

# EM Resident

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## How To Care

Strength and  
Comfort in  
Vulnerability

Cauda Equina  
or Just HSV?

Thyrotoxicosis:  
A Case of  
Storming  
Hormones

Necessity,  
the Mother of  
Invention

Emergent Diving  
Injuries

The Patient's  
Decision-Making  
Capacity



# Searching for the Light

HOPE AMID HELPLESSNESS IN THE ED



# Penn State Health Emergency Medicine

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# Strength and Comfort in Vulnerability

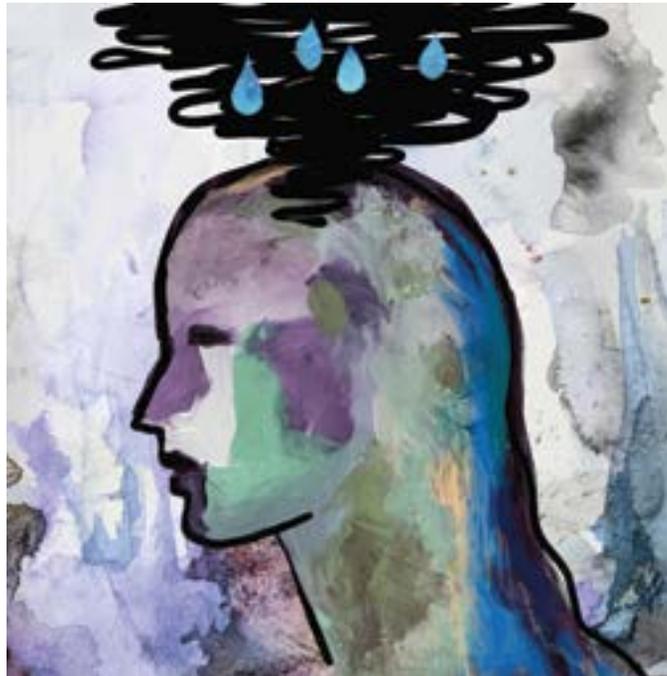
**O**n Jan. 2, Buffalo Bills safety Damar Hamlin, 24, suffered cardiopulmonary arrest on the field against the Cincinnati Bengals. Reports showed that return of spontaneous circulation was achieved after approximately nine minutes of advanced cardiac life support. These nine minutes and their sequelae have traumatized Hamlin, his loved ones, and thousands of fans. They have also shed light upon the emotional toll of a daily occurrence for many of us in emergency medicine.

It's easy to forget that the majority of the population has never witnessed cardiopulmonary arrest. The natural response of shock and fear is one we have grown accustomed to through our line of work. While this ability to compartmentalize is an occupational necessity, it is prudent to acknowledge the emotional trauma throughout of even a single shift. Starting with the recognition that we, too, deserve safe spaces and time for human responses to the devastating situations we encounter will allow us to construct both individual and systematic safeguards against the cumulative effects of residency training and a subsequent career in the emergency department.

I started one of my shifts last week by taking care of a lovely woman in her 60s who was presenting with two days' history of myalgia. She had completed a course of chemotherapy just two months prior and had been feeling well up until this week. On physical exam, there was left-sided weakness consistent with what her

supportive husband had noticed over the past week. My developing clinical acumen gave me foresight for a right-sided frontotemporal mass, but still, I held my breath and hoped it would be influenza.

Later in the same shift, a woman in her 70s presented after she was found to be unresponsive by her family and hypoxic by EMS. We stabilized her and one of her sons arrived at bedside shortly after. His first words to me: "My father just died yesterday morning." Within a



span of 48 hours, this man and his brother fulfilled the responsibility of determining the code status of their critically ill mother — before they were able to finalize funeral plans for their father.

In writing this, I am hit with the realization I never properly reconciled the emotional effects of this shift. There must be better approaches than the one I take now, which, if I am being honest,

is to try to not think of these patients again. Unfortunately, suppressing the emotional toll does nothing to diminish its effects because ... where else can the sadness go?

