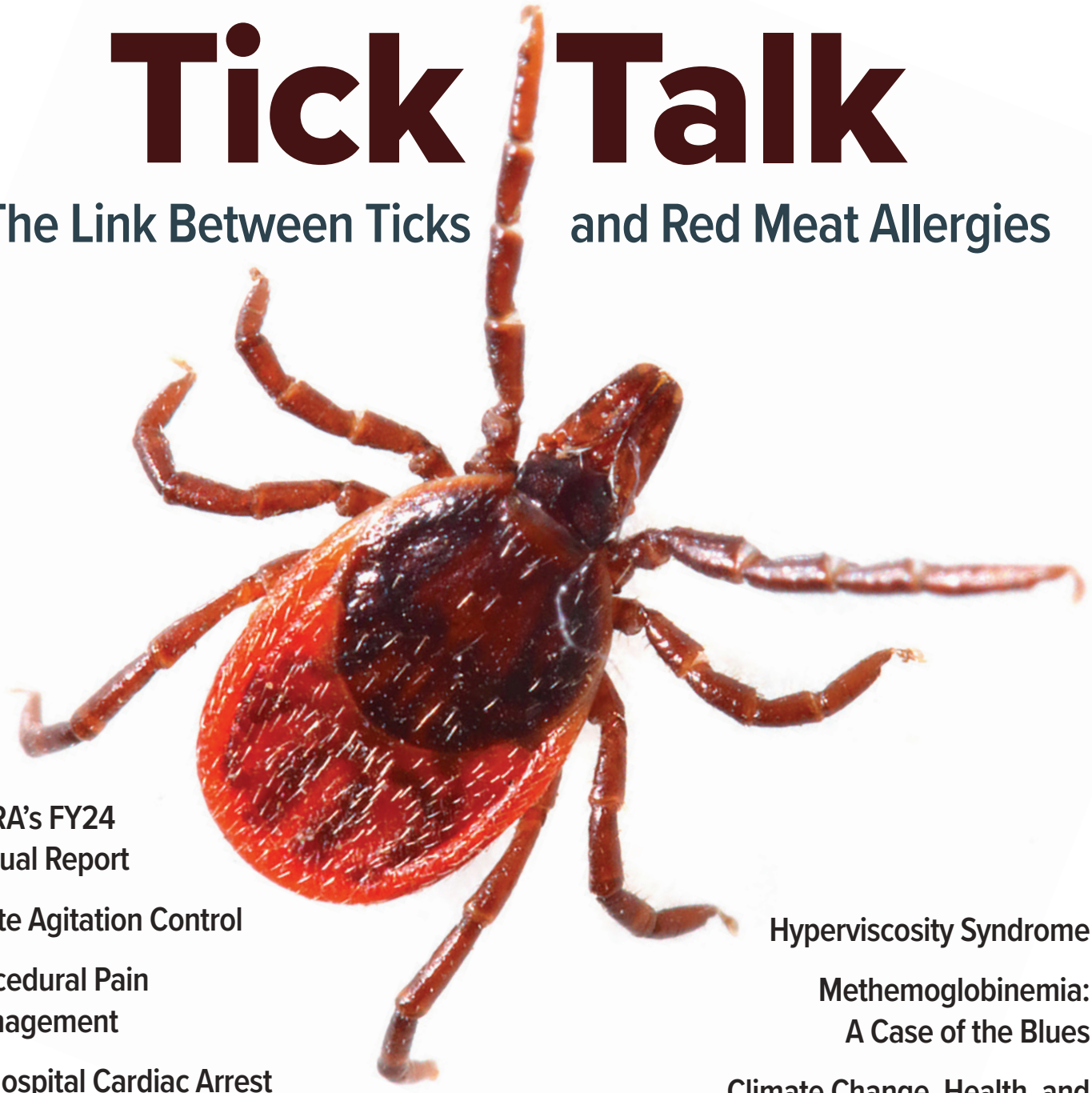
Tick Talk

The Link Between Ticks and Red Meat Allergies



EMRA's FY24 Annual Report

Acute Agitation Control

Procedural Pain Management

In-Hospital Cardiac Arrest

Becoming the Ventilated Patient

Hyperviscosity Syndrome

Methemoglobinemia: A Case of the Blues

Climate Change, Health, and the Emergency Physician

vituity®

ELEVATING CARE MEANS...

Practicing medicine with
freedom from outside ownership.

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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Morgan Sweere
MD, MPH

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

The Third Year Test

I recently began my third and final year of residency. With each passing day and shift, I am closer to reaching that goal and becoming a full-fledged emergency physician.

Last week, I went into my first moonlighting shift. I was a little bit (okay, a lot) anxious walking into a new hospital with new staff and working on my own. Somehow, I sat down at the desk, saw my first patients, and felt right at home. I didn't know where the ultrasound or the tonopen or pretty much anything else was, but I was just home. I started passing the "Third Year Test."

I recognize that some programs are 4 years, but for the sake of this article, I'll refer to all final year residents in emergency medicine as third years. When residents are entering their third year, we are experiencing a myriad of emotions. We are often tired, excited, and scared rolled all up into one. A good portion of our lives up until this point have been heavily structured for us with only a small amount of input from our own choices as far as medical training goes. At the end of this year, we finally exit and enter what feels like an abyss.

The first 2 years of residency are a whirlwind, and they provide a world of perspective when it comes to a career in emergency medicine. Unfortunately, for some, they also contribute to the beginning of burnout. Several studies cite burnout rates among third-year residents as greater than 40 percent. Residency is tough and exhausting, and there are so many factors that contribute to this. This job is not easy by any means. It may be one of the most difficult jobs there is. I still think this is the best job in the world. I couldn't imagine myself doing anything else.

We've all heard of the "3 AM Test" — the test we all had to pass as medical students trying to match into our dream emergency medicine residency. Essentially, you have to be a person who is interesting, personable, and normal enough that other people would want you working beside them at 3 am, when they are their most tired and at a sometimes dull portion of their shift.

I think I'd equate this test to what I would call the "Third Year Test." You are at the end of your residency and your most exhausted, crispy, ready-to-go self you have ever been since starting your medical training. You cannot wait to spend days outside of the hospital walls. If you love this specialty and love coming to work on those days, then I believe you'll love it forever.

I think this is the test that makes me realize I chose the correct specialty. Obviously, I am just a third-year resident, so I can't account for the experience of those who have had long careers and may feel differently. I can only account for my own experiences, thoughts, and hopes for a long, happy, fulfilling career.

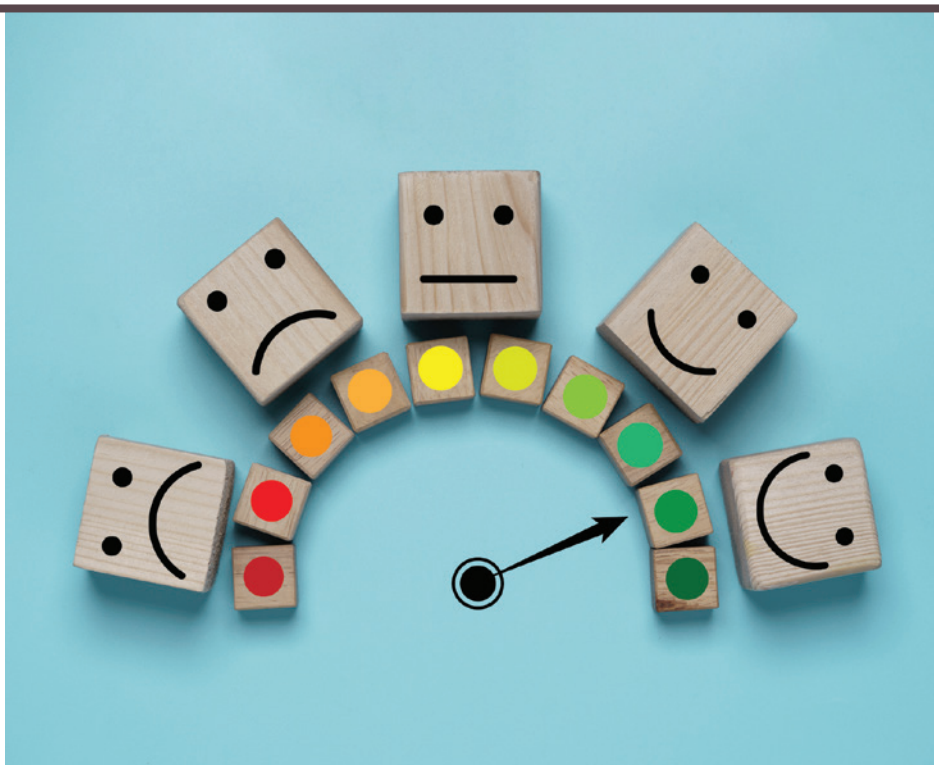
There is truly nothing else like our specialty. When I look at the future of our specialty, where many may see overwhelming challenges, I see so much opportunity.

There is truly nothing else like our specialty. When I look at the future of our specialty, where many may see overwhelming challenges, I see so much opportunity.

As I look at students becoming residents matching into our specialty, I see people not so different from myself.

When I worked as an ED tech for years prior to residency, I once had a physician tell me to "go to vet school" if I was going to do emergency medicine, because I might as well be entering the jungle.

References available online.



I smirk a little bit remembering this, because when I think about students matching into our specialty, I see myself: Just like me, our new EM residents love this field. Just like me, they can be told they'll be "entering the jungle" but they'll do it anyway, because — just like my experience — no one can convince them there's a better fit elsewhere.

That's what I see students doing every day as they enter this specialty: They are choosing emergency medicine. We are matching residents who want to be here, working alongside us for the betterment of the specialty. We are capturing those residents who crave this work and will contribute massively to the care of their patients.

Those same residents will get to the end of their grueling residency process and pass the "Third Year Test." ✶

Morgan Sweere, MD, MPH
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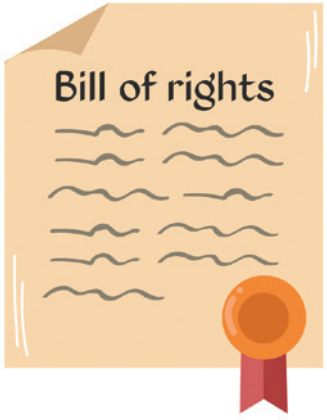
EMRA Adopts Displaced Resident Bill of Rights

The EMRA Board of Directors has approved a measure to provide support to residents whose programs face closure. The Displaced Resident Bill of Rights is as follows:

As evidenced by EMRA Policy Compendium, Section V.XIII, EMRA recognizes the possibility of residency program closure and resident displacement. Further, EMRA urges the protection and support of residents displaced or at risk of displacement due to training hospital or program closure prior to matriculation through the entirety of the emergency medicine residency program.

In recognition of the critical role that medical residents play in our healthcare system and the significant investment they have made in their training and careers, EMRA establishes this Bill of Rights to ensure the protection and support of displaced residents. EMRA affirms the following rights for displaced residents:

1. The right to matriculate into an alternate emergency medicine program for the remainder of their training, ideally with completion within the same projected time course.
2. The right to have support and assistance — including financially — from their respective graduate medical education (GME) office.
3. The right to adequate, timely notification regarding pending program closure and the need to transition to a new residency program.
4. The right for the resident voice to be present, valued, and considered in all settings where program closure is being considered.
5. The right to financial support for any transitions or relocations that occur as a result of program closure.
6. The right to sufficient malpractice insurance coverage provided by their former training institution that ensures protection from claims occurring during their time in the closed program.
7. The right to be excused from clinical duties as needed to interview for new residency positions.
8. The right to timely transfer of resident position funding to their new program.



References available online.

A Specialty Like No Other
An Organization Like No Other

50 years

Emergency Medicine Residents' Association Annual Report FY24

A message from
Blake Denley, MD
EMRA President
Ochsner Health, New Orleans

For half a century now, EMRA has worked tirelessly for its members and for emergency medicine. As I often find myself bragging about EMRA and its accomplishments, I quickly highlight that it is the hundreds of leaders and more than a hundred thousand members in its history who made EMRA what it is today — the oldest and largest independent residency organization in the world.

Emergency departments are the front doors of the hospital, the front lines of health care and the safety net for the entire U.S. health-care system. As you work day in and day out, advocating for your patients all the while, it's important to know there's a group advocating for you too. EMRA is that advocate — a voice that represents us, so that, in turn, we can provide the best care possible for our patients.

As emergency physicians, we have chosen a specialty unlike any other within medicine. My colleague and friend Dr. Morgan Sweere, EMRA's secretary and EM Resident editor-in-chief, mentions in her column how residency-bound medical students choose EM despite its challenges. Clearly, there is something special about emergency medicine.

EMRA, working on the behalf of our membership, continues to focus on 3 main pillars: Advocacy, Leadership, and Education.

Within the **Advocacy** pillar, the EMRA Representative Council adopted an astounding 19 resolutions on behalf of membership in the past year alone. These resolutions included initiatives to improve equity and reduce DO bias, encourage virtual interviews for residency program applicants, and increase EM opportunities for med students, among other measures.

On the **Leadership** front, EMRA's 20 committees organized more than 175 interest-specific meetings, webinars, and other events in FY24. Committee membership totaled nearly 5,000 members! Since its inception, the Leadership Academy has graduated 155 fellows, with an additional 32 currently enrolled in the program.

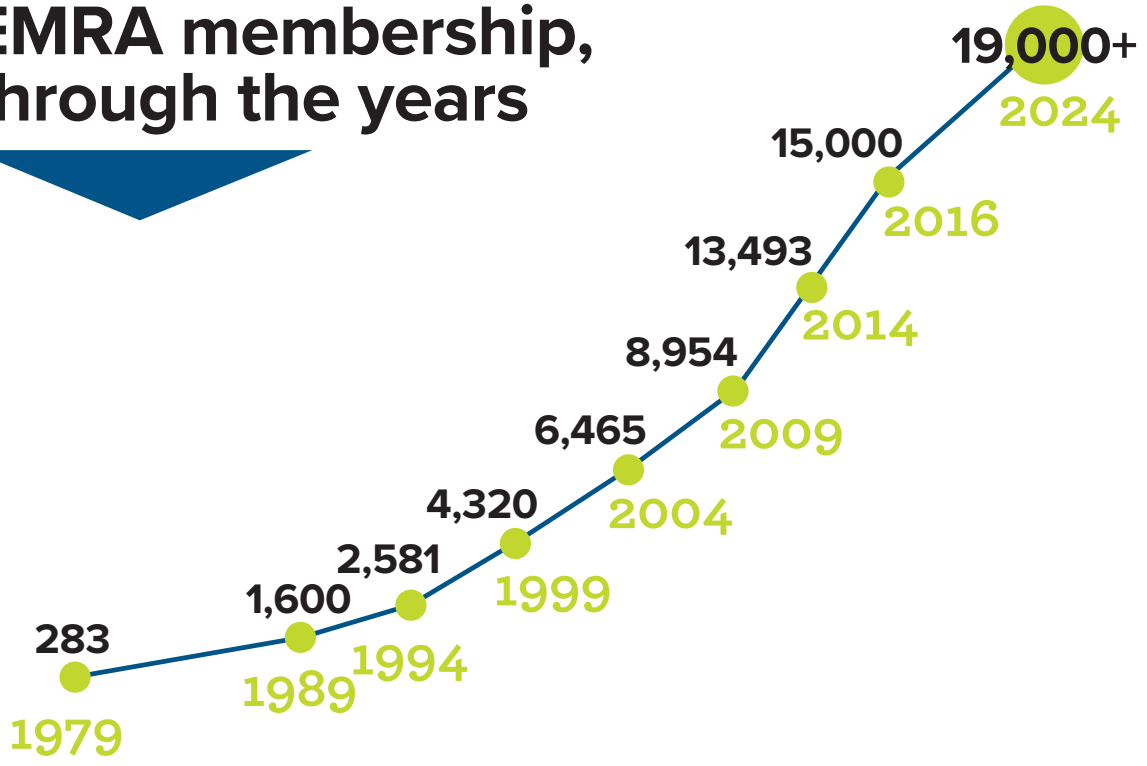
And finally, **Education**. EMRA's publications and podcasts continue as in-demand resources and mainstays of this organization and our specialty. Listeners download EMRA*Cast episodes more than 5,000 times per month! All EMRA publications — including EM Resident magazine (with print distribution of 18,000) and its digital counterpart emresident.org (with monthly views of more than 75,000) — present opportunities for members to become published authors as they build their CVs and advance their careers. With the EMRA Store's successful launch earlier this year, on-shift guides, cards, and other publications will have an even stronger reach.

Only a few noteworthy numbers are mentioned above. On the next few pages, I'm excited to share more highlights of our achievements in the past academic year. And, in 2024, I'm proud to mark the 50th anniversary of our organization.

EMRA

EMRA Annual Report FY24

EMRA membership, through the years



6,967% **Membership growth over 5 decades**

During EMRA's 50 years of existence (est. 1974), membership has increased from 283 in the organization's infancy to more than 19,000 today. Happy 50th Anniversary, EMRA!

EMRA Annual Report FY24

19,094
EMRA members, including medical students, residents, and fellows

>95% of EM residents are EMRA members

EMRA Annual Report FY24

>$3.2M Net assets, invested back into the organization and membership

20

Resolutions proposed and debated by EMRA members and the Representative Council. Of those, 19 were adopted:

- √ Improving equity/reducing DO bias
- √ Virtual interviews for residency program applications
- √ Increasing EM opportunities for medical students (amendment to previous policy)
- √ Addressing misuse and abuse of crowd control weapons
- √ Advocating for equitable implementation of second-look-day programming
- √ Recognizing voting access status as a social determinant of health
- √ Protecting the rights of pregnant patients who use opioids
- √ Immunizations in the ED
- √ Trauma-informed care curriculum incorporation into EM residency didactics
- √ Availability and accessibility of fentanyl test strips in the ED
- √ Language justice and health equity in the ED
- √ Sponsorship and advertising (amendment to previous policy)
- √ The Match and residency and fellowship application
- √ Resident duty hours policy (amendment to previous policy)
- √ Relationship with the biomedical industry (amendment to previous policy)
- √ Firearm safety and injury prevention (amendment to previous policy)
- √ National bias reporting
- √ Emergency medicine disaster preparedness
- √ Decriminalizing human trafficking victims and sex workers
- X Creation of climate change and environmental justice committee (not adopted)

Protecting our training

EMRA is dedicated to ensuring residents receive high-quality training and do not compete with PAs and NPs for procedures in the ED.

27 Educational and leadership partnerships

- AAED
- ABEM
- ACEP
- ACMT
- Advanced Analgesia in the ED
- AEROS
- ALiEM
- AMA
- AMBOSS
- Carol Rivers Board Review
- Coalition on Psychiatric Emergencies
- CORD
- EB Medicine
- EDPMA
- EMCrit
- EMF
- EMPI
- EM:RAP
- Essentials of EM
- HIPPO Education
- Ivy Clinicians
- NEMPAC
- PEER Prep
- PEPID
- PolicyRx
- Resuscitative Tee Workshop
- Rosh Review
- SAEM

9+ Member lifestyle benefits

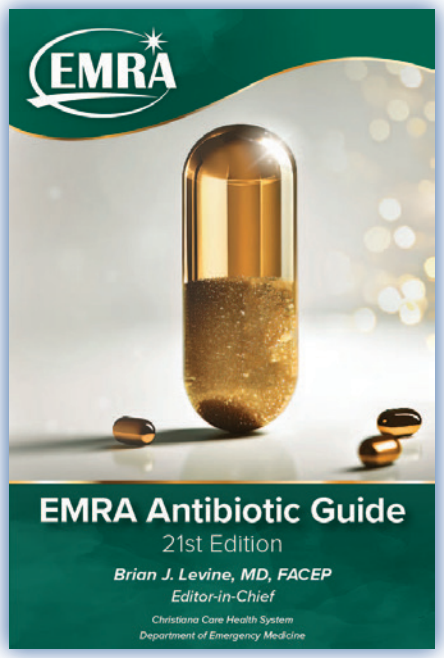
- Careismatic Brands
- Doctors Without Quarters
- Headspace
- Integrated Wealthcare
- Jay Weinberg
- Laurel Road
- NB marketplace
- Pattern
- The Whole Physician
- And more!

EMRA Annual Report FY24

904,500 Free clinical resource giveaways since EMRA's inception

36
On-shift guides and original EMRA resources

3 New or newly-revised EMRA guides, references, and apps



EMRA CAST

5,000+
EMRA*Cast downloads per month

24 New podcast episodes

EMRA HANGOUTS

9
EMRA Hangout sessions for med students to engage with EM leaders and faculty

EM Resident
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75,000+
Monthly views on emresident.org

18,000 Print distribution

EMRA Match

313,494
Page views of EMRA Match for residency program, clerkship, fellowship, and job searches

9,515 Unique users

EMRA Store

1 Successful launch and debut of the EMRA Store

EMRA Annual Report FY24

248 Residency programs with 100% membership (also known as "EMRAfied" programs)

20 Committees

175+ Meetings, webinars, publications

4,844 Members of EMRA's Committees
- *Medical students*
- *Residents*
- *Fellows*

EMRA and ACEP Leadership Academy

187 Leadership Academy fellows

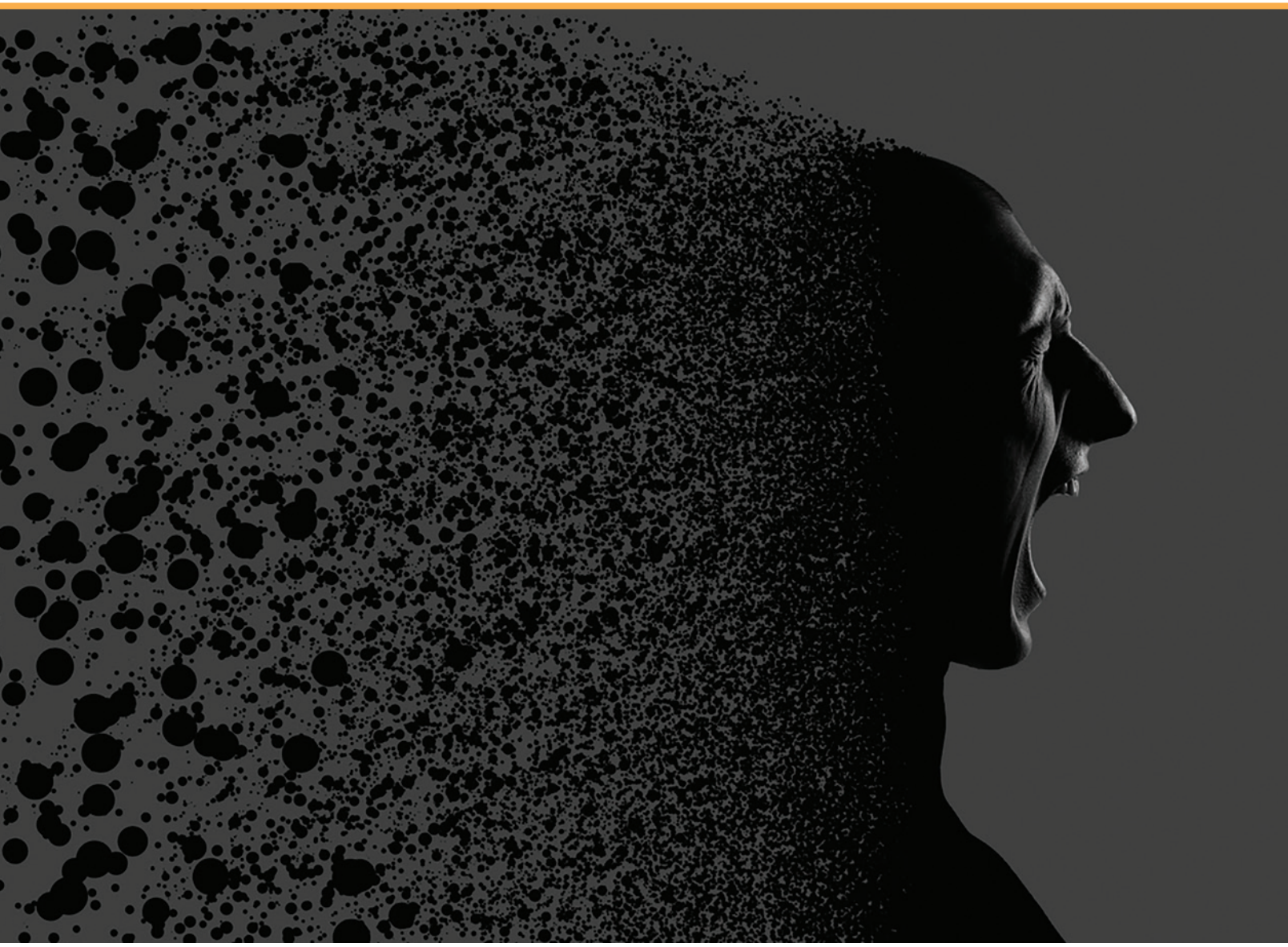
155 graduates and **32** currently in the program

EMRA AWARDS

35+ Awards, scholarships, and grants presented to members

The Emergency Medicine Residents' Association

is proud to mark 50 years in 2024. We remain ever-present and ever-strong as the independent voice of emergency medicine physicians-in-training and the future of our specialty.