These experiences are why I appreciate Dr. Sian Lewis-Bevan's vulnerability in sharing how residency has taught her to care for a breadth of clinical presentations, but the emotional cost has led to challenges in caring about both personal and professional components of life ("How to Care," page 54).

Similarly, Dr. Adiba Matin eloquently highlights the bioethical implications and emotional difficulties associated with caring for patients who may not present so unfortunately end-of-life, but instead may have committed criminal acts ("The Ethics of Caring for an Unfavorable Patient," page 44). These patients, too, are part of the emotional trauma throughout.

While there may never be a simple solution, I personally find that acknowledgement of these heavy subjects alleviates some of the burden and perhaps we, as a generation, can hold

soft spaces to process the challenging situations we face each day. I consider it a privilege to read your fascinating cases, written with more nuanced and patient-centered language compared to the current literature. This time, I am even more touched to bear witness to brave and vulnerable acknowledgements of the ubiquitous emotional toll associated with residency training. ★



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# Necessity is the Mother of Invention. Greenness is its Wild Friend.



**Jessica Adkins  
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EMRA President  
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**A**s EM physicians, we see firsthand the needs and challenges of our healthcare system and how its many moving parts intersect and clash, positioning us to become the innovators our communities need. And as residents in particular, we have the added benefit of not knowing what “can’t” be done. When it comes to innovation, our naivety can be fertile ground for solutions, especially if we feel we are safe to ask questions, and if our institutional leadership is willing to explore new strategies.

In your ED, I hope you feel empowered to confront large-scale challenges by questioning the status quo. For example, if your ED has been hit hard by the boarding crisis, learn what community-wide barriers exist to improving flow. Could increased use of mobile-integrated healthcare and community paramedicine alleviate the burden on our EDs for low-acuity complaints? Could programs like Hospital-at-Home alleviate some of the inpatient strain? Where have these programs been studied? Where have they worked well?

As another example, our patients suffer from high rates of opioid addiction, which makes it difficult to control acute traumatic pain. Increased training in nerve blocks would make our analgesia safer and more effective. To consider solutions on an institutional scale, could an EM resident take an acute pain elective month in which they deploy throughout the hospital to perform nerve blocks and other procedures? To think even bigger, could this proceduralist consultant work be a field to responsibly expand EPs’ scope of practice?

A third example is the saturated job market for EPs in certain parts of the country, while rural EDs often rely on unsupervised NPs to provide care. Is it time to push beyond the rural/urban dichotomy? To provide opportunities in

rural health for EPs living in a larger city, we need telemedicine. Strengthening telemedicine requires legislative advocacy, regulatory advocacy in licensing and reimbursement, and medical education focused on high-quality telemedicine.

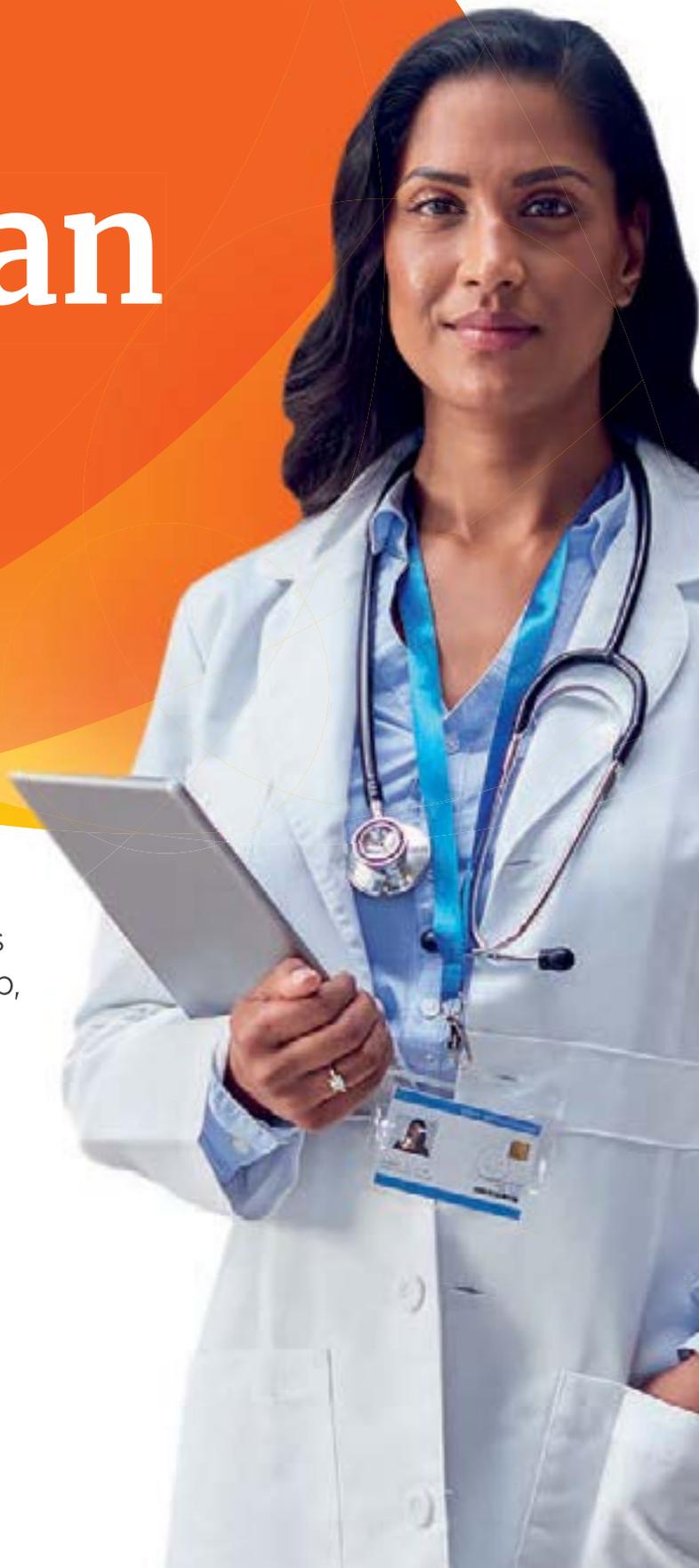
Whatever the issue that makes you most frustrated or excited in EM, you can connect with like-minded residents in EMRA’s 18 committees to discuss it. Learn from each other. Create space for conversations where there are no stupid questions or bad ideas. Connect with mentors willing to lend their

support. EMRA committees are where you can gain a deeper understanding of these issues and where good ideas spread like wildfire.

Though our lack of experience usually feels like a detriment, when it comes to innovative problem-solving, it can be one of our greatest assets. Let’s stay curious, embrace the excitement, and shape the future of emergency medicine. ★



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# What Does it Mean for my Patient to Have Decision-Making Capacity?

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*“The patient wants to leave. Can you please come and have them sign the AMA form?”*

Many of us who work in the hospital, especially the emergency department, have been asked this question — typically when we’re in the middle of doing something else. Often, these patients are challenging to manage, so we print the paperwork in relief and, after a cursory conversation, document the EMR with a quick text “AMA discharge” to cover our bases.

## HOW WELL DO WE IDENTIFY INCAPACITY IN OUR PATIENTS?

Roughly 1 in 4 hospitalized patients lack decision-making capacity<sup>1,4</sup> and clinicians recognize incapacity in less than 50% of affected patients.<sup>2</sup>

## HOW ARE MEDICAL TRAINEES TRAINED TO PERFORM DECISION-MAKING CAPACITY ASSESSMENTS (DMCAS)?

Often, this concept is taught briefly during an introductory ethics course (as capacity is fundamental to the concept of informed consent) or within the psychiatry clerkship of medical school. There are sparse published examples of attempts at educational interventions to help improve this knowledge deficit. Most initiatives develop within the psychiatry department of various institutions, and many demonstrate that direct teaching about capacity improves clinician knowledge base and confidence,<sup>3,5</sup> but there is no standardized way of teaching or testing on this topic.