Strategies and Insights for Procedural Pain Management in the ED

Jenn Mirrielees, MD, MS
Clinical Instructor
EM Administration Fellow
University of Wisconsin Hospital and Clinics

Jada Thompson
Advanced EM Technician
Researcher, Dept. of Perioperative & Orthopedic Surgery
Dell Medical School at UT Austin

Some of the most common procedures in the emergency department are painful and anxiety-inducing for patients. Despite a broad spectrum of pharmacologic and nonpharmacologic interventions, managing procedural pain is complex, stressful, and frequently rushed on a busy shift — and often doesn't meet the patient's expectations.

In a perfect world, we would aim to keep patients calm and comfortable, but in real life, managing pain often falls several rungs on the ED hierarchy of needs when genuine life-and-death situations are present, either for the patient whose life is in danger or elsewhere in the department.

The inadequacy of analgesia during ED procedures has been well-documented,[1,2,3] and every EM resident can remember examples in which we feel that our plan and execution for pain management failed. It is also helpful to

References available online.

reflect on the fact that the effectiveness of any care in the ED, including procedural pain management, never rests with one person.

Understandably, most patients with painful injuries want pain relief, and they want it now. Aside from people living with chronic pain, the average person simply has not been oriented to the reality that sometimes, nothing can be done by a doctor or nurse to take away all their pain and give them complete relief. For some patients, this disappointment is compounded once we explain that general anesthesia simply is not an option in the ED, and many of us will have heard the phrase "knock me out, doc!" some time before graduating residency.

General guidance is to proactively set expectations and recalibrate to a goal of "tolerable pain." The best approach to reaching this goal, acknowledging that in the real world we often fall short, may be a combination of systems-level interventions to address persistent gaps, a high standard for team dynamics and communication, and broadening our personalized multimodal procedural pain plans for each patient.

A MODEL FOR PROCEDURAL PAIN MANAGEMENT

If you have ever been disappointed that any of your most proactive, most patient-centered pain management plans ultimately fell short of the goal of "tolerable pain," there are likely many reasons for this. Many parts of pain management are completely outside of our control (e.g., opioid tolerance and hyperalgesia in patients with chronic pain), while some factors are potentially modifiable through team efforts such as quality improvement initiatives. We created a model of the determinants of procedural pain management strategies (**Figure 1**) to put into context the much larger framework that poses barriers to achieving tolerable procedural pain.

PATIENT FACTORS

Not all injuries are created equal. A patient blessed with strong coping skills with a superficial, linear laceration to the thigh is going to have a better experience with their procedural pain than a polytrauma patient with multiple fractures arriving to the ED after a high-speed motor vehicle collision.

Several non-modifiable patient factors — injury type and complexity, variables contributing to pain tolerance and analgesia efficacy, coping skills, underlying anxiety, psychosocial context of the injury — are meaningful to understand in order to tailor your approach to that patient's care. Polytrauma patients will likely need IV opioids re-dosed throughout their ED course, along with a thoughtful multimodal approach to specific procedures such as hematoma block for distal radius fracture reduction. Anticipating which patients will be most benefited by nonpharmacologic interventions is helpful as well.

Pediatric patients in particular benefit from well-managed parental anxiety and active parental education and involvement, along with distraction

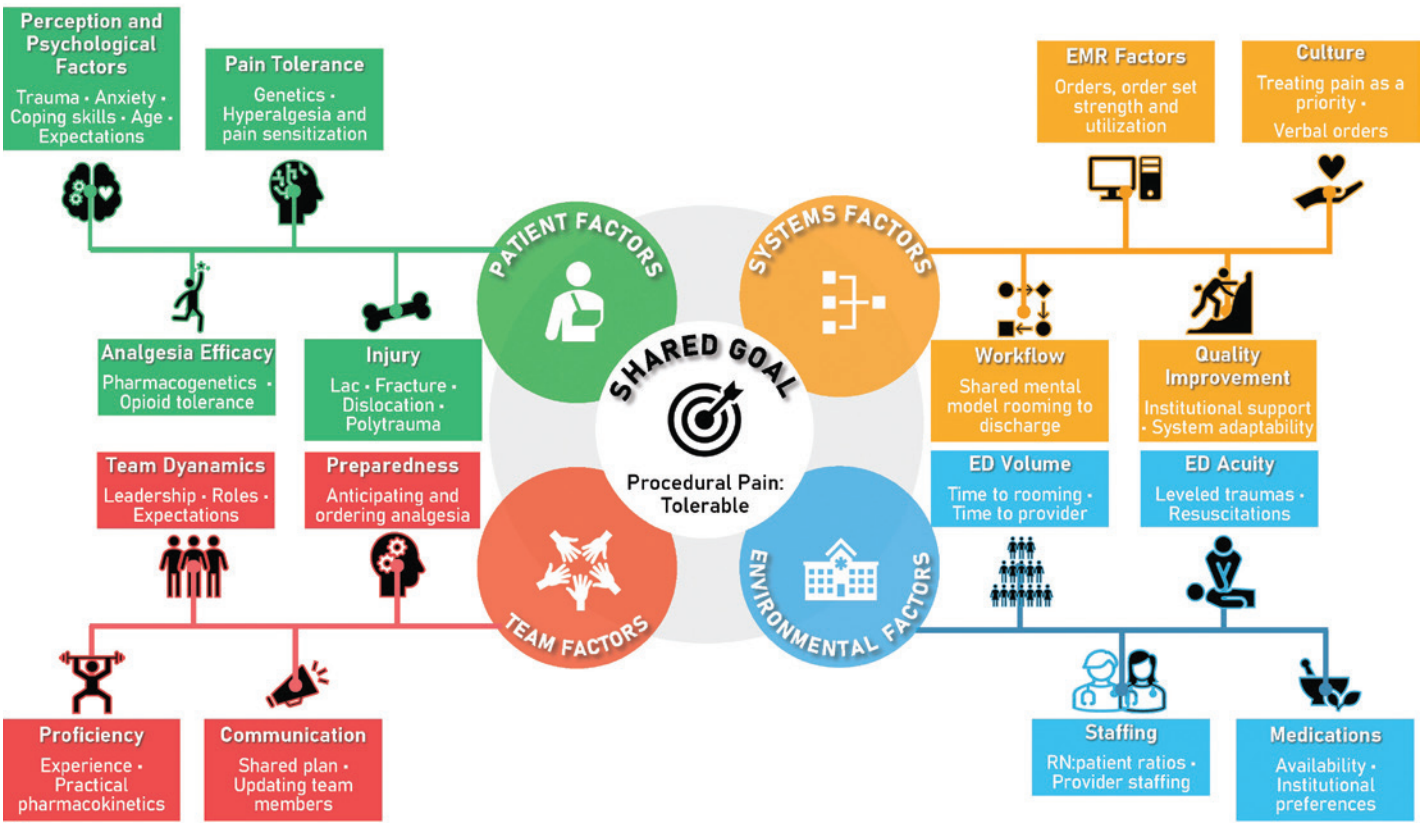


FIGURE 1

References available online.

techniques.[4,5,6] At our hospital, these nonpharmacological approaches are expertly led by support from a child life team member at the bedside.

Among adults, prior traumatic experiences are associated with dysfunctional pain perception.[7] Pain management is more challenging in adult patients with comorbid anxiety and/or challenges with coping skills.[8] Such patients, like pediatric patients, are likely to benefit from similar interventions addressing the psychological inputs to their experience of pain. Offering a warm blanket and turning the lights off has helped put many of these patients at ease in our experience. These types of actions build a sense of trust and compassion, and show that the physician is compassionate toward their pain and suffering.

Notably, there is significant variability in patient perception of provider empathy.[9] This perception in turn affects patient perception of pain.[10] Physicians who self-report higher levels of empathy are actually more likely to be perceived by patients as less caring.[11] This is a reminder to us all to be thoughtful in demonstrating and voicing empathy to our patients, as one of the non-pharmacological adjuncts to managing procedural pain.

Keep in mind as well that patients living with chronic conditions such as fibromyalgia or chronic pain are likely to have undergone very real biological rewiring, creating a state of central pain sensitization and hyperalgesia.[12] Some of these patients may be chronic users of prescription opioids, so anticipate both a tolerance effect for opioid analgesia during their procedure along with an even greater reliance on non-opioid analgesia such as ketorolac.

For all patients, expectation setting is critical. We have had great experience with being direct about a shared goal of "tolerable pain" very early into the ED encounter. It is often apparent from the doorway which patients will require painful ED procedures, and engaging about this topic sooner rather than later gives the patient an opportunity to process the expectation, ask questions, and ultimately feel as satisfied as possible that our plan for managing their pain is thoughtful and offers them the best relief possible outside of what can be achieved in the operating room.

TEAM FACTORS

No emergency physician is an island. Procedural pain management success hinges on a shared plan between the physician and the nurse. The physician's role, in many ways, is to offer a well-informed plan and then empower the nurse to execute it in real time as the patient's pain evolves before, during, and after a painful procedure.

For example, lidocaine-epinephrine-tetracaine (LET) for laceration repairs can be ordered immediately after the patient is roomed, to be available for the nurse at any time after the initial physician assessment. For more complex injuries, thoughtful ordering of PO acetaminophen vs. ibuprofen, IV ketorolac, and/or IV analgesia PRNs as soon as possible after patient rooming improves efficiency as well as the patient experience.

Once a plan is created and shared with the nurse, and medications are ordered and available, team communication and dynamics become important for effective procedural pain management. Getting to know and build trust with nursing colleagues improves every kind of care that we provide in the ED, and pain management is an excellent example of this. Updating the nurse about estimated time to procedure start is crucial if the plan involves IV hydromorphone, with time to peak effect as long as 20 minutes. Appropriate updates also empower the nurse to be available at procedure start, either to directly support the procedure or, at minimum, to check on the patient and offer last-minute insights that strengthen the plan.

Unlike patient factors, the majority of team factors included in our model are directly modifiable, specifically by the physician leading the team. While these factors are generally intuitive, high-level team management insights are not at the forefront of anyone's mind on a busy shift. Reflecting on what we can do better as team leaders gives us the opportunity to be proactive and improve.

ENVIRONMENTAL FACTORS

There are often real-world barriers that even a great team with a great plan will have difficulty overcoming.

For example, the higher the patient:provider and patient:RN ratio, the less focus and attention each patient will receive, unfortunately. Nurses report that staff shortages are a direct contributor to inadequate pain management.[13]

Higher ED volumes and acuity levels are factors as well.[14,15] ED team members and resources are finite, and a critical polytrauma patient or resuscitation elsewhere in the department will pull team members away from other patients.

Another environmental factor to consider is the immediate availability of procedural pain management medications, which is likely to vary from ED to ED. Availability of medications is, of course, also contingent upon nursing availability.

Environmental factors are by their nature difficult to modify and require large-scale change, particularly in addressing staffing.

SYSTEMS FACTORS

The ED is an intuitive home for systems-level process improvements. Our work environment is fast-paced and unpredictable, necessitating emergency physicians and our co-workers to be responsive, adaptable, and efficient.

High-volume ED procedures in particular are amenable to quality improvement work, with prior successes including reducing time to analgesia and improving pain scores.[16,17] An EM resident will learn early into training that most patients presenting to the ED with painful injuries and requiring procedural care will experience similar trajectories in their pain with common analgesia needs, varying by injury complexity and other factors.

A patient's experience with procedural pain management is highly likely to be influenced by systems

References available online.

Quick Review: Ins & Outs of Managing Procedural Pain

L.E.T.'s Get It Started

Making Lac Repair as Painless as Possible

Lidocaine-epinephrine-tetracaine (typically a gel, also available as a solution) provides both poke-free topical anesthesia and hemostasis

Excellent topical anesthesia for thin skin (face and scalp) and ~20% may still require local infiltration[1,2]; limited utility for the thick (palms, soles)

Recommended wait time from application to lac repair: 30 minutes

Just as effective (and appreciated!) in adult patients as pediatric patients

Recommended workflow: ask RN to apply LET immediately after you first see the patient → 30 minutes return to complete lac repair and discharge

Welcome to the Block Party

Targeted Local Anesthesia for Extremity Injuries

Hematoma Blocks

Provide local anesthesia directly at the fracture site

Can be done by sight or with ultrasound guidance[3]

May be superior to procedural sedation for distal extremity fracture care[4,5]

Local	Onset	Duration
Lidocaine 1% w/o epi	2-5 min	50-120 min
Lidocaine 1% w/epi	2-5 min	60-180 min
Marcaine 0.25%	5-10 min	240-480 min

We frequently use a 50:50 mix lidocaine 1% w/epi:marcaine 0.25% to combine faster onset (shorter time to procedure start) with longer duration of action (post-procedure pain control); total volume 8-10 mL

Ring blocks

Ideal for digit injuries (laceration,, fracture, dislocation, nail removal)

Dorsal approach: two injections, one into each webspace on either side of the digit, injection volume 2-3 mL per side

Volar approach: single injection deep to digital-palmar crease, injection volume 3 mL (similar effectiveness to 2-injection dorsal approach)

Same medications as hematoma block (see above)

With a Little Help From My Friends

IV Analgesia is a Team Sport

Effective use of IV opioids for procedural pain requires thoughtful coordination with nursing to provide analgesia well-timed to procedure

Requires a good plan, strong communication, timely order placement

We recommend a structured, brief conversation with the nurse and any team members (example: Orthopedic Surgery) to start off on the right foot

IV Analgesia	Peak	Duration
IV Dilaudid	10-20 min	180-240 min
IV Morphine	5-10 min	180-300 min
IV Fentanyl	1-5 min	30-60 min

Don't forget about Toradol IV and Tylenol PO vs IV when appropriate

I Hurt, But I'm Still Alive

Setting Expectations Around Procedural Pain

There is a significant gap between pain management expectations and reality in the ED[7] and inadequate existing guidance for physicians

Setting expectations is a team effort: use "we" statements, aim for matched messaging across the team (RN, consultants)

Frame expectations around "tolerable", the earlier into the ED course the better, remember to be intentional and consistent around this specific goal

Explain that your plan is thoughtful and implementing every reasonable tool, why complete pain-relief is not possible, actively express empathy

Enter Sandman

Procedural Sedation

Procedural sedation is higher risk but at times unavoidable

Common indications: age/developmental stage, coping skills, patient unable to tolerate minimal manipulation due to severity of pain/injury type

Think ahead whenever possible, including expectations setting discussion

If being admitted to a surgical service, discuss option for procedure in OR

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FIGURE 2

References available online.

factors. One example of this is the EMR, particularly order-set utilization. Order sets and protocols for multimodal pain management have been found to improve pain management, promote non-opioid analgesia, and decrease opioid analgesia needs.[18,19,20]

Other systems factors that are more difficult to address include unit culture toward pain management,[21] such as whether nursing is empowered to advocate for individual patients who may not have adequate analgesia prior to starting procedures. An example in which culture (systems factor) and dynamics (team factor) intersect is the use of verbal orders, which has the potential to save time by allowing a nurse to administer analgesia by verbal order only. Understandably, nurses encounter many scenarios in which they may not be comfortable accepting verbal orders, and EM nursing publications have commented on this.[22] An ED culture most supportive of verbal orders is best cultivated through compliance with guidelines for verbal orders including closed loop communication[a] and strong relationships between physicians and nurses.

PROCEDURAL PAIN MANAGEMENT TOOLS

Speaking of improvement, one way that you — as an EM resident-physician practicing right now — can improve your own procedural strategy and success is to become as comfortable as possible with all the options available for pain management in the ED.

We spoke with our RN pool about these topics and have included their insights here to provide depth from the perspectives of ED team members who most closely manage pain at the bedside.

Depending on your training environment and culture, you may already use LET to its full potential every day, or it may not even be stocked in your ED. When we surveyed our nursing colleagues about areas for improvement in procedural pain management, the very first response was an enthusiastic "LET is king!" This came from one of our most experienced ED RNs. LET does need time to get the job done, so remember not to rush royalty. You may already be a stickler for ensuring medication administration at an appropriate amount of time before beginning a procedure, or you may feel so rushed when you're ready to start that laceration repair that you find yourself impatient and wondering if you really need to wait a few extra minutes to allow medications to take peak effect.

Other food for thought gathered from our ED RNs includes overwhelming support for IV acetaminophen. Data in the literature, however, is mixed and primarily focused on post-operative pain. Some studies support IV acetaminophen efficacy over PO,[26] particularly with reducing time to peak effect.[27] Some studies found no difference between the two routes.[28,29,30] We encourage you to do what you think is best for your patients in your own experience.

Another common insight: Setting expectations is everyone's job. Our ED RNs recommended that this be considered a team effort in which physicians and nurses express the same messaging and compassion to the patient.

We have put together a summary of medications for managing procedural pain in the ED (**Figure 2**).

CONCLUSION

Effective pain management is challenging under any circumstances, particularly for patients presenting to the ED with painful injuries. Many factors contribute to how patients experience and manage pain during procedures. Some factors are more immediately modifiable, such as intentional and strategic use of available pharmacological and nonpharmacological approaches to pain management. Other factors are higher level, difficult to influence on the ground, and potential targets for quality improvement interventions.

There is always room for growth, particularly around recurring gaps and systems issues that can be tackled through multidisciplinary team efforts. In your daily practice, set yourself up for success with a good plan, strong standards for team communication and dynamics, and healthy expectations setting. ✶

Editor's note: EMRA provides additional resources for pain management in the ED, including the EMRA and AAED Nerve Blocks and Procedural Pain Management Guide and the Pain Management Guide, 1st Edition. These publications, and others, can be purchased via the EMRA Store and Amazon.

References available online.

A CELEBRATION IN 2024 AS WE HONOR 50 YEARS OF EMRA!

This year, EMRA marks 50 years of serving emergency physicians-in-training, medical students, fellows, and EMRA alumni. Thank you to all our past and present members, partners, and supporters for being a part of our EM specialty-serving organization.

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Breaking the News: A Discussion on Effective Communication of Death Disclosure

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A 55-year-old man presents to your emergency department in cardiac arrest. Despite multiple rounds of epinephrine, CPR, and defibrillation, he does not regain pulses. You call time of death. Shortly afterward, a nurse informs you that the patient's wife and children are gathered in the consultation room to hear an update on his status.

For the emergency physician, communicating difficult news is an important responsibility. Telling a family member that their loved one has died is never an easy conversation, and it requires careful thought and consideration.

After my first death disclosure, I was juggling thoughts about the medical case with the feelings I had about the ensuing family conversation. I wondered if I had said the wrong things, said too much, or said too little. Over time, I realized the content of my conversation mattered less than the way in which I communicated and the environment I established for these difficult moments.

These are some of the lessons I have learned and strategies I have found to help ease the difficult process of disclosing the death of a patient, both for the loved ones and for myself.

PLANNING

As with any endeavor in medicine, teamwork will ensure things go smoothly. Identifying your partners and engaging in collaboration is a must. Prior to the encounter, asking your hospital's social worker and/or chaplain

References available online.

to gather family members in a quiet and secluded waiting room will give you time to prepare. Meet with the hospital chaplain, the emergency department social worker, and the patient's nurse prior to the encounter to confirm the patient's identifying information, and confirm with whom you will be speaking.

Identify any cultural or other customs relevant to the patient's family that may affect their feelings about, and understanding of, the patient's condition. During a painful and difficult conversation, incorrect information, confusion, or miscommunication are the last things you'd want to impart.

A SAFE SPACE

For the family, gathering in a designated space with minimal distractions or interruptions will allow for collection of thoughts. Before entering, take a breath, pause, and gather yourself to transition from the intense experience of running a code to the emotionally challenging role of informing next-of-kin.

THE MEETING

Families rushing to the ED knowing their loved one is experiencing a medical emergency are faced with a bombardment of information, compounded by emotional stress and a hectic environment. When entering the room for the first time, introduce yourself clearly as the emergency physician. Your partners from the ED should also introduce themselves at this time, if they have not already done so. Confirm the relationship of the present individuals to the patient.

During this initial encounter, use neutral body language, make eye contact, and speak in a soft tone. These communication signals aid in easing the tension in the room and help draw focus to the information being communicated.

A DIFFICULT DISCLOSURE

The key information should be stated clearly, without using medical jargon or complicated terminology. In an already complex environment, simplicity is vital. State the nature of the patient's arrival to the ED, and communicate that your team did everything possible to save their life. State clearly that the individual "has died." This direct statement is sufficient, and preferred, as it avoids any ambiguity or confusion. Avoid euphemisms for death, as these can often be misinterpreted and may, in extreme cases, lead some family to believe the patient is still alive.

As the family absorbs the difficult news, they naturally will have a strong emotional reaction, which can be highly variable. Offer your condolences and listen to their questions and statements. If appropriate, let the family know that they can visit their loved one in a designated viewing room.

As expressed earlier, this process is a collaborative one. At this point, allow the chaplain, nurse, or social worker to take over and continue to support and provide information on next steps. Restate your condolences, offer assistance with any further questions, and exit the room.

REFLECTION

In the emergency department, there is no shortage of unfortunate outcomes, sudden and unexpected deaths, and even expected ones. As emergency physicians, we are used to switching gears frequently to put out the next fire.

However, being the bearer of bad news and participating in emotionally taxing situations can take a toll on even the toughest of clinicians. After a disclosure, I try to take an immediate opportunity to sit and digest my thoughts. Even if it only lasts a few seconds, it allows me to process the experience and acknowledge what I went through before moving on to the next task.

There are inevitably some cases that linger in my mind, and I find it helpful to discuss these with my co-residents. Whatever your method of reflection may be, it is essential to find a process that works for you. If you find yourself persistently or unusually troubled by a patient case, seek help from colleagues, mentors, or mental health professionals. Always attend to your mental health after a difficult case.

As emergency physicians, we are uniquely equipped to handle difficult conversations, and we must be deliberate with our words and actions. Although nothing can lessen the impact of losing a loved one, providing a death disclosure in a respectful and effective manner can prevent further harm and distress to a patient's family. Planning ahead, organizing the appropriate individuals, and securing a safe location are essential.

Finally, making sure to reflect and acknowledge our own thoughts can help digest the situation in a healthy way and lessen the emotional impact on physicians. ✶

References available online.

In-Hospital Cardiac Arrest: An Unexpected Turn of Events

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PRESENTING CONCERNS, CLINICAL FINDINGS

A 70-year-old man with a history of coronary artery disease (CAD), hypertension, hyperlipidemia, and diabetes mellitus presented to the emergency department with generalized weakness, decreased oral intake, and diarrhea for 3 days. He had undergone percutaneous coronary intervention (PCI) of the left anterior descending (LAD) artery in 2012.

On arrival, the patient was ill-appearing and diaphoretic. Initial vital signs were significant for blood pressure of 82/49, pulse rate of 86 beats/min, respiratory rate of 16 breaths/min, and oxygen saturation of 98% on room air.