## WHO SHOULD BE DOING DMCAS?

All too often, we inappropriately rely on our psychiatry colleagues to perform capacity assessments on our patients.<sup>4</sup> All physicians are capable of performing capacity assessments; therefore, rarely should a psychiatry consult be required

except in circumstances of underlying psychiatric disease.<sup>8</sup>

## WHEN DID THE CONCEPT OF DECISION-MAKING CAPACITY EMERGE, AND WHAT WERE ITS LEGAL AND HISTORICAL ORIGINS?

The idea of informed consent for procedures emerged just over a century ago (1914 in *Schloendorff vs. Society of New York Hospital*).<sup>7,18</sup> The plaintiff, Mary Schloendorff, lost the case she brought against the hospital where she had endured physical damages, including the loss of an arm and fingers due to gangrene related to a brachial embolism. She attributed these damages to a surgery — removal of a pelvic tumor — for which she had not consented; she believed she was having an “ether exam” and claimed to have said she did not want surgery.<sup>7</sup>

The reasons she lost the case had almost nothing to do with the concept of informed consent and instead relied on the physician-hospital relationship. Physicians were deemed to be independent contractors, making the hospital “immune from any damages as a result of the alleged negligence or potential battery of the physicians, surgeons, and other hospital personnel.”<sup>7</sup>

Within the opinion of the court, written by Justice Benjamin Cardozo, we find the beginning of a legal basis

for informed consent: “Every human being of adult years and sound mind has the right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient’s consent, commits an assault, for which he is liable in damages ... except in cases of emergency where the patient is unconscious and where it is necessary to operate before consent can be obtained.”<sup>7</sup>

Evolving from the idea of informed consent, decision-making capacity has been developed as a concept with legal origins that were first broadly shared by Dr. Paul Appelbaum, which listed the legally relevant criteria including both the patient’s tasks and questions to guide the physician’s assessment approach.<sup>1</sup>

## CAPACITY IS NOT THE SAME AS COMPETENCY.

Capacity is a clinical evaluation performed by all clinicians (not just psychiatrists). Competence is a court determination made by a judge.<sup>8</sup> These words are not interchangeable, although up until about 2010, they were used interchangeably in the literature; even the influential article by Dr. Appelbaum is titled *Assessment of Patients’ Competence to Consent to Treatment*.<sup>1</sup> The two words represent different concepts, and the difference is important. Some patients who are legally



incompetent may still have capacity to make a particular health care decision.<sup>8</sup> Usually, an ethics consult is warranted in these circumstances.<sup>8-9</sup>

**WHEN SHOULD I PERFORM A DMCA ON MY PATIENT?**

Usually, the assessment is implicit, and we assume capacity in our patients “in the absence of a reason to question a patient’s decision-making.”<sup>11</sup> Common triggers for a DMCA include<sup>17</sup>:

- Refusal of care
- Emotional outbursts
- Seemingly irrational decisions
- Intoxication
- Delirium
- Dementia
- Psychosis

This means that any against medical advice (AMA) discharge warrants a good-faith effort to perform a DMCA using the above criteria.

**DMCAS SHOULD BE DECISION-SPECIFIC AND DYNAMIC.**

Capacity for decision-making is both dynamic and relevant to individual choices and does not constitute a static ability for all decision-making.<sup>8,12</sup> Capacity assessments should be performed in the context of an actual decision (or related set of decisions) being made. Health care decision-making capacity is inherently context-specific.<sup>1,12</sup> A patient’s capacity to decline acetaminophen, which requires limited medical understanding and has

fewer risks, may be different from their capacity to decline an abdominal aortic aneurysm repair, which requires more complex medical understanding and has serious risks. This reflects the effort, in all capacity assessments, to preserve autonomy and uphold beneficence. A term for this concept is the sliding scale principle of capacity<sup>17</sup>, which means the stringency of criteria for capacity should vary with seriousness of disease and urgency for treatment.<sup>1</sup> This concept has been controversial but has been endorsed by the President’s Commission for the Study of Ethical Problems in Medicine and Biomedical Behavioral Research and “reflects how courts actually deal with these cases.”<sup>11</sup>

**WHAT DOES A DMCA ENTAIL?**

Clinicians can use three different measures of capacity: cognitive function testing (MME), general impressions of capacity and specific capacity assessments.<sup>12</sup> The most common general impression approach uses the 4 abilities model of capacity introduced by Appelbaum borrows the criteria from legal origins and includes<sup>8,9,10,17</sup>:

1. Understanding
2. Appreciation
3. Reasoning
4. Communication

• **UNDERSTANDING:** Does this patient understand the relevant medical information being discussed, including the risks and benefits of refusing/accepting the recommended option? Use an interpreter. Use visual aids when helpful. Make sure hearing aids are present if needed. Avoid jargon. Adjust language to be appropriate to a person’s age and degree of education.

- *A good test of this is:* Can the patient summarize in their own words what has been discussed?

• **APPRECIATION:** Does this patient appreciate how the choice might affect them personally? This is one of the more difficult abilities to assess because it requires genuine attempts at understanding where your patient is coming from.

- *A good test of this is:* Can the patient appreciate how this choice will affect them personally?<sup>17</sup> Does the patient have a history of choices that align with their stated goals and values? If so, does this choice make sense in that patient’s historical decision-making?

• **REASONING:** Is there any evidence of a reasoning process behind the patient’s decision? This is also one of the more difficult abilities to assess. Can logical weighing of risks and benefits be demonstrated?

- Acknowledge that irrational decisions aren’t necessarily unreasonable in the context of an individual’s life and personal values/goals, but often in complex cases, a psychiatrist and/or ethicist can be helpful.

• **COMMUNICATION:** Is the patient able to communicate a choice? Ensure that the patient has been set up for success here by maximizing their potential in choosing the time and context to perform the assessment.

- Is their decision remaining reasonably consistent over time?

**WHAT TOOLS ARE AVAILABLE TO HELP WITH DMCAS?**

Tools that have been validated to help in these DMCAs include the open-access, Canadian-developed Aid to Capacity Evaluation (ACE),<sup>11,12</sup> which prompts the clinician to ask particular questions and attempts a scoring system to help clinicians objectively assess their patients. Another tool is the MacArthur Competence Assessment Tool,<sup>13</sup> which is not free and requires training to use. This tool has become the gold standard for capacity assessments and is useful in particularly complex cases.<sup>12</sup>

**WHAT ABOUT CONFOUNDERS LIKE INTOXICATION, PSYCHIATRIC ILLNESS, DELIRIUM, AND DEMENTIA?**

The sliding scale principle applies in these scenarios as well. The degree of intoxication and seriousness of the condition both influence the determination of capacity here.<sup>17</sup> Refusing repair of superficial laceration is different from refusing evaluation for a head bleed if the patient incurred significant trauma in a high-mechanism MVC.<sup>17</sup> It is also feasible that a patient with delirium can have capacity for a certain decision if, during lucid periods, they consistently demonstrate the 4 components previously mentioned.

Similarly, patients with underlying psychiatric illness can retain decision-making capacity for certain decisions at different time points within the fluctuations of their disease.<sup>8</sup> For the above scenarios, poor insight, as assessed by the “appreciation” component of the DMCA, is often the strongest predictor of a potential lack of capacity.<sup>17</sup>

**WHAT ABOUT SURROGATE DECISION-MAKERS?**

When someone is determined to not have capacity for a certain decision (or set of

decisions), we look to someone else to decide on their behalf. Predetermined health care proxies are the best-case scenario here.<sup>17</sup> Importantly, patients can be deemed to have incapacity for a medical decision but still may be able to tell us whom they want as their health care proxy if they are able to provide a reasoning process and communicate a choice.<sup>15</sup>

**OUR DUTY TO COMMUNICATE**

We are often biased by our personal feelings for a patient when it comes to how much time and effort we put into communicating with them, and more often than not, when a patient refuses care, the problem is not a failure of the patient’s capacity, but of our capacity for communicating and connecting with them.