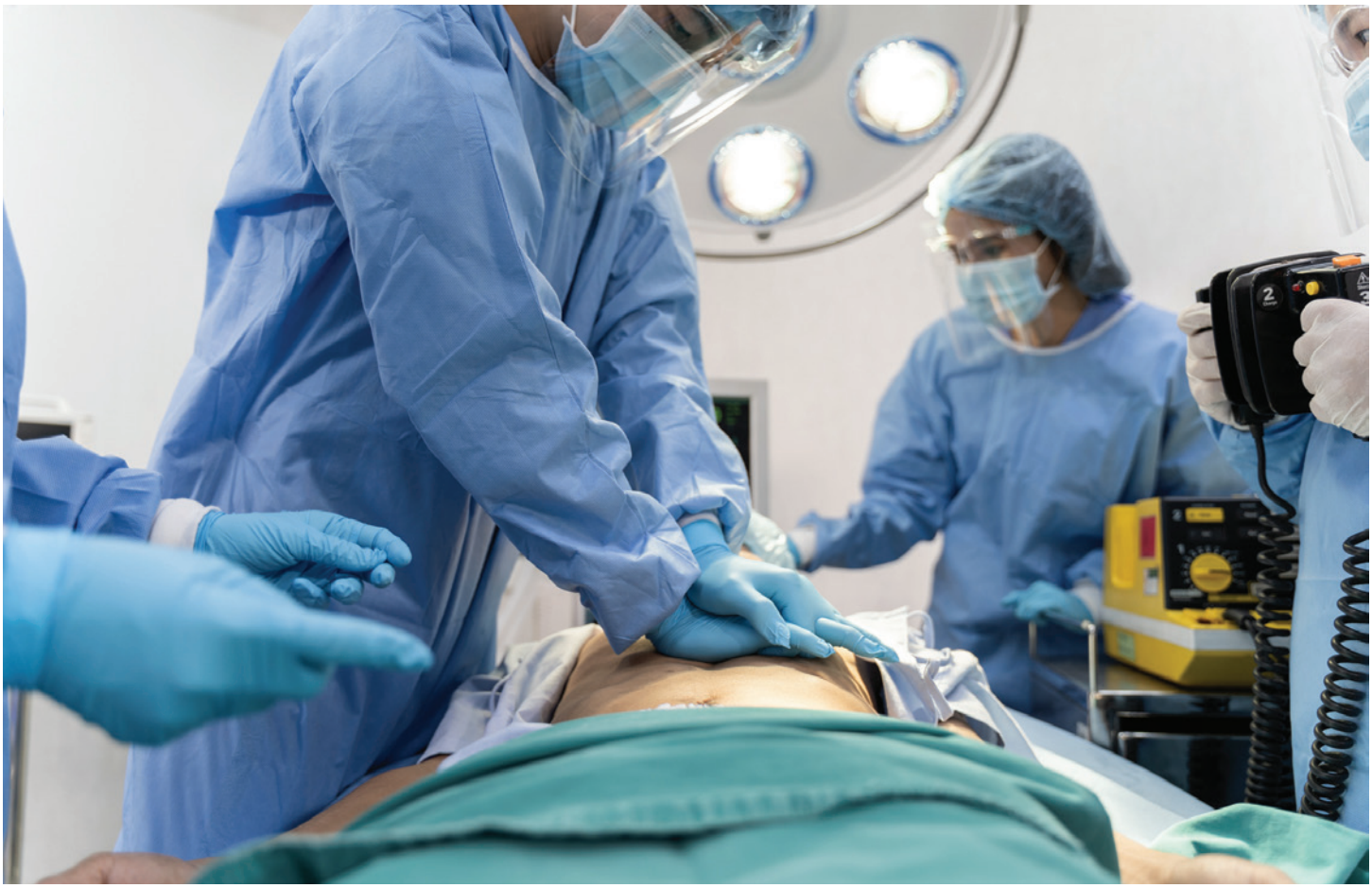
An ECG was obtained on arrival (**Figure 1**). The patient recently had been admitted and treated for left lower extremity cellulitis and osteomyelitis. The patient's presentation to the emergency department prompted a sepsis workup.

CLINICAL COURSE

The patient presented concerning for sepsis because of his recent hospital admission for cellulitis/osteomyelitis, age >65 years, and hypotension. His pulse rate of 86 was within normal limits but potentially blunted by beta blockade.

Initial lab workup was notable for leukocytosis of 14.41, creatinine 1.8, lactic acid of 3, and troponin of 0.153. Of note, potassium and magnesium were within normal limits.

Vancomycin and cefepime were



References available online.

empirically given for sepsis.

Shortly afterward, the patient developed sudden onset dyspnea, which prompted discontinuation of the antibiotics over concern of possible anaphylactic reaction. The patient continued to endorse dyspnea without skin rashes, wheezing, or stridor; therefore, an additional ECG was performed (90 minutes after the initial ECG) during re-evaluation.

The patient was then found to have acute ST elevation suggestive of STEMI (**Figure 2**); therefore, an institutional Code STEMI was called.

Four minutes later, a third ECG (**Figure 3**) was performed by staff because the patient became unresponsive. This third ECG showed ventricular tachycardia. Manual CPR was initiated, while the patient received bag mask ventilation. Defibrillation was performed within the first few minutes of initiating Advanced Cardiac Life Support (ACLS), restoring normal sinus rhythm.

Subsequently, the patient was intubated and transported to a nearby facility with catheterization laboratory capabilities for PCI. The patient was given heparin, ticagrelor, aspirin, and amiodarone. He underwent left heart catheterization, which showed 90% occlusion of LAD and 99% occlusion of the right coronary artery (RCA). The left ventricular ejection fraction was noted to be 50%. PCI with an everolimus-eluting stent system was performed on both vessels with successful revascularization.

The patient did well post-op. He was extubated the following day and discharged home 3 days later.

DIAGNOSIS

The initial ECG (**Figure 1**) showed normal sinus rhythm at 81 beats per minute with ST depression in V1 and V2 and nonspecific T wave changes in V5 and V6. A comparison ECG performed 8 months prior was notable for meeting voltage criteria for left ventricular hypertrophy and bradycardia, but was otherwise within normal limits.

The second ECG (**Figure 2**) was consistent with inferior ST elevation myocardial infarction noted for ST

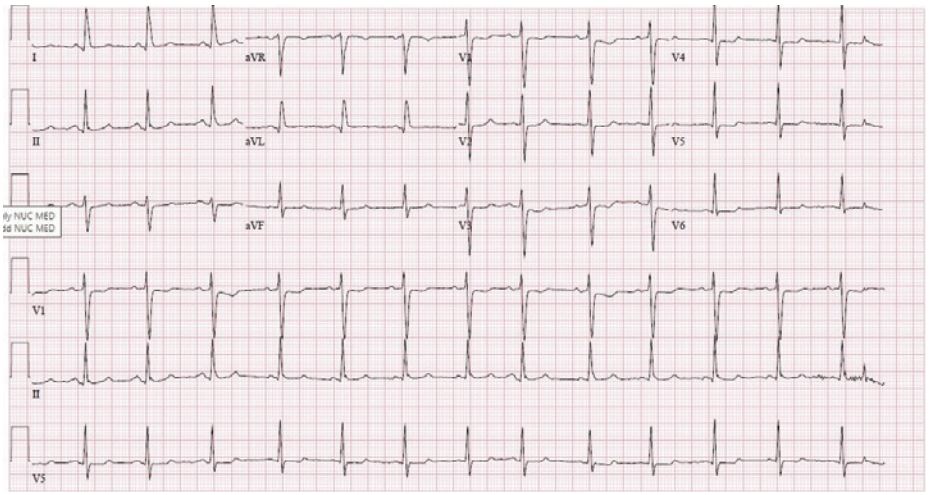


FIGURE 1

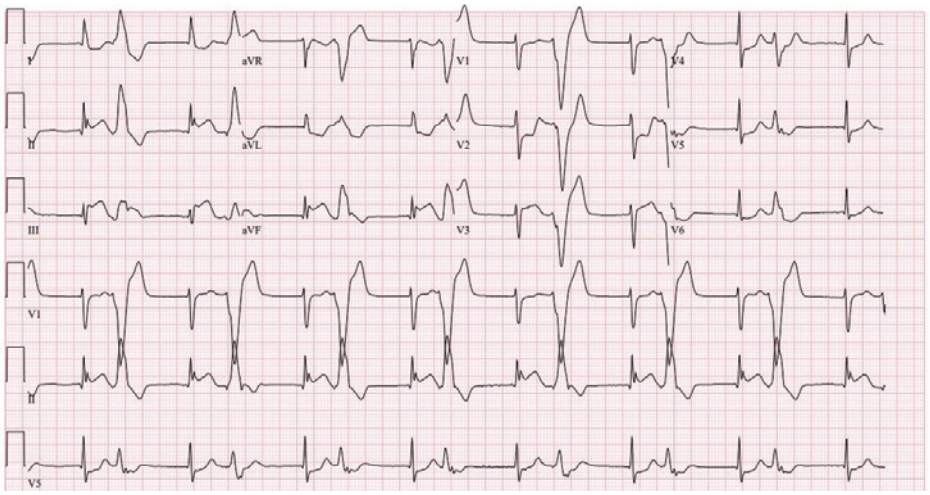


FIGURE 2

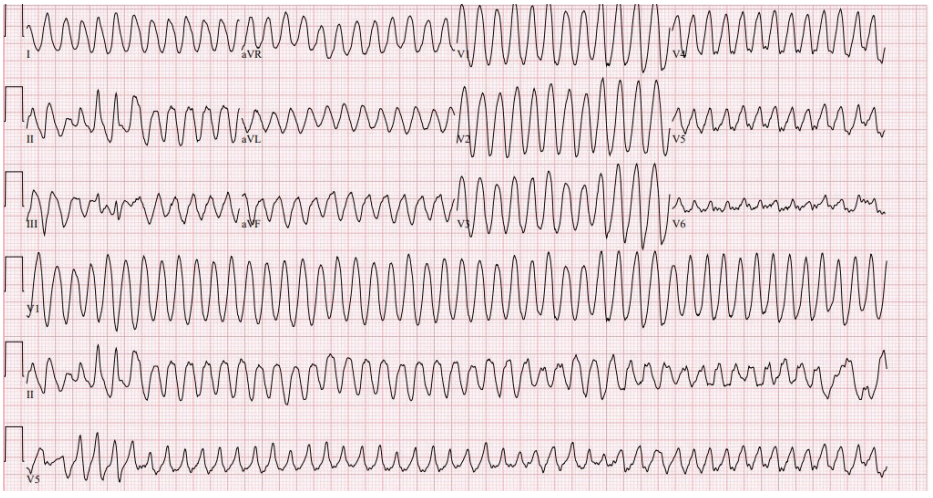


FIGURE 3

References available online.

elevation in leads II, III, and aVF with reciprocal changes in leads I and aVR. This ECG was also notable for multiple PVCs and an accelerated junctional rhythm with hidden P waves at a rate of 94 beats per minute.

The third and final ECG (**Figure 3**) showed wide complex tachycardia at 265 beats per minute consistent with ventricular tachycardia.

DISCUSSION

Identifying a rapidly evolving myocardial infarction in the ED in a patient with a distracting presentation necessitates a timely diagnosis and prompt provider evaluation. As discussed previously, this patient presented with clinical and laboratory findings consistent with sepsis. On initial presentation, there were no typical symptoms of myocardial ischemia such as chest pain or shortness of breath.[1]

However, the patient was noted to be diaphoretic. Atypical presentation of chest pain — such as gastrointestinal discomfort, nausea, and vomiting, which are commonly associated with elderly females or diabetic patients — was not evident.[2]

Although the patient did have an elevated HEART score of 5 on presentation, the use of this risk stratification device may not be applicable in this case because of the absence of chest pain or suspicion of ACS on presentation.

In the setting of cardiac arrest, time from onset of ventricular fibrillation/ tachycardia to defibrillation is crucial.

The initial ECG showed normal sinus rhythm and mild elevation in troponin (0.153). Troponin was initially thought to be elevated due to myocardial injury in relation to sepsis, which is seen in 85% of adults with sepsis.[3]

Approximately 1 hour after arrival, the patient began experiencing sudden onset dyspnea, which initially was thought to be associated with the administration of antibiotics. ST elevation changes in the setting of anaphylaxis were reported to be seen in Kounis syndrome, which is thought to be due to the release of histamine following mast cell activation leading to coronary artery vasospasm, plaque erosion, or even rupture.[4]

However, in the absence of cutaneous (blanching rashes) or respiratory findings (i.e., wheezing/stridor) on physical exam, anaphylaxis was unlikely in this case. Recognition of the patient's change in status and symptoms prompted a rapid re-evaluation, leading to identification of evolving myocardial ischemia. ✶

TAKE-HOME POINTS

- Always be prepared for a change in patient status. When these situations arise, quickly and thoroughly re-evaluate the patient with a new and broader differential diagnosis.
- Keep atypical presentation of myocardial infarction as a differential diagnosis, especially in patients with advanced age and with risk factors for atypical presentation such as diabetes mellitus, hypertension, and hyperlipidemia.[2]
- Rates of in-hospital cardiac arrest are 9-10 per 1,000 hospital admissions, and the survival rate is approximately 20%. Prompt re-examination and evaluation of the patient, as well as efficient transition to ACLS when identified, is of the utmost importance.[5]
- In the setting of cardiac arrest, time from onset of ventricular fibrillation/tachycardia to defibrillation is crucial. According to the American Heart Association, defibrillation in fewer than 2 minutes of recognition of a shockable rhythm has been associated with greater rates of long-term survival at 1-5 years.[6]

References available online.

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TICK TALK: The Link Between Ticks and Red Meat Allergies

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Ticks are the culprits behind illnesses such as Lyme disease, Rocky Mountain spotted fever, and babesiosis. Additionally, an unexpected association is found among ticks, a small oligosaccharide called alpha-gal, and red meat allergies. This association, in certain instances, can lead to severe anaphylactic reactions, posing a potential threat to life.

A recent article in the Annals of Emergency Medicine addressed alpha-gal as an increasingly prevalent trigger for anaphylaxis, stating that as many as 10% of patients diagnosed with idiopathic anaphylaxis have alpha-gal syndrome.[1,2]

HISTORY

In 1991, Anthony Deutsch, MD, observed the link between tick bites and red meat allergies and presented his findings to the Georgia Allergy Society and the CDC. He documented a limited case series of 10 patients who experienced hives or anaphylaxis after consuming red meat several weeks to months after being bitten by ticks. Unfortunately, no additional statements or follow-ups were made for many years.[3]

In 2004, the FDA approved cetuximab, a chimeric mouse and human antibody against EGFR, used to treat colorectal cancer. However, post-market

References available online.

surveillance revealed that many patients developed allergic reactions, including anaphylaxis, to this drug. Concerns arose when these allergic reactions were noted to follow a geographic distribution.[1,4,5]

In 2008, a paper in the New England Journal of Medicine identified galactose-alpha-1,3-galactose (i.e., alpha-gal) as the cause and confirmed the geographic distribution of allergic reactions to cetuximab; investigators documented allergic reactions in 0.6% of control subjects in Boston and 20% in Tennessee.[6]

In 2009, a team of allergists from Australia published a study involving 25 patients who had developed allergic reactions after consuming red meat. The study established a strong link between alpha-gal and red meat allergy, as all the patients had experienced tick bites before developing the allergy.[7] Since then, additional reports from various regions around the globe have emerged. In the United States, red meat allergy from tick bites is mainly found in the southeastern region, which correlates with the North American lone star tick (Amblyomma americanum).[1,5,6]

PATHOPHYSIOLOGY

Galactose-alpha-1,3-galactose is an oligosaccharide attached to glycoproteins and glycolipids of cells and tissue of non-primate mammals.[8] Humans do not have the enzyme (alpha 1,3-galactosyltransferase) to assemble alpha-gal from galactose.[8,10] A proposed teleological reason is found in a 1996 paper published in Nature, which showed that anti-gal antibodies in human serum quickly inactivate retroviruses produced from animal cells, thus protecting our distant ancestors from infections.[9] The reason we lack this enzyme remains largely unknown, but it may have an evolutionary basis.

But what about ticks? Some studies have provided a molecular basis for endogenous alpha-gal synthesis in ticks, which appears to be the leading theory.[10] Direct inoculation of alpha-gal from the saliva in a tick's bite results in subsequent IgE-mediated allergic sensitization, leading to severe allergic reactions upon subsequent exposures to alpha-gal.[11,12] Allergists from the University of Virginia have hypothesized that tick bites might be the only cause of alpha-gal syndrome in the United States.[1]

CLINICAL PRESENTATION

The symptoms of alpha-gal syndrome are similar to those of a typical allergic reaction (i.e., urticaria and angioedema).[1] However, this condition differs from other allergic reactions, as it often includes more gastrointestinal symptoms such as nausea, vomiting, diarrhea, abdominal pain, and heartburn.[13] These symptoms are believed to be caused by gut wall edema. The abdominal pain

References available online.

can be so severe that some patients have undergone surgical procedures, including exploratory laparotomy, appendectomy, and cholecystectomy, among others.[13,14] Apart from these symptoms, anaphylaxis caused by alpha-gal is similar to anaphylaxis caused by other factors.

DIAGNOSIS

When diagnosing an allergic reaction caused by alpha-gal, the first step is being aware of the existence of this syndrome and considering it during the evaluation process. It is important to note that anaphylaxis from alpha-gal typically has a delayed onset, with symptoms occurring 2 to 8 hours after eating.[15] This differs from typical anaphylaxis caused by other allergens, where symptoms usually appear within minutes.

To diagnose alpha-gal syndrome, a clinician can send a blood sample on a red-top tube to test for IgE antibodies to alpha-gal. A level greater than 0.1 IU/μL is said to be abnormal.[1,23] It is essential to note that this test will not result during the patient's emergency department visit but can be very helpful during follow-up visits, as the ultimate treatment for alpha-gal is avoidance of possible exposures.

TREATMENT

In instances of anaphylaxis stemming from alpha-gal syndrome, immediate intervention mirrors standard procedures for any anaphylactic response, which include epinephrine, airway management, and resuscitation fluids.[16] Monitor for a minimum of 6 hours; recognize that the potential for biphasic reactions may extend beyond this observation period.[16,17] Another consideration is the use of H1 and H2 blockers and steroids, as the allergen may persist in the digestive tract.[1]

For patients exhibiting solely skin-related symptoms, there is no specific treatment protocol; however, antihistamines may be considered.[1] Upon discharge from the emergency department, patients should receive an epinephrine injector, training on its proper use, preventive measures, and clear return instructions.[1]

CONCLUSION

According to a CDC study, alpha-gal syndrome may affect around 450,000 individuals in the United States. The incidence is expected to rise, likely due to climate changes favoring the expansion of tick populations.[18,19] A recent study revealed that 42% of health-care providers lacked awareness of the syndrome, and 35% were not confident in diagnosing and treating alpha-gal.20 Emergency physicians need to be well-informed about this syndrome to consider it in the differential and, if feasible, initiate a diagnostic evaluation from the emergency department.[1]

Given documented cases of fatal anaphylaxis resulting from alpha-gal syndrome, if suspicion arises, it is prudent to advise patients to avoid red meat and related mammalian products until they consult with an allergist.[1] A list of these products can be found on the CDC website; this information can be printed and given to the patient.

Other recommended actions include discharging patients with an epinephrine self-injector, providing education on its use, and elaborating on preventive measures to reduce the risk of exposure.

Emergency physicians should stay informed about this developing syndrome to ensure timely diagnosis and appropriate treatment. ✶

Alpha-gal syndrome may affect around 450,000 individuals in the United States. The incidence is expected to rise, likely due to climate changes favoring the expansion of tick populations. A recent study revealed that 42% of health-care providers lacked awareness of the syndrome, and 35% were not confident in diagnosing and treating alpha-gal. Emergency physicians need to be well-informed about this syndrome to consider it in the differential and, if feasible, initiate a diagnostic evaluation from the emergency department.

TAKE-HOME POINTS

- Alpha-gal syndrome is an allergy to an oligosaccharide found in non-primate mammals.
- It is estimated that as many as 10% of patients diagnosed with idiopathic anaphylaxis have alpha-gal syndrome.
- Awareness of this syndrome is essential in order to consider it in the differential.
- If feasible, a diagnostic evaluation can be started from the emergency department.

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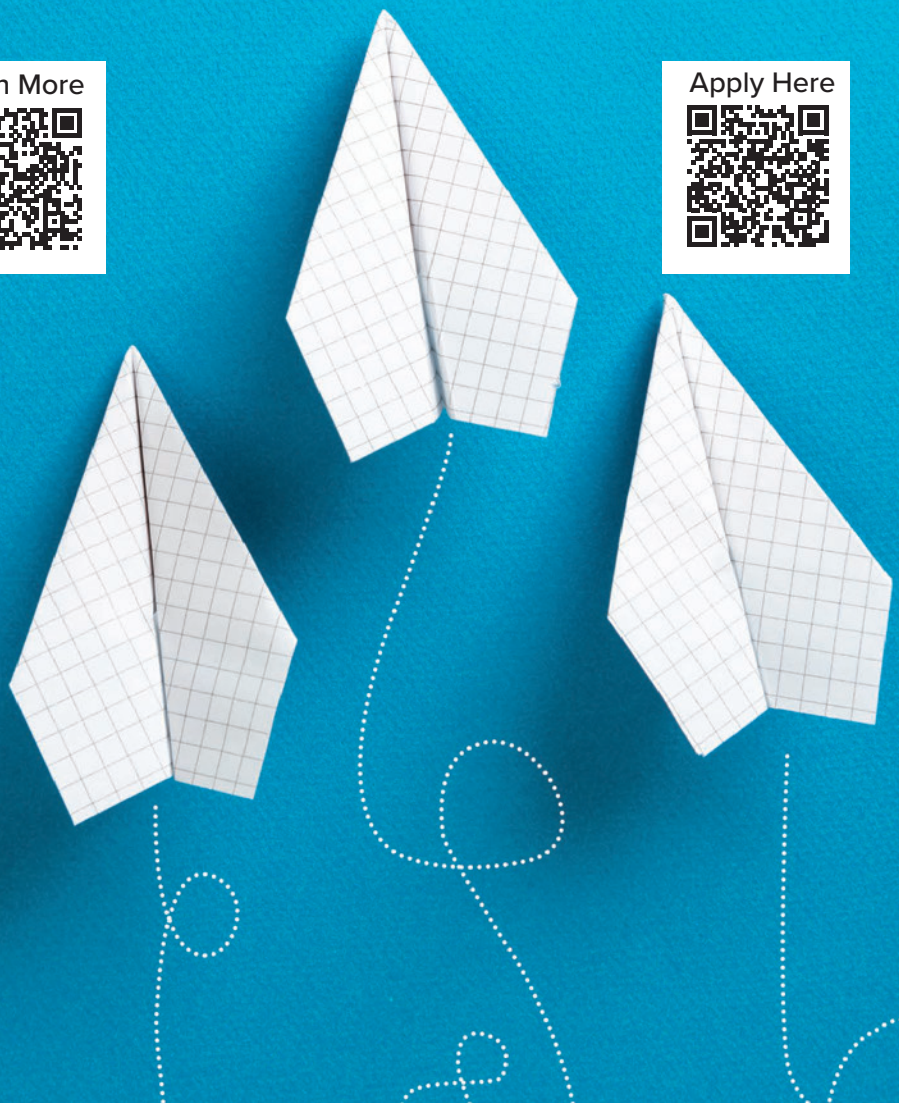

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The Vent Experience: Becoming the Ventilated Patient

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The room was full of 8 other emergency medicine residents (including myself), a respiratory therapist, an EM attending physician, and an critical care fellowship-trained EM attending physician.

At the center of the room was the star of the show: the ventilator itself.

We went through the ground rules first. Try to relax. Don't resist the ventilator. And if you feel like your head is going to explode, then remove the mouthpiece.

Next came the obligatory "No one is required…We only want volunteers" speech.

With that, onboarding was complete.

I stepped up to the vent, grabbed the tubing from the respiratory therapist (RT), applied my nasal clamp, and attached my mouthpiece to the biofilter. With my heart beating quickly, I heard the RT ask, "What settings do you want?"

I was momentarily taken aback. While this was not the first time I had heard this question from an RT, it was certainly the first, and hopefully only, time I would be on the receiving end of these settings. I answered with my standard response, "Volume control."

I took a few deep breaths to check my own capacity and felt good, adding, "Tidal volume 6mL/Kg." Then, not

References available online.

wanting to make it too hard on myself, I said, "Peep 8 mmHg and respiratory rate of 12."

The machine was set. Now all I had to do was relax and let a machine do something that I had been doing all by myself for the last 29 years straight.

There was a moment when I looked at the RT, and she looked at me. This was my last chance to back out. No, I told myself, I was ready, and it was go time. My thoughts drifted back to the feeling I had riding a bicycle for the first time without training wheels; at some point I just had to hop on and start pedaling.

At first, I couldn't figure out how to stop breathing for myself. Do I just hold my breath and wait for the machine to take over? Do I try to time my current breathing with the machine? How do I stop myself from breathing? Can I stop myself from breathing? I decided that starting at the end of my own max tidal volume exhalation would be the best approach. I took 2 deep breaths for luck (and some "breathing room"), exhaled as much air as possible, bit down as hard as I could on the mouthpiece, and waited.

It felt like an eternity.

Possibly secondary to my own residency-related cardiovascular deconditioning, or maybe my tachycardic heart demanding more oxygen, I began to feel that familiar desire to breathe. Suddenly there was a pressure in the tube and my lungs began to expand. The sensation was unique as I felt my lungs being pushed open from the inside. Just when I thought my lungs couldn't expand any farther, the ventilator stopped, and my lungs passively deflated.

To say that the first few breaths were uncomfortable would not be an exaggeration. Luckily, my body adjusted quickly, and then it surprisingly became very easy. I felt almost lazy sitting there without needing to exert effort in breathing for myself. We worked our way through different settings, experimenting with different tidal volumes, respiratory rates, and pressure settings. We even tried more nuanced vent settings, such as airway pressure release ventilation (APRV). Finally, when I had reached the crest of my experience, I removed the mouthpiece and nasal clamp and resumed responsibility for my own ventilation.

The intubation of critically ill patients, and their subsequent placement on mechanical ventilation, is a common scene in the emergency department. In the growing context of hospital overcrowding, the boarding of intensive care unit (ICU) patients in the ED — specifically ventilated patients — is becoming more commonplace. Consequently, emergency physicians are required to manage the vented patient far longer than previously expected.

Having a solid background in ventilator management is becoming something of a necessary skill for the emergency physician. Understanding mechanical ventilation, its different modes and settings, is paramount to providing intubated patients with the proper pulmonary therapy for their individual conditions.

Most ventilator management education for residents occurs during their months spent on ICU rotations; however, familiarity with different vent setting and vent modes is a skill not as easily obtained.

To that end, the actual in vivo exposure of emergency medicine residents to mechanical ventilation allows for a robust kinesthetic learning experience. With a simple mouthpiece and nose clip, residents can experience different ventilator modes through direct connection to an actual functioning ventilator while varying individual tidal volumes, respiratory rates, and pressure settings. This provides a unique understanding of different aspects of mechanical ventilation that are not as well appreciated using other learning methods.

The setup for such an experience is straightforward. All that is absolutely required is a biofilter to keep the circuit sterile for each participating resident, a mouthpiece to attach to the ventilation tubing, and, of course, a ventilator. However, to truly learn from this experience, it is beneficial to have a respiratory therapist present to help with vent settings and adjustments, and an attending physician comfortable with the different vent modes and settings. In our case, this was an EM attending physician with fellowship training in critical care.

During this experience, each resident had a chance to voluntarily "hook themselves up to the vent" via their personal mouthpiece. Starting in volume control/assist control, each resident selected their own personalized tidal volume, respiratory rate, and positive end expiratory pressure, then bit down on the mouthpiece, and tried to relax enough to truly experience mechanical ventilation.

Such experiences certainly cannot replace the more typical didactics-style teaching that provides the required basic understanding of mechanical ventilation common to all EM physicians. These unique experiences, however, can give EM residents a more nuanced understanding of different ventilator modes and build empathy for what our patients actually experience while being treated with mechanical ventilation. ✶

References available online.

Climate Change and Health: Why Emergency Physicians Must Lead the Charge

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Emergency physicians should be environmental activists. The effects of a warming planet contribute directly to exacerbations of chronic obstructive pulmonary disease, asthma, heart failure, atrial fibrillation, cardiac arrest, heat illness, and more.

As we see more severe and frequent extreme weather events, hospital systems will see increased strain — with mass casualty events affecting the function of emergency departments.

As emergency physicians, we should talk about climate change with our patients every day. Health-care providers are in a unique position to influence the social and policy landscape to effect climate change.[1] We should leverage that position to help our patients better understand the links between climate change and human health.

Emergency physicians should acknowledge that climate change is a contributor in the development of pathology. A paper published in 2020 by Sorenson et al describes the range of pathologies that are exacerbated by climate change.[2] This is further outlined by the Environmental Protection Agency (EPA).[3]

Compounding the issue is that climate change affects minority groups and the poor disproportionately.[4,5] These are some of the most vulnerable patients who present to our EDs.

According to the Intergovernmental Panel on Climate Change (IPCC), there are critical areas for change that can have a profound effect on limiting our current

References available online.

emissions and the subsequent impact on the climate. These include:

- Advocating for systemic and policy changes at the institution level[6]
- Transitioning away from fossil fuels to renewable, nuclear, and fossil fuel with carbon-capture technologies[7]
- Land-use change to repurpose for more significant carbon sequestration[7]
- Individual action daily to reduce emissions through consumer-driven change, including choosing renewable sources for energy production, reducing food waste, and reducing consumption of foods that have a high emissions footprint[7]

Possible co-benefits from addressing climate change include biodiversity conservation, water availability, food security, income distribution, efficiency of the taxation system, labor supply and employment, improved urban planning, and the sustainability of developing countries' growth.[7]

CLIMATE CHANGE CONTRIBUTORS, WAYS TO ADDRESS

Since the pre-industrial era, anthropogenic carbon emissions have been driving global warming. This has wide-ranging effects on the climate and inhabitants of the planet.

There is global consensus that we need to keep warming below 2 degrees Celsius by the end of the century to avoid climate catastrophe.[7] We need to reduce carbon emissions significantly over the next decade to achieve this. While eliminating emissions, we need to simultaneously preserve land to act as a sink and sequester legacy carbon.

Johnson et al argued that bold and innovative action is possible and practical for reversing climate change.[8] We need to stop emissions and regenerate ecosystems to sequester carbon. Practically, this means transitioning to renewable energy sources, halting and reversing deforestation, and finding innovative ways to relay this information to the public.

The pathways to achieve this mandate are complex. ACEP has a policy to "advocate for initiatives to reduce the carbon footprint of emergency departments and their affiliated institutions through energy conservation and health care waste reduction and recycling."[9]

To generate willpower for change, we need to see the many co-benefits and leverage them to motivate policy action.

FOSSIL FUEL COMBUSTION

The production of energy from combustion of fossil fuels is a leading cause of health-care sector emissions — so much so that if the U.S. health-care sector was a country, it would be in the top 15 global carbon emitters.[10]

Public sentiment supporting a transition away from fossil fuels as a means for energy production is gaining momentum and spans several industries. The drive for decarbonization of our economy is of utmost importance, and this includes the health-care sector.

Hospital systems have been working to reduce their emissions impact. There are numerous examples of hospital systems shifting to renewable energy and thus reducing their emissions footprint.[11] In 2011, Austin-Travis County EMS saved 14.2 metric tons of carbon emissions by changing its fleet to lighter vehicles and hybrid electric/gas vehicles.[12] A 4-step approach to curbing carbon emissions in Sweden has reduced emissions while simultaneously improving access to health care and more rapid management of disease.[13]

DEFORESTATION

Deforestation may seem like an odd topic to include in an article directed at emergency physicians. However, when addressing the issue of climate change, it is unavoidable.

Deforestation contributes to climate change through land-use change with direct emissions associated with raising livestock and indirectly through reduced carbon dioxide sequestration. Deforestation is driven in large part by farming to support animal agriculture.[14]

Consumption of large quantities of red meat has been associated with increased risk of many chronic diseases, including coronary artery disease, diabetes, hypertension, and cancer.[15] Addressing the effects of deforestation on climate change will have a co-benefit of decreased production of red meat, thus increasing our production of plant-sourced foods for direct consumption.

A dietary pattern more centered on plant-based food will reduce the burden of many chronic diseases and, subsequently, the pressure on EDs for acute exacerbations of these diseases.[16]

Specifically, reducing red-meat consumption and preferentially increasing fruit and vegetable intake could avoid 5.1 million premature deaths per year through 2050.[17] The EAT-Lancet commission demonstrated a reduction of 11 million premature deaths each year through w050 with universal adoption of a plant-rich diet and severe limitation of red-meat consumption.[18]

Encouraging our patients to consume more plant-rich diets is an effective strategy to improve health as well as limit impact of direct and indirect emissions.

CLIMATE CHANGE-RELATED ILLNESS

Air Pollution

The most significant environmental cause of disease and premature death is air pollution. In 2015, diseases caused by pollution were responsible for an estimated 9 million premature deaths, which represents 16% of all deaths worldwide — 3 times more than AIDS, tuberculosis, and malaria.[19]

According to Landrigan, pollution-related diseases also result in health-care spending of up to 1.7% in high-income countries and 7% in middle-income countries, particularly those which are rapidly developing. This represents approximately $4.6 trillion per year. The main contributor to global pollution is the burning of fossil fuels. Combustion of these fuels disproportionately affects low-income countries. Burning of fossil fuels in high- and middle-income countries leads to 85% of airborne particulate pollution in low-income countries, contributing to the burden of climate change-related mortality in those countries.[19–22] Worldwide, air pollution is responsible for approximately 9 million premature deaths annually.

References available online.

Emergency physicians will increasingly shoulder the burden of a warming planet and its effects on human health unless we work to reduce emissions and reverse climate change. We must address the effects of climate change on the progression and exacerbation of disease. We should advocate to our hospitals to introduce changes and make our systems more sustainable and reduce their impacts on carbon emissions.

Air pollution is a mix of solid particles and gases in the atmosphere.[21] Particulate matter emitted from factories, fires, dust, pollen, and mold spores may be suspended in the atmosphere. Ozone is a secondary pollutant that is generated when volatile organic compounds are oxidized in the presence of nitrogen oxides.[23] This ozone is generally concentrated in urban environments and is referred to as smog.

There is a positive association between ozone as an air pollutant and many conditions, including cardiopulmonary disease, hospitalization from all causes, and respiratory diseases (asthma and COPD).[21] Pratt et al performed a burden assessment and found that the number of excess asthma exacerbations in EDs in the United States had a median of 2,403. There was an association with elevated ozone from smoke due to wildfires from 2005-14.[23]

This positive association between exacerbation of respiratory disease and concentration of air pollutants (nitrogen oxide, sulphur oxide, ozone, PM10, PPM2.5) has been demonstrated throughout Asia, Australia, and Europe, representing a significant burden on health-care systems considering future projections of climate change.[24–28]

Allergens and Pollen

Aeroallergens include tree pollen and grass seeds. Climate change affects surface temperatures and alters ecosystems, resulting in prolonged pollen seasons in the spring and increasing grass seed during the summer. These situations are especially susceptible to temperature changes.[29,30]

Demain reviewed the indirect effect of prolongation of pollen seasons due to climate change through alterations in ecosystems.[31] A retrospective chart review performed by May et al of an urban ED showed a positive correlation between asthma exacerbation presentations and tree pollen counts.[5] Neumann projects a 14% increase in ED presentations due to pollen and seed-related exacerbations by 2090. They predict that this increase can be held to 8% with more controlled greenhouse gas emissions.[29]

Diseases Carried by Vectors

With changes and interruptions to ecosystems and human encroachment into previously wild areas, interaction and potential for the breakthrough of vector-borne illnesses has become an area of concern. This has become particularly relevant following the 2020 global pandemic caused by the SARS-CoV-2 virus, which is thought to have come from horseshoe bats via an intermediary species, likely a pangolin.[33]

As human settlements expand and our behaviors increasingly bring us into contact with animals, the risk of zoonotic disease breakthroughs into human populations will remain with associated ecosystem destruction. This presents challenges for emergency physicians and health-care workers alike, as their role in assessing and monitoring for such diseases will become increasingly important.[34]

Temperature Extremes

As we experience the effects of climate change, extreme weather events will continue to become more frequent. These events include extreme heatwaves, which result in the exacerbation of several illnesses.

An analysis of ED visits following a heatwave in Sydney, Australia, in 2011 showed increases in all-cause visits by 2%, all-cause ambulance calls by 14%, and all-cause mortality by 13%, with people over 75 years at greater risk.[35]

In 2003, an unprecedented heatwave in France led to 14,800 excess deaths. The effects were seen most prominently in Paris. The public health department noted 2,600 excess ED visits, 1,900 excess hospital admissions, and 475 excess deaths despite rapid organization aims to prevent harm.[36]

Similar results have been found in the U.S., Asia, and Europe, with increases in ED presentations as a direct effect of heat-related illnesses as well as exacerbations of congestive heart failure, atrial fibrillation, and renal colic.[37–41] Kingsley et al projected up to a 1.6% increase in all-cause mortality and a 25% increase in heat-related mortality by the end of the century if global temperatures continue to rise at current trajectories, with disproportionate effects noted in those under 18 and over 65 years of age.[42]

Wildfires

With wildfires come increased concentrations of particulate matter (PM), which have been associated with exacerbation of respiratory diseases.

There have been significant fire events around the globe since 2000, the effects of which have been well-documented in the medical literature. In 2007, an extensive wildfire event occurred in Victoria, Australia, which showed a positive correlation between PM2.5 secondary to smoke and asthma exacerbation, increasing ED visits by 1.96% on the day of exposure.[43]

A comprehensive analysis of ED visits from 1996-2007 showed increased same-day visits for asthma, COPD exacerbation, and all non-trauma presentations on days when PM10 and PM2.5 were above the 99th percentile, with the effect remaining for all days when the atmospheric particulate matter was elevated. The analysis also noted a 2-day lag in presentation for ischemic

References available online.

heart disease.[44]

Following a Southern California wildfire event in 2007, there was an increase in presentation for dyspnea by 3.2 visits per day and asthma diagnoses by 2.6 visits per day for a single metropolitan ED.[45]

In 2012, a significant wildfire event in Colorado demonstrated a positive association between local concentration of PM2.5 and asthma exacerbation. The effects between exposure and presentation extended for up to 3 days in this study.[46]

These findings have been replicated in numerous studies, showing strong positive correlations between increased concentrations of PM2.5 and exacerbation of respiratory illness (asthma and COPD) that result in presentation to the ED.[47–49]

INCREASED STRAIN ON EDS, EMS

There is expert consensus that with climate change, there will be greater demand on particular hospital departments, namely the ED and on EMS in the prehospital setting. Clinically, emergency medicine will likely see a shift in demand for its services greater than current annual rates. The ED has a broad-based clinical mission and plays a vital role in urgent and emergent ambulatory care, safety-net provision, and increasingly urbanized populations.

As natural disasters such as floods become more regular occurrences as a result of climate change, it will be necessary for organizations and health-care workers to focus on disaster preparedness and take necessary steps to mitigate emissions at a systemic level through policy change and patient education.[38,50,51]

Natural disasters will call upon departments to be resilient and flexible with their systems to care for patients in multiple scenarios. There may be surges of patients within a fully functioning department. There may be direct effects on the department, limiting its capacity to serve its primary function.

In 2017, Hurricane Harvey in Houston resulted in a severe flooding event, which reduced hospital capacity with loss of beds to water damage, reduced staffing, and inability to transfer patients. The department was able to show resilience through flexibility. However, it noted that emphasis needs to be placed on preparedness and flexibility and preventative measures through community education and direct policy action to limit emissions that would worsen climate change.[50]

CONCLUSION

How is all this relevant to emergency physicians? To answer simply: We will increasingly shoulder the burden of a warming planet and its effects on human health unless we work to reduce emissions and reverse climate change. We must address the effects of climate change on the progression and exacerbation of disease. We should advocate to our hospitals to introduce changes and make our systems more sustainable and reduce their impacts on carbon emissions. ✶

References available online.

Hyperviscosity Syndrome: A Differential Diagnosis for Altered Mental Status

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Altered mental status (AMS) is a common presentation to the emergency department. Some studies suggest that mortality rate can be up to 1 in 10 for patients presenting with AMS.[1] In the ED, an AMS patient requires a time-sensitive evaluation with primary survey — ABCDE: Airway, Breathing, Circulation, Disability, and Exposure, adding F for fingerstick glucose and initial vitals.[1,2] After this initial assessment, a history and physical examination will allow you to evaluate for other causes. The differential diagnoses could be categorized as: primary central nervous system (CNS), metabolic, toxicologic, infectious, or other.[1,2]

Hyperviscosity syndrome belongs in the "other" category. It classically presents as a triad of neurological deficits, visual disturbances, and mucosal bleeding. It is also associated with blood dyscrasias such as erythrocytosis, leukocytosis, or thrombocytosis.[3] It is a hematologic-oncologic emergency given that increased plasma viscosity slows blood flow, causing hypoperfusion of tissues.[3]

CASE

A 48-year-old man with a history of hypertension and recently diagnosed bilateral lower extremity deep vein thrombosis presented to the ED via emergency medical service. A family member called EMS when the man became minimally responsive during the day. The patient was on an anticoagulant.

On initial evaluation, the patient was oriented to self and place but appeared confused and lethargic. He was minimally responsive to questions, speaking slowly, and unable to provide a history.

Initial vitals were blood pressure 150/92; pulse 105 bpm; respiratory rate 22; O2 saturation 97% on room air; and temperature 98.8 degrees Fahrenheit.

References available online.

The patient was empirically treated with D50. Physical exam was remarkable for ill appearance, dry mucous membranes, poor skin turgor, disorientation, and inability to follow commands.

Family members reported that the patient had been behaving unusually for the past month and drinking alcohol more than usual after the death of a sibling. Two weeks prior to presentation, he had complained of bilateral leg pain, at which point he was diagnosed with bilateral deep vein thrombosis and started on apixaban. One week prior to presentation, the patient felt nauseous and lightheaded, and family members took him to a hospital. He was diagnosed with decreased renal function and discharged after being managed with diuretics. One day prior to his presentation to our ED, a family member noticed that the patient appeared dehydrated with dry mucous membranes, was speaking incoherently, and fell after standing up.

Initial lab results (**Table 1**) showed normocytic anemia, thrombocytopenia, elevated ionized calcium, and kidney injury.

A non-contrast head CT scan did not show signs of hemorrhage, edema, or tumors. However, chest, abdomen and pelvis CT scans did show evidence of bony lesion at T11 vertebral body (**Image 1**).

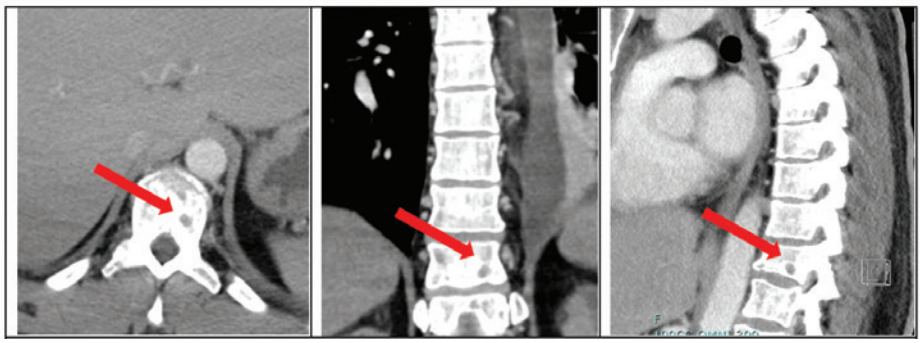

IMAGE 1: CT chest (axial, coronal, and saggital views) showing lytic lesion. CT chest showing bony lesion on T11 vertebral body (shown with red arrow).

TABLE 1. INITIAL LABORATORIES	
CBC	**POC BMP**
WBC 12.5	Na 139
Hgb 8.0	K 4.9
Hct 25.4	CO2 32
MCV 97.3	BUN 53
Plt 132	Cr 8.2
	Ionized Ca 1.91
	Glucose 317

The patient was admitted to the medical ICU for close monitoring of altered mental status. Given his elevated BUN and creatinine, there was a concern for acute renal failure contributing to AMS and a likely requirement of emergent hemodialysis. The nephrology team agreed with beginning emergent hemodialysis and added a new differential of blood dyscrasia because of elevated total serum protein of 16.8.

Additional labs showed the patient had an elevated protein gap (or gamma gap, the difference between total serum protein and albumin) of 14.7, which raised the concern for hyperviscosity syndrome.

Hematology service recommended aggressive IV fluid hydration while preparing for urgent plasmapheresis. The patient promptly received plasmapheresis, which improved his mental status. He later received hemodialysis, which improved his renal failure.

The patient was subsequently diagnosed with multiple myeloma as the cause for hypercalcemia, requiring a total of 6 sessions of plasmapheresis and 2 sessions of hemodialysis. Chemotherapy was initiated. The patient was discharged after 40 days at the hospital.

DISCUSSION

This case was an atypical presentation of hyperviscosity syndrome, given that initial presentation was AMS alone as opposed to the classic triad of neurological deficits, visual disturbances, and mucosal bleeding. Initial management was further complicated by frequent clotting of blood samples and limited amount of information from the patient's past medical history.

However, a tool that showed to be useful was the total serum protein, which is consistent with paraproteinemia most commonly caused by monoclonal gammopathies.[4] This patient presented with hyperCalcemia, Renal failure, Anemia, and Bony lytic lesions (features collectively known together as CRAB), which was concerning for multiple myeloma. The protein or gamma gap is a useful tool for multiple myeloma diagnosis because it has been shown to be a quick assessment of disease burden and treatment response.[5] Although the gold standard for diagnosis is serum and urine protein electrophoresis, the turnaround time is 3 to 7 days. In case of emergent plasmapheresis, it is imperative to not delay treatment.[5]

Initial treatment of hyperviscosity includes consulting the hematology-oncology team for further evaluation and emergent plasmapheresis.[6,7] While plasmapheresis may reduce viscosity by 30-50%, it will not treat the underlying disorder, and patients will be concomitantly started on chemotherapy.[6,7] Chemotherapy is mostly directed toward symptom resolution or improvement rather than viscosity measurement itself because patients may have symptom resolution with an

References available online.

elevated viscosity.[3]

As patients with hyperviscosity syndrome are usually dehydrated, hydration with 1-2 liters of normal saline while awaiting plasma exchange is common practice.[3] Another temporizing measure, especially if plasma exchange is unavailable, is phlebotomy by extracting 1-2 units of blood while concurrently replacing it with normal saline.[3] However, this treatment is generally reserved for severe cases when patients present with coma or seizures.[3]

CONCLUSION

Altered mental status is a common presentation that requires a time-sensitive evaluation, given its mortality rate.1 Although presentation for AMS could be addressed under broad categories such as structural CNS, metabolic, infectious, or toxicologic, an additional category worth considering is blood derangements, such as blood dyscrasias.[1,2] Our case demonstrated the difficulties of a time-sensitive evaluation in a patient with hyperviscosity syndrome.

Important steps to determine patient management include recognizing the CRAB features suggestive of multiple myeloma and determining the gamma gap. In our case, this directed management toward immediate hydration and plasma exchange as temporizing measures. This treatment is meant to improve symptoms but not necessarily tend to the viscosity, although an argument can be made for assessing treatment response via the gamma gap.[5] ✶

Altered mental status is a common presentation that requires a time-sensitive evaluation. Although presentation for AMS could be addressed under broad categories such as structural CNS, metabolic, infectious, or toxicologic, an additional category worth considering is blood derangements, such as blood dyscrasias.[1,2] Our case demonstrated the difficulties of a time-sensitive evaluation in a patient with hyperviscosity syndrome. Hyperviscosity syndrome is a hematologic-oncologic emergency.