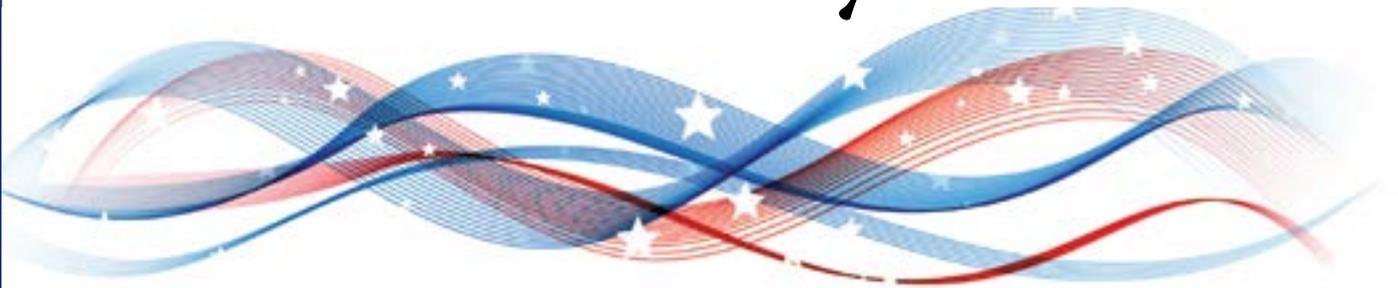
“We are obligated to do our best (without coercion) to help patients overcome their reluctance to accept care that is in their best interest. Only by talking to them, to find out what their concerns are and to respond to these concerns, can we do this. Even with patients who lack capacity and will not be allowed to refuse care, such communication is important because it may help us devise a plan with which the patient will cooperate, such cooperation being ethically and technically preferable to struggling with a combative patient.”<sup>16</sup>

When we determine a patient to lack capacity, our responsibility to protect them from harms related to restraint must become a priority and our Emergency Departments should have a systematic approach to the safe restraint of patients.<sup>14</sup>

Our responsibility to attempt therapeutic relationships and try to understand where our patients are coming from is not “window dressing.” Capacity assessments often, but not always, appear at moments of disconnection with patients and caregivers. If in these moments, the reflexive question, often in defense of the patient’s well-being, for most of us is, “Does this patient have capacity for making this decision right now?”, then we are at least on the right track. ★

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# Emergency Medicine in India: Paths Crossed and What Lies Ahead

*The specialty has advanced substantially from its nascent origins. The country's medical community has recognized the need for quality emergency care, but further progress must be made in EM research, care delivery, and future-physician training to optimize patient outcomes.*

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**E**mergency medicine in India has advanced substantially from its nascent origins and continues to expand at an exponential pace. The country's healthcare community has recognized the need for quality emergency care as the number of citizens affected by motor vehicle accidents, communicable and non-communicable diseases, and natural disasters continues to grow.

The Society for Emergency Medicine in India (SEMI) was established in 1999, and EM was recognized as a specialty by the Medical Council of India by 2009. Decision makers acted

quickly to recognize the importance of training quality emergency physicians and enhancing patient care across the country.

Around the world, emergency departments have served as the face of the hospital, but lower middle income countries have been slow to adopt this model until recently. Previously, EDs in India would be staffed by junior medical doctors who were limited in their emergency care due to low resources, limited clinical training, and poor administration. Many hospitals believed there was no need for an around-the-clock service, but soon realized the importance of prompt and good quality emergency care.

India loses more than 3% of its gross domestic product due to traffic accidents.<sup>1</sup> With the dramatic increase in traffic accidents across the country, healthcare providers have highlighted the damaging impact poor emergency care can have on the economy.

We were fortunate to interview Dr. Arun Nandi, associate professor of emergency medicine at the Icahn School of Medicine at Mount Sinai Hospital in New York City. Dr. Nandi serves as ACEP's ambassador to India, Nepal, and Sri Lanka.