TAKE-HOME POINTS

- Consider blood dyscrasias when evaluating altered mental status.
- Hyperproteinemia is mainly caused by monoclonal gammopathy but might be seen in inflammatory diseases. Consider multiple myeloma as a differential for an elevated protein or gamma gap.
- Hyperviscosity syndrome is a hematologic-oncologic emergency. Give intravenous fluids as patients will be dehydrated. In cases of coma or seizure, consider phlebotomy if unable to promptly perform plasma exchange.
- Patients will require admission for further management, which may require multiple sessions of plasmapheresis and testing to determine the etiology that will ultimately guide treatment.

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Introducing the 'Fiver': A Modernized Alternative to the 'B-52' for Acute Agitation Control

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Violence against health-care workers caused by acutely agitated patients is an unfortunate reality for many of us. When attempts at verbal redirection do not successfully de-escalate the situation, expedient medication administration becomes a key step to minimize the damage and maximize the safety of both the patient and hospital staff. If the agitated patient refuses an offer of oral or sublingual medication, clinicians often rely on intramuscular (IM) or intravenous (IV) administration to achieve adequate behavior control.

Many medications are available for use either individually or in combination for the acutely agitated patient. Although the classic "B-52" combination of 5 mg haloperidol, 2 mg lorazepam, and 50 mg diphenhydramine is often considered first-line therapy and has maintained longstanding popularity across emergency medicine, recent literature and specialty society policy recommendations suggest that 1 or more of these medications may not be the optimal pharmacologic choice under these high-stress, time-sensitive circumstances.

To address this, we introduce the "Fiver": a combination of 5 mg droperidol, 5 mg midazolam, and 50 mg diphenhydramine. The name "Fiver" is meant to be a clever memory aid to quickly recall the 5 mg dosing of both the antipsychotic and benzodiazepine medications used.

Similar to the traditional B-52 cocktail, the Fiver includes 1 antipsychotic medication and 1 benzodiazepine medication, used in combination with diphenhydramine. The medication combination included in the Fiver also aligns with the droperidol-midazolam combination that received a Level B recommendation in the most recent ACEP Clinical Policy on Severe Agitation,[1,2,3] with the simple addition of diphenhydramine. Droperidol may be replaced by 5 mg olanzapine based on facility availability or provider preference. Like any reliable anti-agitation cocktail, it can be either IM- or

References available online.

IV-administered.

WHY DROPERIDOL INSTEAD OF HALOPERIDOL?

Droperidol has been shown to have faster onset and greater efficacy compared to haloperidol, with no evidence of increased adverse events. A meta-analysis published by Zun et al.[4] found that 5 mg IM droperidol had effect within 5-10 minutes, compared to 15-30 minutes for 5 mg olanzapine and 20-60 minutes for 5 mg haloperidol.

When you and your staff are dodging punches and trying to avoid showers of spit, these minutes matter. The observational study published by Klein et al.[5] found that patients who received 5 mg IM droperidol or 5 mg IM olanzapine required fewer additional doses for agitation control in the 60 minutes following the initial dose and fewer overall doses during the ED encounter than patients who received 5 mg IM haloperidol. There were no differences in adverse events among the 3 study groups. Taylor et al. found that this clinical effect was further seen when droperidol and midazolam were administered together (as the Fiver proposes), again with no difference in adverse effects.[6]

DOESN'T DROPERIDOL HAVE A BLACK BOX WARNING?

Great question, and we are glad you asked. Droperidol developed a negative reputation when it received a black box warning for QT prolongation in 2001, but current literature has shown that droperidol has a well-established safety profile at doses of 5 mg and lower.[7] In fact, the study published by Klein et al.[5] included nearly 5,000 patients who received 5 mg IM droperidol for agitation control, with zero incidences of torsades de pointes, the dreaded arrhythmia associated with QT prolongation.

ACEP agrees that droperidol is safe in patients without established history of QT prolongation who receive appropriate dosing, as an updated clinical policy was published in 2021 highlighting its safety profile and recommending droperidol use in circumstances including acute agitation.[8]

The potential risk of QT prolongation is further minimized when olanzapine is used as the antipsychotic medication of choice.

WHY MIDAZOLAM INSTEAD OF LORAZEPAM?

Both are effective and either benzodiazepine can ultimately be used, but the current literature demonstrates that midazolam has a faster onset than lorazepam. Goldfrank's *Toxicologic Emergencies* textbook reports the onset of IM midazolam to be 5-10 minutes compared to 20-30 minutes for IM lorazepam, and the onset of IV midazolam to be 1-2 minutes compared to 5-20 minutes for IV lorazepam.[9] Again, in the context of acute agitation control, these minutes matter.

Many facilities across the United States are also facing a lorazepam shortage, making midazolam an ideal replacement both from a clinical and practical standpoint.

WHY INCLUDE DIPHENHYDRAMINE?

Classic teaching dictates that diphenhydramine reduces the risk of extrapyramidal side effects associated with antipsychotic medications such as haloperidol, and the meta-analysis performed by Mokthari et al. supports this.[10] Diphenhydramine also has sedative effects due to its antagonism of the H1 histamine receptor and has been shown to improve sedation for procedures including colonoscopy[11] and bronchoscopy.[12]

BUT WHAT ABOUT KETAMINE?

It is true that ketamine can be rapidly administered for agitation control as well, and 5 mg/kg ketamine monotherapy is discussed in the 2023 ACEP Clinical Policy with Level C recommendations.[1] However, the critical potential side effects of rapid ketamine administration include laryngospasm and emergence reaction, each of which represents added risk to either the patient or hospital staff when encountered. Neither of these side effects are known to be a potential complication of the Fiver.

For these reasons, we propose the Fiver as a modernized alternative to the B-52 for acute agitation control in the emergency department. ✶

Disclaimer: The views expressed in this work are those of the author and do not reflect the official policy or position of the Department of the Navy, Department of Defense, or the U.S. Government.

References available online.

Getting Started in EM Research: Lessons from Rising Stars

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The EMRA Research Committee interviewed 4 soon-to-be legends in the emergency medicine research community, from residents to established faculty, to understand their experiences. Our interviewees — Mat Goebel, MD, MAS; Corey Hazekamp, MD, MS; Vinitha Jacob, MD, PhD; and Laura Harding-Jackson, DO, PhD — discussed how they got their start in EM research, relevant training, current focuses and niches, and more.

EMRA RESEARCH COMMITTEE: How did you get involved in research?

DR. GOEBEL: While I was working as an ED tech, I got involved with data abstraction for QI. I took an interest in driving change with data. The chair of research for the ED took me under his wing to get involved with several research projects, and the rest is history!

DR. HARDING-JACKSON: I became involved in research during my freshman year of college. My first job was actually working in a greenhouse for a lab studying virus spread among biofuel crops. I learned that I loved the scientific method and the lab atmosphere, and I later found my way to a biomedical sciences lab, which is where I discovered my love of medicine.

DR. HAZEKAMP: I became interested in research as an undergraduate at the University of Colorado where I researched the genetic variability of Rocky Mountain bighorn sheep herds which led to my first research grant — a National Science Foundation Research Experience for Undergraduates award. This is a fully funded award meant to enable undergraduate researchers to engage in mentored research for a whole summer, which prompted my interest in graduate school. Originally intending to complete a PhD, during graduate school, I became interested in medicine and pivoted to medical school, where I fostered my passion for research focusing on public health.

DR. JACOB: I was involved in research from a young age, in high school, and continued to stick with it! My early exposure to molecular biology and bench research was eye opening, and I was able to continue this work as it applies to emergency medicine.

EMRA RESEARCH COMMITTEE: What sort of formal and informal training have you had relevant to your research work?

DR. GOEBEL: I took a gap year during med school to get a masters in clinical research from UC San Diego. Following residency, I did a 1-year research fellowship, during which I got a certificate in clinical research from Tufts.

DR. HARDING-JACKSON: I completed my PhD during medical school through a combined DO-PhD training program. Having PhD experience, while not necessary to be involved in research as a resident, has been very helpful in facilitating involvement with more basic science research.

DR. HAZEKAMP: My formal research training was primarily in basic science research; however, I also had informal training in epidemiology, which consisted of basically reading interesting manuscripts and putting together research projects. Since starting residency, I was fortunate enough to have been chosen for a National Institute on Drug Abuse-funded, Society of Academic Emergency Medicine Foundation (SAEM)-sponsored, mentor-facilitated training award, which provides direct experience in researching solutions for substance use disorders through a mentor-guided project. Currently, I am participating in the SAEM ARMED course, which teaches junior researchers about various research techniques and the intricacies required to apply for NIH funding.

DR. JACOB: I have a PhD in biomedical sciences and did a K12 research fellowship.

EMRA RESEARCH COMMITTEE: What is the focus of your current initiatives? How did you find/are you finding your niche?

DR. GOEBEL: I am mainly focused

References available online.

on prehospital research, especially pertaining to STEMI and the use of AI. I just chased what I found interesting. I was originally an EKG tech working on STEMI metrics, and I continued to find interest in that area.

DR. HARDING-JACKSON: As a 1st-year resident, I am just getting started in the emergency medicine research world! I first connected with a few potential mentors during interview season. After I joined the residency program, we met to discuss project ideas that would be feasible with a busy residency schedule. While becoming comfortable in clinical training was my top goal for intern year, it was helpful to explore opportunities for research involvement. I just began a project evaluating markers of neural recovery in anoxic brain injury, and I am excited to see where the work takes us! There is always a component of mentorship to finding this as well. Seek out a mentor who will foster and support your passion and excitement for a subject!

DR. HAZEKAMP: Of course, research is my outlet from the rise and grind of clinical emergency medicine. Currently, my research focuses on how to improve how we treat opioid use disorder in the emergency department, though I'm expanding my scope to include substance use disorders in general. Even as a medical student, it was apparent that we can improve how we treat substance use disorders in the emergency department. EM researchers have produced some practice-changing data in the past 3 years. It's an exciting time to be involved in this area of EM research.

DR. JACOB: I make preclinical models of sepsis and inflammation to test drug candidates. I was able to do so by looking out to see what problems/topics interested me in residency and spending that time to reflect on what sort of research I liked and didn't like.

EMRA RESEARCH COMMITTEE: What motivates you?

DR. GOEBEL: I want to use data to drive patient-care improvements. Also, I will always be chasing the dopamine hit of getting my first publication.

DR. HARDING-JACKSON: I am curious by nature and I love discovering new things! Knowing that a project that I designed or led improved our knowledge of a subject is so rewarding. When it comes to medical research, improving knowledge of a disease process or treatment can mean improving the quality of life for patients to come.

DR. HAZEKAMP: I find research and scholarship to be a mechanism that allows me to avoid burnout in emergency medicine. I was taught early on that establishing a personal niche can prevent burnout. One thing I love about research in general is coming up with a research question, formulating a research plan

References available online.

INTERVIEWEES

Mat Goebel, MD, MAS

MAT GOEBEL, MD, MAS
Dr. Goebel is an emergency physician and former paramedic, EKG tech, and sound engineer. Originally from the Bay Area, he graduated from med school at UC San Diego, where he also obtained a master of advanced studies degree in clinical research. He completed EM residency at UMass Chan Baystate Medical Center. He remained at Baystate to complete an NIH-funded research fellowship, during which he obtained an additional certificate in clinical and translational research from Tufts University. Dr. Goebel is currently an EMS fellow at Baystate.

Laura Harding-Jackson, DO, PhD

LAURA HARDING-JACKSON, DO, PHD
Dr. Harding-Jackson is a 1st-year emergency medicine resident at the University of Michigan. She completed her dual-degree at Michigan State University College of Osteopathic Medicine, where she studied neuroelectrophysiology. She loves EM and is interested in combining her background in neurophysiology with emergency medicine, including seizures, stroke, and anoxic brain injury.

Corey Hazekamp, MD, MS

COREY HAZEKAMP, MD, MS
Dr. Hazekamp is a senior emergency medicine resident at NYC H+H/Lincoln. He completed medical school at the University of Illinois College of Medicine at Rockford and was previously a researcher at the Lurie Children's Hospital of Chicago. His current research interests are in health-care disparities, health equity, and substance use disorders, including social determinants of health like eviction and youth development.

Vinitha Jacob, MD, PhD

VINITHA JACOB, MD, PHD
Dr. Jacob is a clinical assistant professor and former K12 Scholar in the department of emergency medicine at the University of Michigan. She focuses on the development and repurposing of sepsis therapeutics and biomarkers using preclinical models including in zebrafish model systems. She obtained her undergraduate degree in chemical engineering from Princeton University and her MD and PhD degrees from the Icahn School of Medicine at Mount Sinai. She completed her residency in emergency medicine at Yale New Haven Hospital.

and carrying out the work all the way to publication. Seeing my ideas and thoughts in a peer-reviewed publication provides immense personal satisfaction and gives me professional gratification.

DR. JACOB: Mostly, I just inherently love it and am driven by curiosity. I also want to make an impact on sepsis care.

EMRA RESEARCH COMMITTEE:
What skills do you think are most important in emergency medicine research? How have you been gaining those skills?

DR. GOEBEL: I think having some level of coding skills can really set you apart. When you are asking people to get you data, they generally assume you want the data all neat, tidy, and ready to use. Data doesn't come that way, and cleaning the data is a LOT of work. With some basic skills in R you can say, "Hey just give me the raw data and I'll take care of it." People are VERY willing to help you. Same when you work with a statistician. You can say, "I have clean data. I just need help running the right tests." This also lowers the barrier to people being willing to help you.

DR. HARDING-JACKSON: Accountability and proactivity are certainly important skills that come to mind. Working on a research project is very different from a class project. Instead of being given assignments to complete, you design the

References available online.

assignments yourself and then complete them. Holding yourself accountable for the work takes significant self-motivation and discipline, especially during a busy residency schedule. Organization and careful planning are key to managing these commitments.

DR. HAZEKAMP: Organization and patience. In my experience, being prepared and organized can make or break a research project.

DR. JACOB: Persistence, resilience. Learn by doing it. Seek additional training as needed, and find mentors and colleagues who can teach you something.

EMRA RESEARCH COMMITTEE: **What are some challenges (expected or unexpected) that you've encountered in your path to research, and how have you overcome them?**

DR. GOEBEL: Securing grant funding is much much much harder than I expected. Also there is so...much... writing. And I've discovered I don't really like writing, which is a huge (personal) barrier.

DR. HARDING-JACKSON: It can be difficult to make time for research while also fulfilling your role as a medical student or resident. Overcommitment and time management can be major challenges. I make sure to communicate with my mentors early and often to keep them updated on my current schedule and appraised of any conflicts.

DR. HAZEKAMP: Focusing on one question. Sometimes I get so excited about an idea that I have trouble narrowing down my thoughts to a single question that I want to answer.

DR. JACOB: Unexpected results, experiments that don't work out. You have to keep focused and not get dismayed. All results are meaningful.

EMRA RESEARCH

EMRA RESEARCH COMMITTEE: **What advice would you give to folks who want to get started in research, but don't have an EM research infrastructure at their institution?**

DR. GOEBEL: Feel free to cold email and direct message folks at other institutions who are working in areas that you like!

DR. HARDING-JACKSON: Most residency programs will have a point-person to help residents become involved in research, as a scholarly project is a residency requirement. This is a great place to start! Let them know that you are interested in a project and ask if they can point you toward an appropriate mentor. If this is not an option at your program, I would recommend connecting with other residents who do research to ask these questions. You may also search the NIH database for individuals with national grant funding to find active projects with contact information for the lead investigator.

DR. HAZEKAMP: Every institution should have an institutional review board (IRB). I would figure out what you will need to get a project approved by the IRB. Keep your research question focused and narrow. Recognize your limitations. Start with a project that is achievable within 1-2 years. Most importantly, find a mentor.

DR. JACOB: Find ideas/topics that are interesting to you, and talk to others at your institution and even outside of your institution about your thoughts to find mentors.

EMRA RESEARCH COMMITTEE: **What advice would you give medical students and residents interested in research?**

DR. GOEBEL: Make friends with the people doing quality improvement or compliance. They are using 80% of the same skills, and know how to get data.

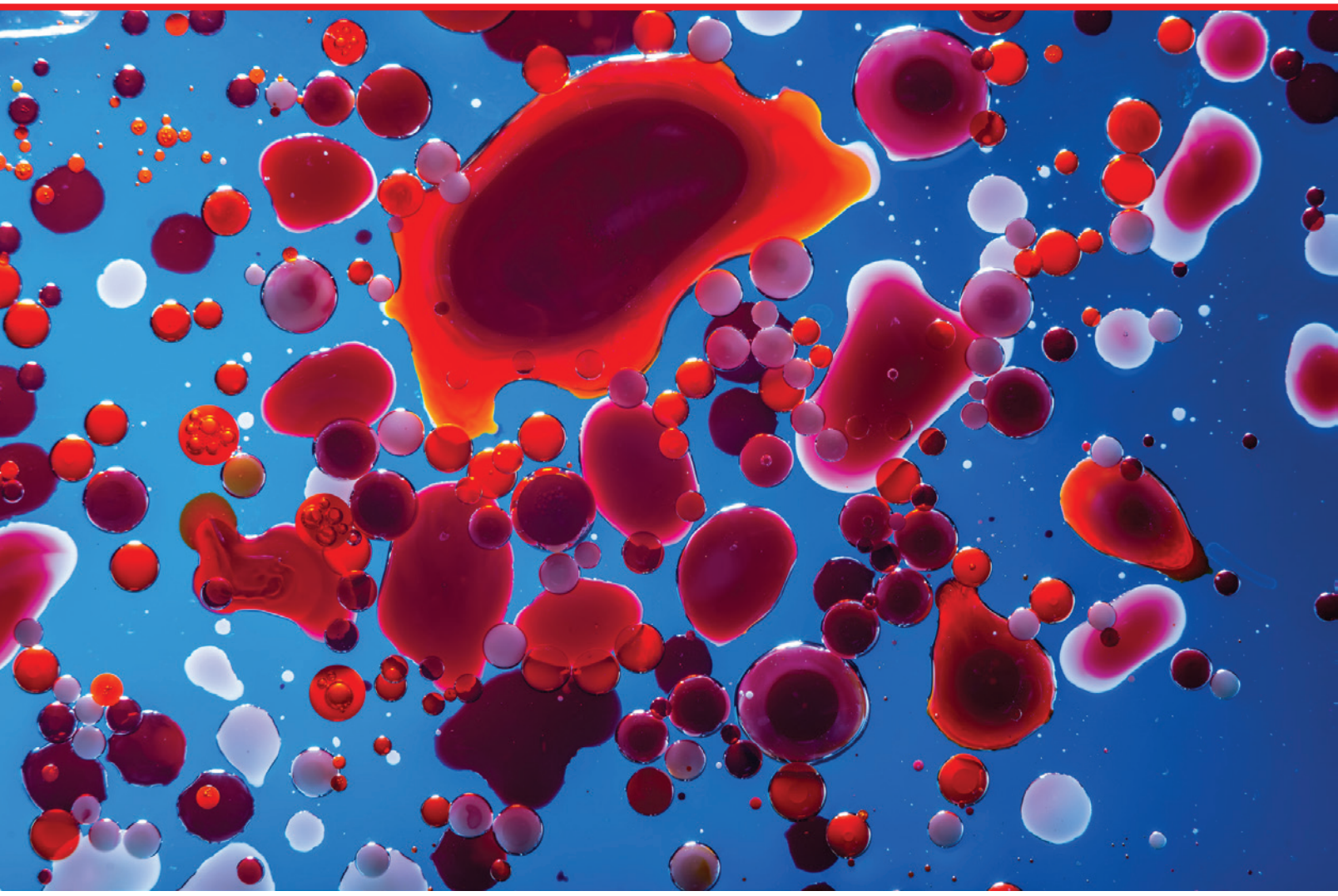
DR. HARDING-JACKSON: 1) Don't be afraid to ask: Email physicians or professors and let them know why you are interested in their work. Ask about possible involvement! 2) Communicate: Always reach out when you are having trouble meeting a deadline or have a conflict arise, student/residency schedules are busy, and most mentors are very understanding of this. 3) Be engaged: Show your mentors that you are excited about the research by asking questions and engaging in conversation about the studies. You do not need to have an MS or PhD in order to be successful in a physician-scientist career path. If you love the science and are motivated to pursue it, you will find opportunities!

DR. HAZEKAMP: Find a mentor. The experiences, knowledge, and skills I've gained from active mentorship have been invaluable. Focus on a topic you enjoy. You'll need to read through a lot of literature; no one wants to read papers on a topic that isn't exciting to them. EM is a broad field. There are a million things you can research — find something that gets you excited.

DR. JACOB: Be a closer — someone who is able to see things through and get your projects across the finish line. This is how you'll gain trust of your collaborators and mentors and be able to get from idea to publication. The only projects that matter are the projects that are taken to the end and published or shared in some way. Just get started; you learn by doing! ✶

Want to learn more about EMRA's Research Committee? Visit emra.org/be-involved/committees/research-committee.

References available online.



Methemoglobinemia: A Case of the Blues

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INTRODUCTION

Methemoglobinemia is a rare but serious — yet treatable — hematological condition that can lead to death if not identified in a timely manner.

In methemoglobinemia, the iron in hemoglobin becomes oxidized. In a normal erythrocyte, the ferrous (Fe2+) state allows oxygen to easily offload to oxygenate tissues. However, in methemoglobinemia, the iron on the hemoglobin is oxidized and becomes the ferric (Fe3+) state.

When this happens, the oxygen does not get picked up by the hemoglobin and, therefore, does not oxygenate the tissue. Furthermore, if only a portion of the red blood cell contains the ferric state and the other contains the ferrous state, the ferrous state is still unable to pick up the oxygen as it causes a left shift in the hemoglobin curve (**Figure 1**). This causes decreased oxygen delivery to the tissues.[1]

Methemoglobinemia can be acquired or hereditary.[2] Topical benzocaine is a known cause of acquired methemoglobinemia and is applied for transesophageal echocardiograms, among other procedures and uses. Cyanosis, a low pulse oximetry reading, and a high partial pressure of arterial oxygen on arterial blood gas (ABG) should aid in diagnosis.

Methemoglobinemia is a serious condition and can lead to death. Although benzocaine is known to cause methemoglobinemia, it is still rare.[3]

In a large case series study, methemoglobinemia occurred in 1 out of 1,499 transesophageal echocardiograms (TEE). In another study — a random

References available online.

Methemoglobinemia — a serious but treatable condition that presents with generalized cyanosis, an oxygen saturation percentage of mid- to high 80s, high pO2, dark-colored blood, and hypoxia resistant to 100% oxygen — can result from topical benzocaine. It is important to consider methemoglobinemia and perform an ABG if a patient presents similarly. If such signs are present along with a methemoglobin level of 30% or greater, and methemoglobinemia is determined to be acquired, then methylene blue is the treatment of choice.

sample of 190 patients with the same sedation, sex, body mass index, and left ventricular systolic function — patients who developed methemoglobinemia were found to be anemic, hospitalized, or with acute systemic infection.[3]

An ABG is crucial in diagnosing methemoglobinemia and should be the first test. Most ABGs can detect methemoglobinemia by its absorbance spectrum of 631 nanometers (expressed as a percentage).

Partial pressure of arterial oxygen (pO2) is an indicator of how much oxygen is being delivered to the tissue and how much dissolved oxygen is in the blood. Methemoglobinemia causes a falsely elevated pO2.[4]

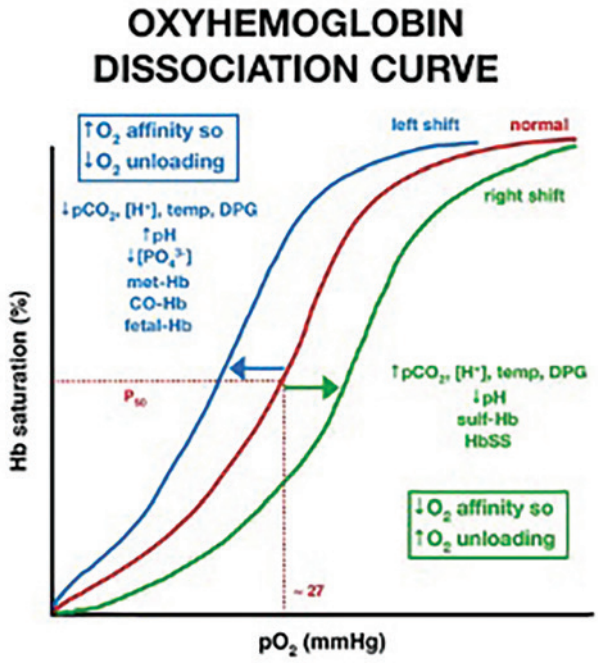

FIGURE 1: Left shift resulting in hemoglobin not being able to pick up oxygen in methemoglobinemia.

Pulse oximetry cannot determine how hypoxic a patient truly is in methemoglobinemia. The wavelength of the pulse oximetry can absorb the light of methemoglobinemia and is unable to determine if red blood cells (RBC) are saturated or not.[5] An oxygen saturation of 85% is caused by a high percentage of methemoglobinemia.[6]

A retrospective series, performed in 2 teaching hospitals involving 138 patients with acquired methemoglobinemia, revealed the majority were caused by dapsone. However, 5 of the participants were, in fact, associated with 20% benzocaine spray with a mean methemoglobinemia level of 43.8%. Furthermore, it was shown that 94% of all the subjects in this study were anemic.[7]

CASE REPORT

The patient in our case was a 76-year-old Caucasian woman with multiple comorbidities, including a history of type 1 diabetes and a lumbar laminectomy 2 months prior. She presented to the ED with recurrent surgical site pain and a fever of 101 degrees Fahrenheit. Her previous laminectomy was complicated by methicillin-resistant staphylococcus aureus (MRSA), necessitating incision and drainage and treatment with intravenous daptomycin for 6 weeks. Blood cultures were obtained, and the patient was discharged due to negative workup in the ED. Two days later, the blood cultures were found to be positive for MRSA, and she was called to return to the hospital.

The patient was admitted that day for bacteremia and diabetic ketoacidosis (DKA). DKA was effectively treated with intravenous (IV) insulin and IV fluids, and it resolved 2 days later. Because of the positive MRSA blood cultures, the patient was started on daptomycin twice a day. Infectious disease and orthopedic specialties were consulted to assist in management. An MRI was performed upon the orthopedist's recommendation. The MRI revealed a deep lumbar fluid collection.

The patient was taken to the operating room 2 days after admission for excisional debridement of lumbar wound. Infectious disease recommended 6 additional weeks of IV daptomycin, followed by an indefinite course for suppression if cultures were sensitive to daptomycin.

A transthoracic echocardiogram was ordered 1 day before the orthopedic surgery and exhibited findings concerning for a small vegetation that was mobile on the posterior leaflet of the mitral valve. As a result, cardiology was consulted. Cardiology recommended a TEE. The patient was taken to the catheterization lab 4 days later for a TEE. There, she received benzocaine spray before the procedure.

After the TEE, she became very cyanotic and was having difficulty breathing, with an O2 saturation of 85-88%. She was placed on bilevel positive airway pressure (BIPAP) ventilation with

References available online.

FiO2 of 100%. At that time, an ABG was drawn, and the blood appeared dark. The ABG showed pH of 7.48, pCO2 of 44.9 mmHg, pO2 of 574 mmHg, and HCO3 of 33.2 nmol/L.

A computed tomography pulmonary angiogram was performed to rule out pulmonary embolism; it was negative. Despite BIPAP of FiO2 of 100%, the patient remained diffusely cyanotic. She was transferred to the ICU, and another ABG — testing specifically for methemoglobin levels — was completed. Her methemoglobin was found to be 42.3% (**Figure 2**). She received 1 dose, 100 mg (1.5 mg/kg), of methylene blue.

Another ABG was done 1 hour later. Methemoglobin level was 0.5%, showing improvement. She was taken off BIPAP after the methylene blue infusion, as her hypoxia resolved. She was tested for glucose 6 phosphate dehydrogenase (G6PD) deficiency, which resulted as 262, the normal value being 127-427 U/10E12 red blood cells. TEE showed no endocarditis. The MRSA was found to be sensitive to daptomycin, and this regimen was continued.

The patient remained clinically stable and was moved out of the ICU the following day. Three days later, a peripherally inserted central catheter (PICC) line was placed, and she was discharged home where she would continue to receive daily antibiotic infusions.

Patient's Arterial Blood Gas	
Hemoglobin	11.9 g/dL
SO_2	96.90%
O_2Hb	58.30%
MetHb	42.30%

FIGURE 2

DISCUSSION

Determining the cause of methemoglobinemia is important because the cause determines treatment. In fact, first-line therapy is contraindicated in some types of inherited forms.[8]

In acquired methemoglobinemia, it is important to diagnose promptly and begin treatment immediately to resolve hypoxia. Medications, whether used appropriately or in settings of overdoses, are the main contributors to acquired methemoglobinemia.[9]

In our case, topical benzocaine was the culprit. However, how benzocaine causes methemoglobinemia isn't well-known.[2]

The studies described earlier were similar to our presented case. Oxygen saturation of 85-88%, abnormally high pO2 of 574 mmHg, BIPAP with FiO2 of 100% that did not resolve hypoxia, and dark-appearing blood were all characteristic signs of methemoglobinemia, as seen in the aforementioned studies. Our patient's methemoglobinemia was 42.3%, similar to the mean values in previous studies, specifically acquired methemoglobinemia after TEE.[7] Furthermore, she had bacteremia, which further increased her likelihood of acquiring methemoglobinemia.[3] Also, 1 dose of methylene blue caused her to revert to her previous state, further confirming an acquired etiology.

If methemoglobin level is 20-

References available online.

45%, dizziness, dyspnea, tachycardia, coughing, weakness, and headache can occur. If the level is greater than 45%, decreased levels of consciousness and fatigue can occur. When above 55%, cardiac arrhythmias or failure, acidosis, seizure, and coma can occur. If 70% is reached, death can occur.

First-line treatment (if levels are above 30%) is 1-2 mg/kg of IV methylene blue.[10] Methylene blue reduces the oxidized state of hemoglobin back to the ferrous state (Fe2+), allowing the hemoglobin to readily pick up oxygen again, resulting in offloading to tissues.

Vitamin C works in the same manner and can be used to treat if methylene blue is contraindicated. Some contraindications for methylene blue are G6PD deficiency because it can cause hemolytic anemia, and pregnancy (category X) because it can lead to intestinal atresia and fetal death, especially if given in the second trimester.[11] Furthermore, G6PD deficiency should be considered if the patient does not improve after the first dose of methylene blue. An exchange transfusion can be considered if methylene blue does not resolve methemoglobinemia.[10]

Because acquired methemoglobinemia is a serious condition caused by an array of medications,[9] emergency physicians as well as other care providers should know how methemoglobinemia presents, and all should be familiar with the treatment of choice.

CONCLUSION

Methemoglobinemia — a serious but treatable condition that presents with generalized cyanosis, an oxygen saturation percentage of mid- to high 80s, high pO2, dark-colored blood, and hypoxia resistant to 100% oxygen — can result from topical benzocaine. It is important to consider methemoglobinemia and perform an ABG if a patient presents similarly. If such signs are present along with a methemoglobin level of 30% or greater, and methemoglobinemia is determined to be acquired, then methylene blue is the treatment of choice. ✶

TAKE-HOME POINTS

- Consider methemoglobinemia if oxygen saturation level is mid- to high 80s, cyanosis is not improving with 100% FiO2, and dark-colored blood is present.
- Treat with methylene blue when methemoglobin is 30% or above and methemoglobinemia is determined to be acquired.
- Methylene blue is contraindicated in pregnancy and G6PD deficiency. Vitamin C and exchange transfusions are alternative treatments.

References available online.

Heart Failure in the Young Patient: A Case of Reversible Cardiopulmonary Dysfunction

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CASE PRESENTATION

A 36-year-old woman presented to the ED at a large national referral hospital in Uganda. She was referred there from a regional hospital, where she had been diagnosed with refractory heart failure. Despite being treated with diuretics as well as empiric thiamine for possible beriberi, her symptoms had progressively worsened.

The patient had a 1-year history of worsening neck swelling, a 2-week history of bilateral lower extremity edema, and difficulty breathing. Associated symptoms included palpitations, easy fatigability, orthopnea, dry cough, unintentional weight loss, excessive sweating, fever, and heat intolerance. Other symptoms included headache, dizziness, blurring of vision, and diplopia.

A review of her prior medical history revealed that the patient was treated for pulmonary tuberculosis (PTB) 3 years prior but did not finish the course of antibiotics. Repeat sputum testing for TB was negative. She had no other known chronic illness or medication, reported regular alcohol use with a CAGE score of 3, denied tobacco abuse, and worked as a produce vendor.

On examination, the patient was noted to be alert, had obvious proptosis, was tremulous, and exhibited severe muscular wasting. An anterior neck mass was noted, which measured 5x5 cm. This mass was noted to be firm, non-tender, mobile with swallowing, and had no retrosternal extension. Pemberton's sign (in which the patient raises their arms to assess for facial plethora that would suggest venous obstruction) was negative.[1]

Vital signs were notable for a respiratory rate of 24 breaths/min, SpO2 89% RA, BP 131/73 mmHg, HR 125 beats/minute, and temperature 39.6 degrees Celsius. On chest auscultation, the patient had soft, low-pitched breath sounds with bilateral inframammary crackles. Heart sounds were notable for regular rate and rhythm with a pan systolic apical murmur radiating to the axilla but no rubs or gallops. She

References available online.

appeared clinically dehydrated, had mild pallor, grade II edema of the bilateral lower extremities, increased JVP, and positive hepatojugular reflux. She had no focal neurological deficits.

Prioritizing resuscitation, including airway, breathing, circulation, disability, and exposure, supplemental oxygen was administered for hypoxia at 15 L/minute, and fever was treated with intravenous (IV) paracetamol. Laboratory workup (**Table 1**) was notable for normal anion gap metabolic acidosis, acute kidney injury, hyperbilirubinemia, transaminitis, mild leukocytosis and anemia, and leukocyturia.

Given concern for thyroid storm, the patient was started on carbimazole 10 mg per os (PO) every 8 hours, propylthiouracil (PTU) 300 mg PO every 6 hours, dexamethasone 6 mg IV every 12 hours followed by prednisolone 15 mg every 24 hours, and propranolol 40 mg PO every 12 hours. Lugol's iodine 2 drops PO was started 1 hour after the PTU and continued every 8 hours. Due to ongoing hypertension despite the propranolol, losartan 50 mg PO once daily was also initiated.

Point-of-care echocardiogram (ECHO) in the ED revealed LV hypertrophy with reduced ejection fraction, an incompressible inferior vena cava (IVC), and B-lines suggestive of acute pulmonary edema. An electrocardiogram (ECG) was obtained after the initiation of empiric therapy for thyroid storm and revealed sinus tachycardia. Chest X-ray was notable for small bilateral pleural effusions with cardiomegaly. Thyroid function tests (**Table 2**) resulted after the initiation of empiric antithyroid therapy while the patient was still in the ED and confirmed the suspected diagnosis of hyperthyroidism.

On day 2 of admission, the most likely diagnosis was felt to be dilated cardiomyopathy and congestive heart failure secondary to thyroid storm. Although TSH receptor antibody (TRAb) was not available for confirmation of Graves' disease, this was presumed to be the underlying cause of the thyrotoxicosis, given that it is the most likely cause of hyperthyroidism in young patients. Cardiology and endocrinology were consulted, and the patient was admitted to the endocrine ward.

TABLE 1: Diagnostic Workup

TEST	RESULT	NORMAL RANGE
Sodium	135	136 - 145 mEq/L
Potassium	3.9	3.5 - 5.1 mEq/L
Chloride	112	90 - 110 mg/dL
Bicarbonate	14	22 - 28 mEq/L
Blood urea nitrogen	9	6 - 20 mg/dL
Creatinine	1.7	0.5 - 0.9 mg/dL
Glucose	125	70 - 115 mg/dL
Calcium	7.9	8.6 - 10.2mg/dL
Magnesium	1.8	1.7 - 2.5 mg/dL
Total bilirubin	1.4	0.2 - 1.2 mg/dL
AST	58	8 - 33 U/L.
ALT	46	4 - 36 U/L
ALP	46	44 - 147 IU/L
Albumin	3.2	3.4 - 5.4 g/dL
WBC	13.6	3.6 - 11.0 cells/µL
Platelets	233,000	150 - 372,000/µL
HGB	9.5	10.3 - 15.1 g/dL
HCT	32.6%	31.2 - 45.4%
PT	14.2	11 - 13.5 seconds
INR	1.0	0.90 - 1.10
Absolute lymphocyte count	6.3	0.9 - 2.9
Absolute neutrophil count	8.1	1.5 - 7.0
Urinalysis	Leukocytes, nitrites negative	Negative leukocytes/ nitrites
MTB real-time PCR	MTB not detected	

WBC = white blood count, HGB = hemoglobin, AST = aspartate aminotransferase, ALT = alanine aminotransferase, PT = prothrombin time, INR = international normalized ratio, MTB = Mycobacterium tuberculosis, PCR = polymerase chain reaction

TABLE 2: Thyroid function test report

Free T3	44.00	2.30-4.20 pg/mL
Free T4	Not reported	
TSH	0.010	0.60-4.0IU/Ml
TSH receptor antibody (TRAb)	Not reported	

DISCUSSION

Thyroid storm is a life-threatening spectrum of hyperthyroidism often caused by either poorly treated/ undiagnosed hyperthyroidism or excessive use of thyroid hormone replacement therapy.[2] It is a rare medical emergency with an estimated prevalence of 16% in patients hospitalized with thyrotoxicosis.[2]

Although the exact mechanism of thyroid storm is unclear, theories such as a rapid increase in thyroid hormone levels, sudden cessation of antithyroid drug therapy, increased sensitivity

References available online.

to catecholamines, and heightened responses to thyroid hormone at the cellular level have been documented.[3]

Though frequently preceded by an acute precipitating event such as infection or thyroid surgery, more than 25% of cases of thyroid storm may not have any identifiable precipitating factor.[4]

In the ED, thyroid storm is often seen in patients with undiagnosed or untreated hyperthyroidism,[4] a similar presentation in our patient. Generally, Graves' disease is the most common cause of thyroid storm (as with thyrotoxicosis); other causes include toxic multinodular goiter and toxic adenoma.[5]

While the presentation is often similar to that of thyrotoxicosis, thyroid storm can present with cardiopulmonary manifestations that can be life-threatening[6] with reduced exercise tolerance being the most common cardiovascular manifestation. Patients with goiter may demonstrate a positive Pemberton's sign in which bilateral arm elevation causes facial plethora and engorged neck veins due to obstruction of the thoracic inlet by the enlarged thyroid. Although less common, atrial fibrillation (AF), pulmonary hypertension, angina pectoris, and reversible heart failure with reduced ejection fraction occur in about 1-6% of patients with undiagnosed hyperthyroidism.[7]

This case underscores that thyrotoxicosis and/or thyroid storm can induce heart failure in patients without pre-existing heart conditions, a phenomenon that has been documented as thyrotoxic cardiomyopathy (TCM).[8]

TCM occurs in 1-2% of subjects with untreated/poorly controlled hyperthyroidism commonly associated with Graves' disease.[2] A longstanding hyperdynamic state demonstrated by volume expansion, increased resting heart rate, enhanced LV contractility, and reduced systemic vascular resistance collectively result in an excessive increase in cardiac output before the eventual development of high-output heart failure.[9] AF (if it occurs) further impairs the systolic dysfunction in thyrotoxicosis,10 resulting in the compromise of LV filling and, hence, diminished cardiac output. Combined with respiratory muscle weakness associated with thyrotoxicosis, this leads to cardiopulmonary failure, as seen in this patient. Both inspiratory and expiratory muscles are affected by thyrotoxicosis, often causing type 2 respiratory failure, which rapidly improves with antithyroid therapy.[11,12]

It should be noted that thiamine deficiency (i.e., beriberi) can also cause high-output heart failure and can present with similar symptoms to TCM.[13] Though thiamine deficiency is not uncommon in low- and middle-income countries,[14] and alcohol use disorder places patients at higher risk for this condition,[13] the patient in this case did not improve with the administration of empiric thiamine. In addition, she had additional clinical examination findings (such as proptosis) and diagnostic findings (such as low TSH and elevated T3/T4), which pointed to TCM as the underlying etiology of her symptoms.

MANAGEMENT

Unlike many causes of heart failure often requiring diuresis, TCM responds well to rate control with reversal of hyperthyroidism. Cardiac remodeling seen in TCM is also frequently reversible, as evidenced by the restoration of normal LV function after attaining euthyroidism in patients who initially developed a reduced ejection fraction of <50%.[15] Control of the sympathomimetic symptoms and inhibition of synthesis and release of thyroid hormones prevents decompensation.[16,17] This was the case for our patient, who initially had a reduced EF on bedside ECHO, which normalized upon becoming euthyroid on follow-up.

Because of the life-threatening nature of this condition, early diagnosis and treatment are essential. In the ED, certain scoring criteria — such as Burch-Wartofsky Point Scale (BWPS) — have been suggested,[18] based on the degree of organ dysfunction in the setting of thyrotoxicosis. In this scoring system, a score of 45 points or more is highly suggestive of thyroid storm, whereas a score below 25 makes thyroid storm unlikely, with a score of 25-44 suggestive of an impending storm.[18] Although the BWPS has been documented to have a low specificity,[19] our patient scored 65.

In combination with physical manifestations of thyroid disease, this was highly suggestive of thyroid storm, which informed the decision to initiate treatment of storm-awaiting low TSH levels and the abnormally high free T3/T4 levels that were later demonstrated in this case. Although we were unable to obtain laboratory markers for Graves' disease, examination findings in support included diffuse goiter and orbitopathy.[20] Other clues for Graves' disease in this case included risk factors such as the female sex and age between 30-50 years.[21]

The mainstay of therapy for this patient involves beta-blockers, glucocorticoids, thionamides, and iodine.[17,22] Thionamide therapy involves the use of antithyroid drugs such as propylthiouracil and methimazole to inhibit thyroid peroxidase (TPO), the chief enzyme responsible for the formation of T3 and T4.[10] In addition to the antithyroid effects, early initiation of thionamides has also been shown to improve or even reverse heart failure.[23]

Likewise, beta-blocker therapy offers similar cardiac benefits and can reduce or even completely resolve cardiomyopathy, particularly when prolonged tachycardia is likely the cause, as seen in tachycardia-mediated cardiomyopathy.[4] Evidence also suggests that PTU and propranolol prevent the conversion of T4 to T3, contributing to the reversal of thyrotoxic cardiomyopathy.[24] The left ventricular ejection fraction (LVEF) subsequently may increase by 28% to 55% after treatment for thyrotoxicosis.[17] Propranolol with beta-2 blockade increases systemic vascular resistance, abating cardiovascular collapse and potentially preventing AF (the most common cardiac arrhythmia seen in hyperthyroidism, occurring in up to 15% of patients).[25]

Glucocorticoids such as hydrocortisone and dexamethasone decrease the peripheral conversion

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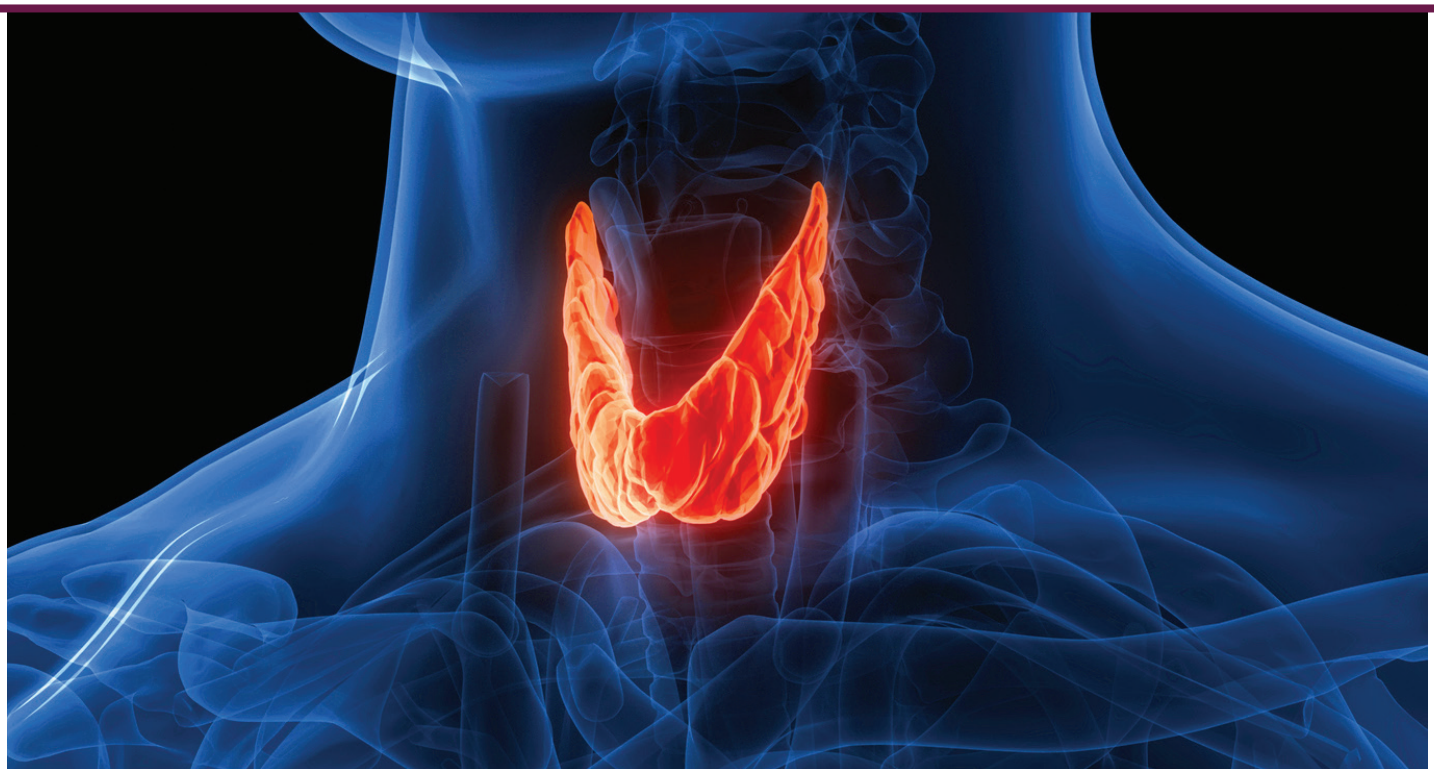

of T4 to T3, prevent relative adrenal insufficiency due to hyperthyroidism, and help improve vasomotor symptoms.

Similarly, iodine compounds (i.e., Lugol's iodine or potassium iodide), administered at least 1 hour after the thionamide, block the release of preformed thyroid hormone, a paradox known as the Wolff-Chaikoff effect (which lasts for up to 2 weeks).

Administration of iodine compounds less than 1 hour after the thionamide can cause paradoxical worsening of symptoms by increasing thyroid hormone production.[26]

One novel component to be considered for the management of refractory hypertension despite beta-blockade in patients with thyroid storm is the addition of an angiotensin 2 receptor blocker (ARB), which was administered in this case. Thyroid hormones are suggested to upregulate angiotensin 2 receptors in the cardiac conduction tissue, which causes remodeling of the myocardium in cases of dilated cardiomyopathy. Literature suggests that ARBs may help reduce this remodeling, thus combatting one of the underlying causes of heart failure in thyroid storm.[27]

Finally, the definitive treatment for thyroid storm is the removal of the underlying cause, which likely represents surgery or radioactive iodine ablation therapy for a patient with Graves' disease.