Discussing the differences between the Indian and U.S. populations, Dr. Nandi noted that patients presenting to the ED in the Indian subcontinent often present at the late stages of their illnesses. Few patients visit doctors for primary care. Patients, therefore, present late and with greater severity and pose a challenge for resource-limited doctors. Timely and standardized emergency care in India is essential to improve health

outcomes and economic well-being. Emergency medical services across the country vary from state to state with no centralized system, further complicated by terrain, weather, and geopolitical conflict.

India's EM infrastructure was limited in its early development partly because stakeholders believed illnesses could be better treated by specialists than emergency physicians. However, research showed the effectiveness of EM interventions that expedited care and improved outcomes of time-sensitive conditions like trauma, stroke, and myocardial infarction.

Medical infrastructure is a large determinant in the standard of care delivered and, while India has advanced significantly in the past two decades, there is much left to learn. To improve patient outcomes and the quality of emergency care, India needs to focus on three aspects: education, delivery of care, and research.

Regarding education, administrators must emphasize the development of standardized training for emergency physicians, with ample opportunities for employment and promotion, including simulation-based and experiential learning.

For proper delivery of care, EDs must be given adequate space within the hospital for triage, diagnosis, and transfer of care.

Lastly, research into health outcomes and quality improvement is needed to enhance patient care through continuous quality improvement, especially for common ED presentations such as stroke, MI, toxicologic emergencies, and polytrauma.



We also interviewed Dr. Sushant Chhabra, ACEP's ambassador liaison from India, chief of emergency medicine at Manipal Hospital, New Delhi, and a regional leader of SEMI.

When asked about the EM training programs in India, Dr. Chhabra said they are carried out under different banners. The MD (postgraduate degree) and DNB (Diplomate of National Board) postgraduate programs host EM across a few hospitals in India, but the number of positions for trainees is insufficient.

Then, there are 3-year training programs like the Masters in Emergency Medicine (MEM), an international program affiliated with George Washington University. This is a structured program with equal weight for academics, clinical skills, and research. EM faculty from the United States, United Kingdom, Australia, Singapore, and India train residents to grow into qualified emergency physicians.

A similar program called CCT-EM (certificate of completion of training in emergency medicine) has been

implemented by SEMI.

In addition to these major 3-year programs, there are minor 1- to 2-year diplomas and fellowships in EM awarded

by local universities like Medvarsity Apollo.

Since Dr. Chhabra's EM residency in 2011, there has been a significant





expansion in training programs. This expansion came about as healthcare systems began to recognize the importance of EM in hospital operations and revenue. However, Dr. Chhabra believes extensive progress — which includes gaining governmental support to establish the emergency department as the “face” of the hospital across the Indian subcontinent — has yet to be made. The biggest challenge is a lack of EM-trained faculty. Hence, within some courses there is “self-learning,” or teaching by a specialist who isn’t an emergency physician. In his research paper, Dr. Chhabra conducted a survey of the perception of medical education among EM residents in India and concluded that programs must adopt a “student-centered educational atmosphere” that will ultimately lead to well-trained emergency physicians tending to the population of India.<sup>2</sup>

When discussing the impact of the COVID-19 pandemic, Dr. Chhabra described it as a double-edged sword that enabled emergency physicians to

### **Global health leaders should note the resourcefulness and adaptability of India’s emergency physicians in treating severe, end-stage diseases at their first presentations in the ED, similar to situations in many marginalized areas within the United States.**

attend important medical conferences virtually, but also deprived them of the “hands-on” experience pivotal for their residencies. Furthermore, in India — just like in many other nations — the COVID-19 pandemic exposed weak points in the healthcare system, overwhelming the country’s doctors and hospitals. Limited access to primary care facilities caused patients with milder COVID cases to occupy beds at hospitals when many of them could have managed

their symptoms in an outpatient setting. Challenges with organization, logistics, and disaster management complicated this public health emergency and showcased the scarcity of resources. Dr. Chhabra believes that by improving facets of the country’s fundamental healthcare infrastructure, from disaster management to primary care, patient outcomes will improve as a whole.