CASE RESOLUTION

The patient showed remarkable improvement during admission while receiving ongoing thionamide, steroid, iodine, and propranolol therapy. Conventional heart failure therapy, including diuretics, was not utilized given that the suspected underlying cause was thyroid storm.

By day 2 of admission, the patient's oxygen requirements had decreased, and oxygen was completely weaned by day 3. She was discharged 10 days after hospital admission with a plan for total thyroidectomy upon reaching a euthyroid state. Discharge prescriptions included PTU, propranolol, Lugol's iodine, and prednisolone.

On endocrinology follow-up 2 weeks after discharge, the patient was clinically stable with normal vital signs, clear lungs to auscultation, and resolution of peripheral edema. Her bedside ECHO demonstrated a normal EF. Repeat laboratory testing revealed normalization of her TSH and free T3 (T4 testing was unavailable). The patient was subsequently referred to endocrinology for further care. ✶

TAKE-HOME POINTS

- Thyroid storm is a potentially fatal manifestation of untreated thyrotoxicosis and may result in cardiopulmonary dysfunction.
- Cardiopulmonary failure is potentially reversible with prompt antithyroid therapy.
- Physicians should have a low threshold to treat and test for thyrotoxicosis in young patients with new-onset heart failure, as early treatment has the potential to improve patient outcomes.

References available online.

Not Just Rectal Bleeding: A Case of Fournier's Gangrene

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CASE PRESENTATION

A 59-year-old man with a history of recently diagnosed anal squamous cell carcinoma and alcohol use disorder presented to the ED with rectal bleeding and pain that had worsened over a week. On arrival, the patient had low-normal blood pressure but otherwise normal vitals, and was uncomfortable appearing and rigoring. An exam revealed perianal erythema, fluctuance, induration, and tenderness, with necrotic bullae in the perineum. General surgery was urgently consulted, and the patient received fluid resuscitation and broad-spectrum antibiotics.

Labs were significant for WBC 31.2 bil/L, Hgb 12.3 g/dL, Na 135, Cr 2.3, CO2 14 mmol/L, anion gap 26, glucose 149 mg/dL, INR 1.3, CRP 529 mg/L, and lactate 12.6 mmol/L. A CT abdomen/pelvis with contrast revealed stranding and gas tracking from the right buttock and perineum through the retroperitoneum to the aortic bifurcation in the abdomen, concerning for an ascending necrotizing infection such as Fournier's gangrene.

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Fournier's gangrene is a rapidly progressive necrotizing soft tissue infection of the perineal, genital, or perianal regions. An eponymous disease named after French dermatologist and venereologist Jean Alfred Fournier (1832–1914 CE), Fournier's gangrene has been described even in earlier historical records by Persian physician Abu Ali al-Husayn ibn Sina (980–1037 CE).[1]

While rare (<0.02% of hospital admissions), Fournier's is highly morbid with a mortality rate approaching 40% even with optimal treatment; delays in diagnosis and care can increase the mortality rate to 88%.[1] Key risk factors include diabetes mellitus, alcohol use disorder, immunosuppressive states, colorectal malignancy, recent trauma or surgery to the area, and the use of SGLT2

References available online.

inhibitors, although a significant portion (up to 30%) of patients do not have any comorbidities.[1,2] Male sex is associated with a higher incidence of Fournier's, but female patients are more acutely ill upon presentation, experience longer hospitalizations, and have higher rates of multiorgan failure and case fatality.[2]

Necrotizing soft tissue infections are typically categorized by the involvement of any or multiple soft tissue layers, including cellulitis (epidermis, dermis, subcutaneous tissue), fasciitis (fascia), and myositis (muscle). In Fournier's, infections are typically polymicrobial involving genitourinary, gastrointestinal, and cutaneous organisms which produce endotoxins and tissue-destructive enzymes that facilitate rapid extension along fascial planes into surrounding pelvic organs and deeper structures.[1,2] This results in fulminant tissue destruction, ischemic gangrene, and often systemic toxicity.

PRESENTATION AND DIAGNOSIS

Fournier's gangrene, as with other necrotizing soft tissue infections, is a clinical diagnosis. Early clinical manifestations include localized erythema without sharp margins, edema and induration, itchiness, and tenderness. As these findings can be present in much more common infections and dermatologic conditions, early cases can be very difficult to identify — up to 75% of early cases are misdiagnosed.1 Later manifestations include bullae formation, crepitus, skin necrosis, and purulent drainage.[1]

Clinicians should thoroughly inspect the genitourinary and perianal regions for these malignant and sometimes indolent findings, especially in patients who appear systemically ill with fever, malaise, or hemodynamic instability. Severe pain out of proportion to exam should also raise clinical suspicion for a necrotizing infection.[1]

Laboratory testing is nonspecific and should not be used in isolation to diagnose Fournier's, but it can assist with triage and prognostication. Patients may have a leukocytosis with left shift, elevated inflammatory markers, electrolyte abnormalities such as hyponatremia, metabolic and lactic acidosis, and signs of renal failure. Blood cultures may be positive in a subset of patients but more so in monomicrobial necrotizing infections.[3]

While the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score is a clinical decision tool used in the diagnosis of necrotizing infections, its sensitivity for Fournier's gangrene among ED patients is unacceptably low (68-80%).[2] However, one study suggests that a LRINEC score ≥9 may be a useful predictor of mortality in Fournier's.[4] Other risk calculators including the Fournier's Gangrene Severity Index (FGSI) have been developed to predict disease severity but are not routinely used in the ED setting.[2]

Imaging can help diagnose, but cannot rule out, Fournier's. CT is the most sensitive (88.5%) and specific (93.3%) in diagnosing a necrotizing infection.[1] The presence of gas on X-ray is highly specific (94%), but poorly sensitive (49%).[2] Point-of-care ultrasound can be utilized to identify "dirty shadows" as a sign of subcutaneous gas.[2] MRIs are cost- and time-intensive and are not recommended as an initial imaging modality. Critically ill patients may need immediate surgical intervention if there is high clinical suspicion, and their disposition to the OR should not be delayed to obtain imaging. Ultimately, definitive diagnosis only occurs during surgical exploration.[2]

MANAGEMENT

Fournier's gangrene is a surgical emergency. Time to surgical intervention is the most significant modifiable risk factor for mortality in these necrotizing infections, and early intervention can halve the mortality rate.[2]

Surgery involves radical exploration and aggressive, wide debridement of necrotic and gangrenous tissue, often done in stages.[1] Depending on the anatomy involved, the surgical team may comprise any combination of urology, general surgery, colorectal surgery, OB/GYN, and plastic surgery.

Broad-spectrum antibiotic therapy is a cornerstone of medical management. Empiric therapy should cover gram-positive, gram-negative, and anaerobic organisms. Acceptable regimens include a carbapenem (e.g., ertapenem) or beta-lactamase inhibitor (e.g., piperacillin-tazobactam), plus an agent with MRSA activity (e.g., vancomycin or linezolid), plus clindamycin for its antitoxin and mortality benefits.[3] Doxycycline should be added for those with wounds exposed to freshwater (Aeromonas) or sea water (V. vulnificus).[3] Antifungal coverage can be considered for those with significant risk factors.

Because electrolyte derangements often co-occur, fluid resuscitation is warranted, especially in cases of sepsis or septic shock. Preferred treatment is with balanced crystalloids such as PlasmaLyte or Lactated Ringer's.[1] Patients with hypotension and organ hypoperfusion refractory to appropriate fluid resuscitation should be started on vasopressors, particularly norepinephrine.[2] With surgery being the only definitive treatment for Fournier's, medical interventions should not delay prompt surgical consultation and intervention.

After debridement, patients may require reconstructive surgeries and/or vacuum-assisted wound closure devices, and those with significant perineal or anorectal involvement may need fecal diversion such as a temporary colostomy to promote wound healing.[1] If available, hyperbaric oxygen therapy can be used as a post-operative adjunct, as it has been shown in some studies to facilitate wound healing and decrease mortality.[5]

CASE CONCLUSION

The patient was taken emergently to the OR with general surgery, colorectal surgery, and urology for wide debridement. Post-operatively, he was admitted to the surgical ICU with septic shock and multiorgan system failure and required multiple OR takebacks, including loop colostomy creation. The patient was continued on broad-spectrum antibiotics. He had a prolonged hospital course and was discharged after a 2-month stay. ✶

References available online.

Xylazine, Illicit Drug Combo: A Deadly Cocktail on the Rise

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The majority of overdose-related fatalities in the United States are currently attributed to synthetic opioids, including fentanyl. The introduction of adulterants into the drug supply is an alarming trend noted by poison control centers across the country. The combination of opioids and adulterants, such as xylazine, is thought to create a potentially more deadly exposure.

Xylazine is an alpha-2 agonist and is used as a veterinary tranquilizer for its analgesic and muscle relaxant properties. When mixed with fentanyl, it is known on the street as "tranq" or "tranq dope." Xylazine also has been known to be mixed with cocaine, heroin, and other drugs to create deadly cocktails. It can be injected, snorted, swallowed, or inhaled.

Xylazine was first noted to be an adulterant in Puerto Rico in the early 2000s and eventually was used as a drug on its own. Xylazine is often used by drug manufacturers to stretch drug supplies, as it is relatively inexpensive and is known to create similar psychoactive properties. Many users do not know they are consuming xylazine-laced opiates, while some users specifically seek it out.

Due to the relatively recent introduction of xylazine to the U.S. drug supply, there have not been many studies on its effects. Xylazine is known to cause hypertension and tachycardia followed by central nervous system depression, bradycardia, hypotension, and possible death.

Effects can be prolonged, which is why some users seek it out.

Some consumers of xylazine report withdrawal symptoms similar

References available online.

to opiate withdrawal. A multicenter study by Love et al found that opioid overdoses with xylazine exposures were actually less severe than isolated opioid exposures. Both groups in the study had large naloxone requirements, and it is hypothesized that this is due to the CNS depression associated with xylazine that masquerades as opioid overdose.[1] Currently, there isn't an antidote for xylazine overdose.

As of 2021, the northeast region of the United States has seen the greatest increase in xylazine identifications. In 2022, approximately 23% of fentanyl powder and 7% of fentanyl pills seized by the DEA contained xylazine, and xylazine-fentanyl mixtures had been found in 48 out of 50 states.[2] Despite xylazine exposures increasing, xylazine is not currently classified as a controlled substance in the U.S. Controlled Substance Act.

Emergency physicians must keep multiple things in mind when treating patients presenting with suspected

EMRA TOXICOLOGY

xylazine exposure. First and foremost, it may be difficult to diagnose xylazine exposure from the ED, as routine urine drug screens do not detect xylazine. Xylazine test strips have been developed recently and are available for patients to test on their own drug supplies. Xylazine can also be detected by chromatography-mass spectrometry testing.

If an exposure to xylazine is suspected, naloxone should be administered to reverse any opiate co-ingestion. Physicians suspecting xylazine exposure are encouraged to report it to the FDA's MedWatch Adverse Event Reporting Program.

Aside from immediate effects related to xylazine use, patients with a history of xylazine use by injecting the drug are known to develop necrotic skin ulcers, which may appear at areas distant to the site of injection. They are typically large and more rapidly progressive than other types of ulcers. Although the mechanism is not fully understood, it is thought that alpha-2 related effects on peripheral tissues cause vasoconstriction that ultimately leads to skin breakdown. These wounds need to be treated with incision and drainage, antibiotics, and surgical consultation if debridement is necessary.[3] ✶

Editor's note: This paper was authored by leaders of EMRA's Toxicology Committee.

References available online.

Blue Dye, Gotta Try: A Case of Metformin-Associated Lactic Acidosis

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INTRODUCTION

Metformin-associated lactic acidosis (MALA) is an unusual complication in the setting of metformin use or overdose. This condition is often a diagnosis of exclusion due to its rarity compared with more common causes of anion gap metabolic acidosis such as septic shock, diabetic or alcoholic ketoacidosis, toxic ingestion, and renal failure.

MALA has a 30-50% risk of mortality, especially in patients presenting with pH <7.1 or lactic acid >25 mmol/L.[1-3] Aggressive supportive care is necessary to improve survivability.

We present a suspected case of MALA requiring intensive, collaborative medical care among emergency medicine, critical care, nephrology, toxicology, and internal medicine allowing for a remarkable patient recovery.

CASE PRESENTATION

A 58-year-old man with a history of hypertension, type 2 diabetes mellitus, remote alcohol use disorder, and suicide attempt (nearly 30 years ago) arrived at

References available online.

the ED via EMS for altered mental status and frequent falls within the previous 24 hours. The patient's roommate called medics and provided history at the scene. For EMS, the patient initially moved all extremities and was mildly combative and then became somnolent during transport. ED personnel did not record initial EMS vital signs.

Upon arrival at the ED, the patient had a Glasgow Coma Scale (GCS) of 3, indicating a high risk of impending brain injury or death. His initial vital signs included heart rate of 112 beats per minute, blood pressure of 94/50 mmHg, respiratory rate of 28 breaths per minute, rectal temperature of 90.4 degrees Fahrenheit, and pulse oximetry of 86% on 15 liters/minute of oxygen using a non-rebreather mask.

The patient's ECG revealed atrial fibrillation with a ventricular rate of 108, incomplete left bundle branch block, and nonspecific ST segment abnormalities concerning for possible lateral subendocardial injury. These ECG findings heightened consideration of acute coronary syndrome, cardiomyopathy, or massive pulmonary embolism.

Oral intubation was performed for both airway protection and emergent management of acute hypoxic respiratory failure. An initial 1 liter IV fluid bolus of sodium chloride 0.9% solution was administered to enhance the patient's mean arterial pressure. Additionally, an external convection warming unit (3M™ Bair Hugger™) was applied to manage the patient's hypothermia.

Given the abnormal vital signs, sepsis of unknown origin was likely for this patient. Consequently, broad-spectrum antibiotics consisting of vancomycin and a blend of piperacillin and tazobactam were administered. Additionally, the patient received 30 milliliters/kilogram of IV isotonic crystalloid fluids in compliance with current sepsis guidelines. Despite fluid resuscitation, the patient remained hypotensive, prompting the initiation of 2 IV vasopressors, norepinephrine bitartrate and vasopressin.

Upon electronic chart review, the patient's home medications included metformin, insulin, oxycodone, and aspirin. Earlier in the day, the patient had contacted his primary care provider's office complaining of fatigue, generalized malaise, and shortness of breath, and he was referred to the ED.

Noteworthy initial lab results included a venous blood gas (VBG) revealing pH of 6.5, bicarbonate of 3.3 mEq/L, and partial pressure of carbon dioxide (pCO2) 42.1 mm/Hg, consistent with a combined metabolic and respiratory acidosis with a superimposed metabolic alkalosis. His anion gap was greater than 49 mEq/L.

Given the patient's profound anion

Table 1. Principal laboratory findings

Test & Reference range	Result	Test & Reference range	Result
Chemistry		*Hematology*	
Sodium (135-145)	139 mEq/L	Hemoglobin (13.5-17.5)	10.7 g/dL
Potassium (3.5-5.1)	5.0 mEq/L	WBC (4.5-11)	8.63 K/uL
Chloride (98-108)	90 mEq/L	*Blood gas*	
Bicarbonate (21-32)	<5 mEq/L	pH (7.32-7.42)	6.5
BUN (10-20)	67 mEq/L	pCO2 (41-51)	42.1 mmHg
Creatinine (0.5-1.3)	11.74 g/dL	pO2 (25-40)	126 mmHg
eGFR	< 5	HCO3 (24-28)	3.3 mEq/L
Anion gap (10-20)	> 49 mEq/L	Glucose (65-99)	74 mg/dL
Phosphorus (2.7-4.5)	16.9 mEq/L		
Magnesium (1.6-2.4)	2.5 mEq/L	*Miscellaneous*	
Liver Function		Lactate (0.6-2.0)	19 mmol/L
Alkaline Phosphatase (40-150)	116 U/L	Alcohol (< 10)	< 10 mg/dL
AST (0-45)	32 U/L	Acetaminophen (< 0.5)	< 0.5 mcg/mL
ALT (0-40)	14 U/L	Salicylate (0.0-0.2)	0.4 mg/dL
		Troponin (</= 22)	140 ng/L, 127 mg/L
		Beta-hydroxybutyrate (0.0-0.3)	9.1 mmol/L

ALT, alanine transaminase; AST, aspartate aminotransferase; BUN indicates blood urea nitrogen; eGFR, estimated glomerular filtration rate; pCO2, partial pressure of carbon dioxide; pO2, partial pressure of oxygen; WBC, white blood cells.

References available online.

gap metabolic acidosis and lactic acidosis, further investigation for possible toxic ingestants, such as alcohol, ethylene glycol, methanol, salicylates, and acetaminophen, were pursued.

Additional lab testing revealed blood urea nitrogen (BUN) of 67 mEq/L, creatinine of 11.74 g/dL, and glomerular filtration rate (GFR) of 5, indicative of acute renal failure. (Approximately 5 months prior to presentation, the patient's baseline creatinine was 1.2 g/dL.) His beta-hydroxybutyrate was significantly elevated at 9.1 mmol/L, presuming a marked ketotic state, but blood glucose was within normal range, making diabetic ketoacidosis or hyperosmotic hyperglycemic syndrome a less likely etiology for his metabolic derangement. Remaining ED lab studies included unremarkable liver function testing and CBC. Salicylate, acetaminophen, and ethanol blood levels were negative for elevation.

Advanced radiological imaging was performed due to the patient's altered mental status and recent history of falls. This imaging included CTA head and neck, CT pulmonary arteries, CT abdomen and pelvis with IV contrast, and CT cervical, thoracic, and lumbar spine without contrast. All imaging studies were negative for acute pathology or evidence of recent traumatic injuries.

The emergency physician initiated a sodium bicarbonate IV infusion to address the patient's profound acidotic state. Nephrology agreed with the continuation of this treatment and recommended initiating emergent dialysis with continuous renal replacement therapy (CRRT) after the patient's arrival to the ICU. This constant, 24-hour-per-day dialysis treatment allows for steady correction of ongoing acidosis and electrolyte derangements in hemodynamically unstable individuals with acute renal failure. CRRT can also improve uremia and assist with clearance of dialyzable toxins.

On the patient's arrival to ICU, the intensivist contacted the poison control center due to concerns for possible toxic ingestion. The center's toxicologist advised starting both IV fomepizole and high-dose vitamin therapy due to the potential of ethylene glycol or methanol toxicity as a contributor to the patient's anion gap metabolic acidosis and renal failure. Both therapies were initiated, and the toxicologist remained in communication with the critical care team in the days following.

During the initial ICU course, the patient's condition continued to decline, requiring the addition of epinephrine for vasopressor support and stress dose steroids. Despite these additional supportive therapies, the patient's acidosis continued to deteriorate. His pH became undetectable on blood gas machine analysis, indicating a level lower than the machine's cut-off value of 6.5. IV sodium bicarbonate ampules were administered in addition to the sodium bicarbonate infusion to correct the ongoing metabolic derangements.

Despite significant resuscitative measures, including 3 vasopressors, sodium bicarbonate infusion and boluses, and CRRT, the patient's metabolic acidosis and hemodynamic instability continued to worsen. His prognosis remained poor, and he was now considered in refractory shock. Due to this, methylene blue was administered as an IV bolus followed by tapper. The hope was that it would act as a vasopressor-sparing agent in vasoplegic, distributive shock. It acts to inhibit nitric oxide synthesis, thereby alleviating vasoplegia and promoting an increase in blood pressure.[4,5]

On hospital day 2, marked improvement occurred in the patient's clinical status after initiating methylene blue. Due to progressive resolution in the patient's metabolic state, the intensivist discontinued the sodium bicarbonate infusion. Additionally, as the patient demonstrated consistent improvement in his mean arterial pressure, 2 of the vasopressors at maximum dose were steadily weaned and then discontinued, leaving only norepinephrine bitartrate.

The patient continued to make significant clinical improvements. On hospital day 3, now off vasopressors, his CRRT was transitioned to intermittent hemodialysis (HD). Following negative blood culture results, antibiotics were discontinued on the 5th day of hospitalization, and the patient underwent a successful extubation. After a steady improvement in alertness, the intensivist questioned the patient about the circumstances leading to his medical emergency, but unfortunately, he did not recall any imperative details. He denied any intentional ingestions or suicidal ideation leading up to his hospitalization. His expanded drug screen and volatile panel only demonstrated an elevated acetone level of 11, likely due to his initial severe ketotic state.

Renal recovery and urine production returned for the patient during his hospitalization, so intermittent HD ceased after 3 sessions on hospital day 9. The following day, he was transferred to a regular medical floor with a relatively uneventful recovery, except for episodes of delirium. After 3 weeks of hospitalization, the patient was discharged home with weekly outpatient physical therapy sessions.

Given the lack of any other identified etiology for the patient's profound metabolic acidosis, the multidisciplinary care team surmised that the patient experienced MALA in the setting of metformin use. Most likely, an unspecified illness led to dehydration and AKI, which contributed to the development of this rare disorder in the days preceding his hospitalization.

DISCUSSION

Metformin is a biguanide anti-hyperglycemic agent commonly prescribed for patients with type 2 diabetes mellitus and polycystic ovary syndrome. This oral medication is not metabolized by the liver and is excreted unchanged in the urine.[1] Due to its unique pharmacokinetics, metformin can accumulate in the body during times of acute kidney injury or in patients with a history of chronic kidney disease.[1-3] As a biguanide, metformin has a well-established risk of lactic acidosis, though much lower than phenformin, an antihyperglycemic medication of the same class.[1]

References available online.

Metformin's mechanism of action causes increased lactate production by blunting hepatic gluconeogenesis.[3] This disruption in metabolism leads to increased pyruvate production, resulting in elevated lactic acid, and thus increased serum lactate levels.[1-3]

Metformin-associated lactic acidosis is defined as an elevated lactic acid and metabolic acidosis in the setting of metformin use with numeric values of pH <7.35 and lactate >5 mmol/L.[1]

Due to this drug's action on the liver, MALA starts as a type B lactic acidosis as it alters pyruvate metabolism in the Cori cycle at the mitochondrial level. This type of acidosis occurs from impaired cellular functioning, while type A lactic acidosis is defined by tissue hypoxia and impaired tissue perfusion.[5]

Even though MALA starts as type B lactic acidosis, it can progress to include type A because of compounding, multifactorial organ dysfunction leading to hemodynamic instability, tissue hypoperfusion, and hypoxia.[6,7] MALA can provoke vasoplegic shock, with a profoundly low systemic vascular resistance. Vasoplegic shock is a type of distributive shock with smooth muscle derangement causing abnormal vasoconstriction leading to systemic hypoperfusion.

A test obtaining serum level of metformin is not readily available and offers little utility in making the diagnosis or predicting the severity of MALA.[3] As a result, such testing is infrequently ordered.[8] Mortality is more readily predicted in MALA by a pH <6.9 and serum lactate >25 mmol/L.[3,9]

One retrospective study in the ED suggested that MALA should be suspected in patients with septic shock who take metformin and have a serum lactate >8.4 mmol/L and creatinine >2.9 mg/dl.[3,6] When these criteria were met, the specificity of MALA was 99 percent.[3,9]

MANAGEMENT

Management of this disorder is primarily supportive with the goal of correcting underlying lab derangements created by vasoplegic shock. Sodium bicarbonate infusions are suggested in pH <7.15.[10] Hemodialysis is recommended if the patient demonstrates severe metabolic acidosis pH <7.1 and lactic acid concentration >20 mmol/L. HD can also be indicated if the patient's acid-base disorder fails to improve in 2-4 hours of bicarbonate therapy or if there is presence of shock, kidney injury, or liver injury.[3]

Methylene blue has been used for refractory shock, but its utility and wide acceptance is limited due to a lack of controlled studies. Although its mechanism of action is unclear, it is thought to inhibit nitric oxide production, thereby helping to restore vascular tone.[11-13] Additionally, it has a catecolamine-sparing effect through its action against guanylyl cyclase for patients with distributive shock.[4,5]

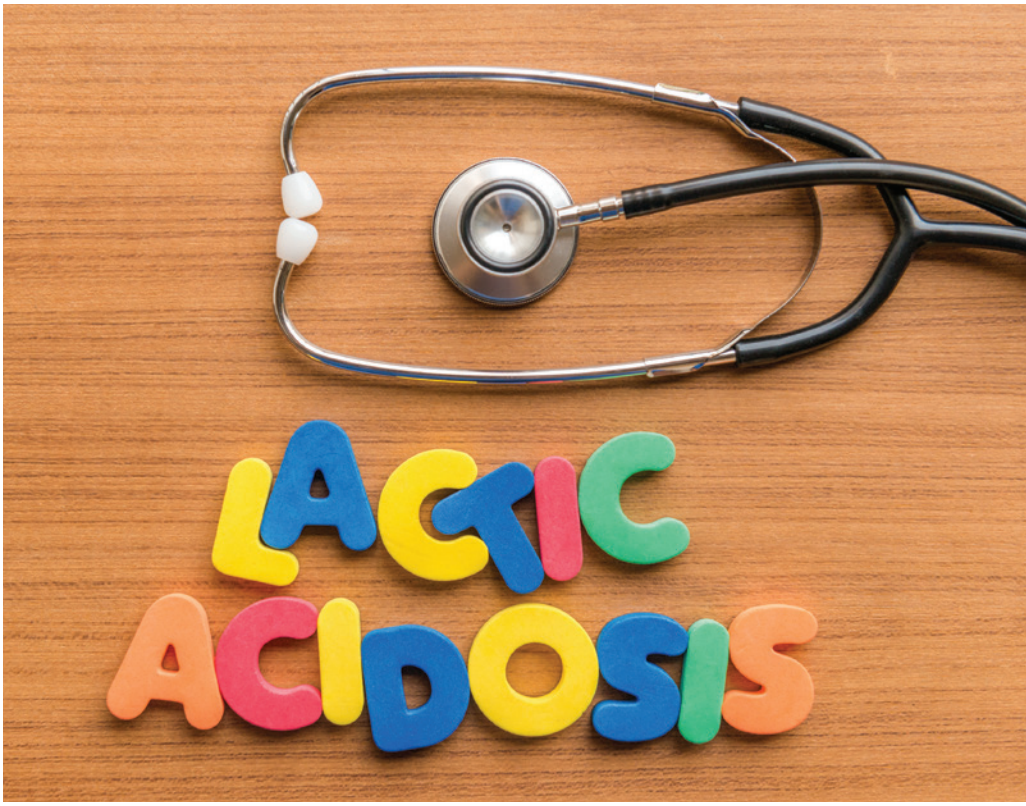
While methylene blue has been well-established in vasoplegic shock in cardiothoracic surgery patients, its utility as an adjunct to vasopressors is not as frequently employed.[13] There are a few other case reports of methylene blue use in MALA patients who showed significant improvement in vasoplegic shock when it was used as an adjunct to routine vasopressors.[11,12]

CONCLUSION

MALA is a difficult diagnosis to make due to lack of specific lab testing revealing toxicity. However, this disorder should be considered in patients presenting with marked hemodynamic instability with strikingly elevated lactic acid levels, whose home medication list includes metformin.

The incidence of MALA may increase, given the rising prevalence of type 2 diabetes mellitus and the preference for metformin as the disease's initial treatment of choice.

Notwithstanding, effective interdisciplinary care, with a focus on supportive care measures, can result in the best possible outcome for MALA patients. Adjunctive therapies are likely to be necessary in MALA, and the use of methylene blue has shown anecdotal benefit in some case reports. Further investigation is necessary to enhance our understanding of the utility of using methylene blue in refractory hemodynamic instability in non-traumatic medical emergencies. ✶



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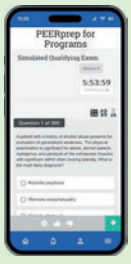

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EMRA

Grant Recipient Conducts Research on PALS-Based PEM Resources

By Imikomobong (Micky) Ibia, MD

Imikomobong (Micky) Ibia, MD

The EMRA Simulation Research Grant has made it possible for me to implement a multiyear pediatric emergency medicine (PEM) study that evaluates the effectiveness, usability, and workload implications of cognitive aids in pediatric advanced life support (PALS) adherence.

Pediatric cardiopulmonary emergencies represent stressful, high-acuity, low-frequency events with high morbidity and mortality. Furthermore, the vast majority of pediatric patients are seen in general EDs by emergency physicians who are not PEM-trained, and some physicians may be uncomfortable treating critically ill pediatric patients.

Currently, cognitive aids to maximize pediatric advanced life support (PALS) adherence exist most commonly as pocket cards, including the widely used American Heart Association (AHA) PALS reference card. Unfortunately, these cards aren't always immediately available at point-of-care during a resuscitation event. Also, they can become outdated and are prone to wear-and-tear.

Only a few smartphone apps are available for PALS, and none have undergone rigorous validation.

In this study, partially funded by the EMRA Simulation Research Grant, we aimed to evaluate the impact of Massachusetts General Hospital's (MGH) PALS app on adherence to PALS resuscitation algorithms using the Clinical Performance Tool (CPT), a validated PALS-based performance score, during simulated scenarios.

As secondary objectives, we aimed to assess whether MGH's app improves correctness and expedites time-to-completion of therapeutic interventions during simulated PALS scenarios, compared to AHA's card.

Additionally, we aimed to assess the app's overall perceived usability and impact on participants' perceived workload during simulated PALS scenarios.

We conducted a prospective, randomized, simulation-based trial of 37 senior Boston-based EM residents. Participants were randomized to either the intervention (MGH PALS app) or control (AHA PALS reference card) group.

On their scheduled day and time, participants came to Brigham and Women's STRATUS Center for Medical Simulation. They were guided through a 15-minute pre-briefing period, during which they completed a survey detailing their baseline characteristics and experiences. They were also given time to familiarize themselves with their respective cognitive aid.

Next, they participated in 3 PALS simulations, each with a duration of 10 minutes. The simulations were:

- A respiratory arrest
- A pulseless nonshockable cardiac arrest
- A pulseless shockable cardiac arrest

Scenario prompts were identical to those described in the derivation and validation studies for the CPT. The scenario order was standardized, allowing for direct comparison across each scenario without influence of order.

Upon finishing, each scenario was debriefed with specific emphasis on teaching about the differences in care of children with respiratory and cardiac arrest.

Participants then spent 10 minutes completing the System Usability Scale (SUS) and NASA Task Load Index (NASA-TLX) surveys.

While final results of our study are pending, our initial analysis suggests no difference in CPT scores, correctness, or time to completion of therapeutic interventions between users of the MGH smartphone app and the AHA reference card.

However, a key finding: Participants reported they were much more likely to carry smartphones on shift vs. cards. According to our survey results, 97% always carried smartphones, and only 5% always carried reference cards. Plus, participants reported significantly easier usability and improved workload with the app vs. the card.

I'm grateful to be able to conduct this important simulation research, made possible by the EMRA Simulation Research Grant. ✶

Editor's notes: Congratulations to Dr. Ibia, recipient of the 2021 EMRA Simulation Research Grant. Dr. Ibia completed residency training at Harvard Affiliated EM Residency at Mass General Brigham, where he served as chief resident. He then pursued a PEM fellowship at Boston Children's Hospital.

The MGH PALS app is available for download via the App Store and Google Play. Also access fast, updated, evidence-based PEM guidance on MobilEM, EMRA's mobile app available for free download. Additionally, EMRA publishes several pediatric-focused resources and guides, including PEM Fundamentals, Basics of Emergency Medicine-Pediatrics, and the Pediatric Qwic Card, all available on Amazon. And finally, search categorically on emresident.org for articles and papers specific to pediatric emergency medicine.

References available online.

A Deep Dive With Judith Tintinalli, MD, MS, FACEP

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empower
Sharing Our Stories

Welcome to EMpower! This section of EM Resident focuses on leadership in emergency medicine — it's where we share the incredible stories of leaders in the EM community. Our EMpower honoree in this edition is Judith Tintinalli, MD, MS, FACEP.

For this entry in our EMPower series, we spoke with the esteemed ***Judith Tintinalli, MD, MS, FACEP****. Not only is Dr. Tintinalli wildly accomplished in the field of emergency medicine, but she is also charismatic, and it's a pleasure to learn from her. A huge fan of EMRA, Dr. Tintinalli shares her pearls as we get to know her a little bit better in this edition of EMPower.*

Dr. Tintinalli is a professor and chair emeritus of the department of EM at UNC Chapel Hill. She graduated from the Wayne State University School of Medicine. She completed residency at the University of Michigan and earned a master's in clinical research design and statistical analysis from that institution as well. She is a prior president of the ABEM, founding president of CORD, past president of AACEM, and former chair of the Liaison Residency Committee (forerunner of the Accreditation Council for Graduate Medical Education).

She is a recipient of the Lifetime Achievement Award from the University of North Carolina Academy of Educators, ACEP's James Mills Award, a National Education Award, and the Order of the International Federation of Emergency Medicine Fellowship. She is an honorary fellow of ACOEP, and ACEP renamed a prestigious award after her: The Judith E. Tintinalli Award for Outstanding Contribution in Education is presented to an ACEP member who has made a significant contribution to the educational aspects of the specialty.

Dr. Tintinalli is double-boarded in EM and IM and is editor-in-chief of the world's largest-selling emergency medicine textbook, Tintinalli's Emergency Medicine. She is a prior board member and deputy editor of the Annals of Emergency Medicine. She's editor-in-chief of AccessEmergency Medicine, the Mcgraw Hill digital library for emergency medicine, and Emergency Physicians Monthly. She is co-editor of EMS: A Practical Guidebook.

First things first, why emergency medicine?

I love everything about it. I would rather die than have a 9-5 job to begin with. I love the diversity, the challenge, the spirit, the teamwork. I've liked it since I was a medical student and had my first rotation in trauma in the ED. It hit me — that's where I want to be.

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

In my intern year, I took a lot of different rotations — cardiology, plastics, etc. — that I thought I would need in the ED. If I started today, I would do an EM residency. I wanted to get the most solid and best kind of degree I could.

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

A lot of changes are happening in emergency medicine. For me, looking back at the beginning, EM was difficult and there were challenges everywhere. Embrace the change and the challenges. You'll look back at them and be happy you did.

What keeps you coming to work every day?

Teaching!

What is your best time management tip?

(1) Working in the ED: Get really good at pattern recognition — asking the right questions, honing in on the decision-making for those issues. Try to be focused, and try to focus on one thing at

References available online.

empower
Sharing Our Stories

Judith Tintinalli, MD, MS, FACEP

one time. (2) For life: Make lists. By the item, I put a little box, and then I "x" the box and cross out the item. I love to see things done. That's what we love about the ED. When your shift is done, you have completed something.

Share a few things that are on your desk right now.
Paperwork for a second family home in Durango, CO, that we just signed on. And EMRA PEM essentials! I don't know how you guys do these books so well, but they are fantastic. Also, Tintinalli in your pocket app information.

What is the best on-shift snack?
It was always pizza!

What is the most recent book you read?
The Name of the Rose

What message would you give to EMRA members?
Get involved and stay involved. We know there are problems in emergency medicine. Let those be an accelerant for you. We are POWERFUL, and we have to learn to capture our power so we can apply it better. Problems accelerate our growth. ✶

References available online.

NEWS & NOTES in Emergency Medicine

Melissa A. Barton, MD

ABEM APPOINTS EXECUTIVE DIRECTOR, PROFESSIONAL AND CLINICAL AFFAIRS

Melissa A. Barton, MD, has been appointed executive director of professional and clinical affairs for the American Board of Emergency Medicine, effective Jan. 1. She will replace Earl J. Reisdorff, MD, who retires in December.

Dr. Barton joined ABEM in 2015 as its inaugural director of medical affairs, focusing on certification activities related to residency training and subspecialty development. While at ABEM, she helped lead multi-organizational outreach activities, including the award-winning Dr. Leon L. Haley Jr. Bridge to the Future of Emergency Medicine and the Coalition of Board-Certified Emergency Physicians. She also led the review process for the EM Model and served as the ABEM liaison for the ACGME Residency Committee-Emergency Medicine.

Dr. Barton is board-certified in EM and has practiced for more than 20 years in Michigan, with her early career at Detroit Medical Center (DMC) Sinai-Grace Hospital, a trauma hospital in northwest Detroit. Immediately following residency, Dr. Barton became associate program director and later program director for the DMC Sinai-Grace Hospital Emergency Medicine Residency Program. She also had a faculty appointment as a clinical associate professor at the Wayne State University School of Medicine.

A member of the Michigan College of Emergency Physicians (MCEP) since 2004, Dr. Barton served as MCEP president in 2010 and has remained engaged with the chapter.

She holds a bachelor's degree in finance from the University of Colorado at Boulder and earned her medical degree from Creighton University School of Medicine. She is currently completing a master of public health from the Milken Institute of Public Health at George Washington University. She completed an EM residency at Sinai-Grace Hospital/ Wayne State University and served as a chief resident during her final year of training.

"With a great foundation brought forward by Dr. Reisdorff, there is no doubt Dr. Barton's effective and diverse experiences in medical education and health care will continue to enhance and lead the development of certification opportunities and partnerships for ABEM, said ABEM Board President Diane L. Gorgas, MD. "We are excited to begin this next chapter with Dr. Barton in her new role."

Kathleen C. Ruff, MBA

ABEM APPOINTS EXECUTIVE DIRECTOR, ADMINISTRATIVE AFFAIRS

ABEM has appointed **Kathleen C. Ruff, MBA**, as executive director, administrative affairs. Ms. Ruff, currently chief administrative officer of ABEM, will assume her new role on Jan. 1.

In her current role, Ms. Ruff leads teams in accomplishing strategic initiatives, programs, and operations. She works with directors and staff on major initiatives including initial certification redesign, Certifying Exam development, and DEI planning, training, and integration. She also oversees strategic planning and projects, budget development, and NCCA accreditation compliance and activities.

Before joining ABEM, Ms. Ruff held executive-level positions at American Board of Medical Specialties, Northeast Ohio Medical University (NEOMED), and NEOMED Foundation. She also served in several roles at Drexel University. She holds a bachelor's degree in business administration and an MBA from Drexel University.

"Ms. Ruff is a strategic and experienced leader and has made a tremendous impact on board certification. She brings a breadth of professionalism and innovation as well as a commitment to excellence that has made a profound impact on ABEM's success," said ABEM Board President Diane L. Gorgas, MD. "We look forward to her expanding her strategic role in leading the future of the organization."

ABEM APPLIES TO ABMS FOR FOCUSED PRACTICE DESIGNATION IN EMERGENCY BEHAVIORAL HEALTH

ABEM has applied to the American Board of Medical Specialties (ABMS) to recognize expertise in Emergency Behavioral Health (EBH) through a Focused Practice Designation (FPD).

ABEM and the American Board of Psychiatry and Neurology have collaborated on this initiative and together will offer this FPD to interested emergency physicians and psychiatrists.

References available online.

The purpose of this FPD is to recognize the expertise held by physicians in the provision of emergency behavioral care. An FPD would facilitate improved access to physicians with specialized expertise to help address the needs of patients seeking acute, unscheduled mental health treatment.

The mental health crisis in the United States and its lack of access to EBH care has created the need for a unique skill set that combines areas within the field of emergency medicine and psychiatry.

Appropriate and timely emergency behavioral health care can be provided to patients who are boarding in the emergency department for days, weeks, or, on occasion, months, while awaiting an open inpatient psychiatric bed.

An Emergency Behavioral Health Focused Practice Designation takes one step forward to address this public health emergency.

A period of public comment is open through ABMS to share your support. The ABMS Committee on Certification (COCERT) will consider this request during its Nov. 4 meeting, and the ABMS Board of Directors may vote on this proposal during its meeting in February.

For more information, visit abms.org/cocert-invites-comments/.

Stanford Schor, MD, PhD

ANNALS ANNOUNCES RESIDENT FELLOW

Annals of Emergency Medicine has selected **Stanford Schor, MD, PhD**, to serve as resident fellow on its Editorial Board for the coming year. Dr. Schor, currently at University of Cincinnati, received his MD and PhD from Stanford University. Each year, *Annals of Emergency Medicine* selects a resident fellow to serve on the Editorial Board. Katie Lebold, MD, PhD, of Stanford University, is the immediate past resident fellow for the journal. Dr. Lebold began her term in October 2023, and her term ends in October 2024.

If you have an idea, issue, or experience you would like to write about, submit an abstract (limit 250 words, double-spaced) through *Annals'* online submission system, Editorial Manager, at www.editorialmanager.com/annemergmed (use the "Residents' Perspective" article type). If your abstract is approved, you will be asked to write the full-length article for the "Residents' Perspective" section.

If you have any questions for Dr. Schor, contact him at annalsfellow@acep.org. ✶

References available online.

Board Review Questions

PEERprep for Physicians is ACEP's gold standard in self-assessment and educational review.

For complete answers and explanations, visit the Board Review Questions page at emresident.org, under "Test Your Knowledge"

Order PEERprep at acep.org/peerprep

1. A 45-year-old patient with alcoholic cirrhosis presents with confusion and increasing abdominal pain and pressure over the past week. A paracentesis is performed, revealing 520 WBCs with 89% polymorphonuclear leukocytes and 11% lymphocytes. The protein level is high, but the glucose level is normal. What is the best course of treatment?

A. Administer a third-generation cephalosporin
B. Administer ampicillin and an aminoglycoside
C. Start probiotic therapy and a fluoroquinolone
D. Withhold antibiotics pending Gram stain and culture

2. Which medication is contraindicated in the treatment of thyroid storm?

A. Cholestyramine
B. Esmolol
C. Iodine
D. Salicylic acid

3. A 22-year-old woman presents with a red and painful arm several days after she scraped it on a fence. She says the redness is "getting bigger by the minute" and rates the pain as severe. Her vital signs include BP 90/60, P 110, and T 38.4°C (101.1°F). Her forearm is warm and erythematous, and there is crepitus. What is the best approach to diagnosing the most likely condition in this case?

A. CT
B. MRI
C. Surgical exploration
D. Wound cultures

4. A 49-year-old man is brought in by his family who say that they cannot take care of him anymore because of his impulsive wandering and disinhibited behavior. The family was told 6 months ago that the patient has dementia, and he has declined progressively since then. What type of dementia does the patient most likely have?

A. Alzheimer's disease
B. Frontotemporal neurocognitive disorder
C. Neurocognitive disorder with Lewy bodies
D. Prion disease

5. Which technique should be attempted first to remove a nasal foreign body without the use of restraints in a young pediatric patient?

A. Back blows
B. Bag-valve-mask positive pressure
C. Forceps
D. Parent's kiss

PEERprep For Physicians

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

References available online.

EMRA ECG Challenge

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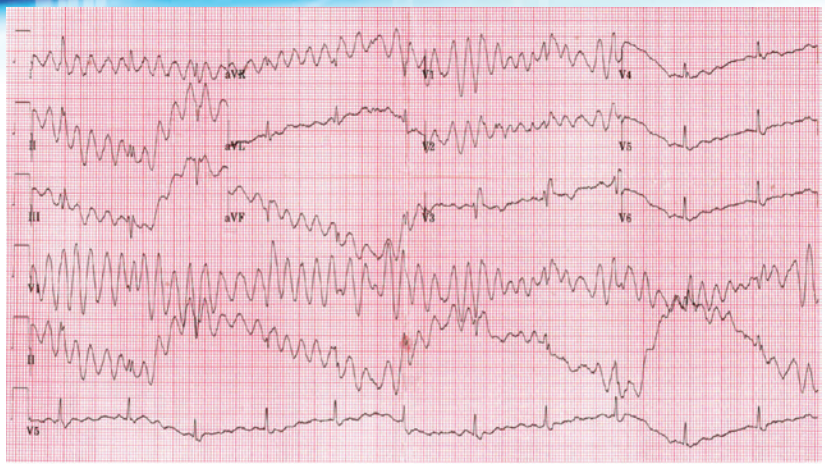
CASE

An 83-year-old male with a past medical history of hypertension, hyperlipidemia, and Parkinson's dementia presents to the emergency department for acute onset shortness of breath. Upon arrival, he was tachypneic with an oxygen saturation of 70% on room air.



What is your interpretation of his ECG?
See the ANSWER on page 68.

References available online.

ECG Challenge

Pseudo-ventricular tachycardia occurs when electrocardiographic artifact mimics polymorphic VT. In this case, the artifact is due to the patient's Parkinsonian tremors. These are a common cause of ECG artifact and prone to misinterpretation and subsequent unnecessary interventions.

ANSWER

This ECG shows normal sinus rhythm at 65 bpm, best seen in leads aVL and V3-V6. There is also likely a right bundle-branch block, best seen in lead V3. The remaining limb leads and leads V1-V2 show an irregular wide complex tachycardia with an average ventricular rate of 320 bpm, no discernible P-waves, and phasic variation (ie, happens over a number of beats) of the QRS complex axis and amplitude. These findings are consistent with pseudo-ventricular tachycardia, which describes when artifact mimics polymorphic VT.

DISCUSSION

Pseudo-ventricular tachycardia occurs when electrocardiographic artifact mimics polymorphic VT. In this case, the artifact is due to the patient's Parkinsonian tremors. These are a common cause of ECG artifact and prone to misinterpretation and subsequent unnecessary interventions.[1] ECG artifact can be broadly classified as pseudo-arrhythmic or non-arrhythmic. Pseudo-arrhythmic artifact can mimic a variety of dysrhythmias, including ventricular tachycardia, atrial fibrillation, and atrial flutter.[2] Non-arrhythmic artifact can lead to misinterpretation of Q-waves, ST-segments, and T-waves. The most common causes of pseudo-arrhythmic artifact are body movement and poor skin-electrode contact.[2] Misinterpretation of ECG artifact affects not only EM physicians, but also cardiologists and electrophysiologists.[2] In one paper from the *New England Journal of Medicine*, 12 patients underwent unnecessary diagnostic testing, admission, or interventions based on misdiagnosis of ECG artifact.[2,3]

It is very important to differentiate pseudo-ventricular tachycardia from true pathologic dysrhythmias. When examining a single-lead telemetry strip or cardiac monitor, concern for significant dysrhythmia should prompt checking alternate leads or obtaining a 12-lead ECG.[2,3] It also may be beneficial for the clinician to be at bedside to observe the patient's movements while the ECG is being recorded.

There are 3 signs that can help identify pseudo-VT on an ECG4:

1. The sinus sign: the presence of normal P-QRS-T complexes in either a limb or augmented lead that is due to one of the upper limb electrodes being free of movement artifact. This is best seen in lead V5 on the case ECG.
2. The spike sign: the presence of regular or irregular spikes throughout the pseudo-VT that is due to the superimposition of the underlying normal sinus rhythm. This is best seen in lead I on the case ECG.
3. The notch sign: the presence of a superimposed notch on the pseudo-VT that is due to the superimposition of the underlying normal sinus rhythm. This is best seen in lead II on the case ECG.

CASE CONCLUSION

This patient was found to have pneumonia and was admitted to the internal medicine service for further treatment.

PSEUDO-VENTRICULAR TACHYCARDIA LEARNING POINTS

- Artifact is common, so all ECG leads should be analyzed in their entirety to rule out artifact-related abnormalities.
  - A manually checked pulse should correlate with the rate on the ECG, rhythm strip, or cardiac monitor.
  - Parkinsonian tremors disproportionately affect the limb leads more than the precordial leads.
- Misinterpretation of artifact can lead to unnecessary interventions.
- The sinus sign, spike sign, and notch sign can help identify artifact-related dysrhythmias.

References available online.

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References available online.

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Envision has partnered with the Dr. Lorna Breen Heroes' Foundation to reduce burnout among healthcare professionals.

Envision has also been recognized for its commitment to clinician well-being as a 2024 Wellbeing First Champion by ALL IN: Wellbeing First for Healthcare.

Post-ACEP Virtual Career Fair

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Contact Jessica Esposito,
National Residency Recruitment Manager.
Jessica.Esposito@EnvisionHealth.com

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