Our conversations with Drs. Nandi and Chhabra were enlightening and informative. We learned about the great advances in emergency care across the Indian subcontinent and potential ways that its delivery could be improved. Global health leaders should note the resourcefulness and adaptability of India’s emergency physicians in treating severe, end-stage diseases at their first presentations in the ED, similar to situations in many marginalized areas within the United States. However, further progress must be made in EM research, care delivery, and future-physician training to optimize patient health outcomes in India. ★



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# Trichobezoar: A Case of Swallowed Hair in an Adolescent Patient

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## ABSTRACT

A trichobezoar is an intraluminal mass in the stomach or intestine composed of undigested swallowed hair. It primarily occurs in adolescent female patients with trichophagia, trichotillomania, and other underlying psychiatric disorders. Presenting symptoms can range from abdominal pain, nausea and vomiting, or an asymptomatic abdominal mass, and can progress to acute complications such as obstruction, perforation, and peritonitis. More chronic complications include weight loss and anemia due to malabsorption. Patients often present multiple times with subtle symptoms prior to being diagnosed.

## INTRODUCTION

Abdominal pain is one of the most common chief complaints seen in the emergency department. Differentials are broad and can encompass nearly every organ system. However, it is important to remember one rare diagnosis: trichobezoar. Trichobezoar is the most common type of gastric bezoar and is associated with psychiatric disorders such as trichotillomania and trichophagia. Approximately 5-20% of those with trichotillomania also engage in trichophagia, which could result in trichobezoar, a large quantity of hair firmly matted together.<sup>1</sup> A trichobezoar will typically cause abdominal pain and nausea/vomiting, but it can result

in erosions, ulceration, intestinal obstruction, gastric perforation, and peritonitis.<sup>1-4</sup>

Here we present a case of an 11-year-old girl with a history of anxiety who presented to the ED with abdominal pain and was diagnosed with a gastric perforation secondary to trichobezoar.

## CASE

An 11-year-old girl with a past medical history of anxiety presented to the emergency department with abdominal pain. Our patient was seen one week prior in the ED for generalized abdominal pain and back pain. At that time, she was noted to have abdominal distension on physical exam and was

discharged home with supportive care without labs or imaging.

The patient presented this visit to the ED with constant abdominal pain associated with multiple episodes of non-bloody, non-bilious emesis. On arrival, her vital signs were heart rate 120-130 beats per minute, respiratory rate 50 breaths per minute, saturating 100% on room air, temperature 98.7°F. On physical exam, the patient was noted to be tearful, pale, and ill-appearing with abdominal distension and diffuse tenderness.

Abdominal X-ray showed a large amount of free air in the abdomen and a dilated bowel loop concerning for “bowel loop containing debris measuring up to 14 cm” (Figure 1). She was empirically started on IV piperacillin/tazobactam. On re-examination in the ED, the patient became hypoxic to 76% on room air and was ultimately intubated, with a repeat X-ray showing an increase in pneumoperitoneum (Figure 2).

After intubation, the patient became hypotensive and was started on norepinephrine. Labs were significant for Hgb 7.4 g/dL, MCV 65 fL, platelets 1,341 bil/L, and WBC 7.8 bil/L. Lactic acid was 8.7 mmol/L, Cr 0.95 mg/dL, and CO<sub>2</sub> 12 mmol/L. The patient was transferred in stable condition to an outside facility for a higher level of pediatric care.

The patient was subsequently given additional intravenous fluids, started on a ketamine drip, and taken to the operating room. In the OR, gastric perforation was identified along the lesser curvature of the stomach near the angularis incisura due to a large gastric trichobezoar, with subsequent removal of the trichobezoar (Figures 3 and 4).

The patient was ultimately continued on antibiotics and extubated. A follow-up upper GI study showed small leakage along the lesser curvature and a postoperative ileus (Figure 5). Psychiatry evaluated the patient. She was diagnosed with generalized anxiety disorder and trichotillomania with trichophagia, and was started on fluoxetine. She was discharged home on postoperative day nine.

## DISCUSSION

Gastric bezoars are an intraluminal mass



FIGURE 1: Pneumoperitoneum on abdominal X-ray with dilated bowel loop

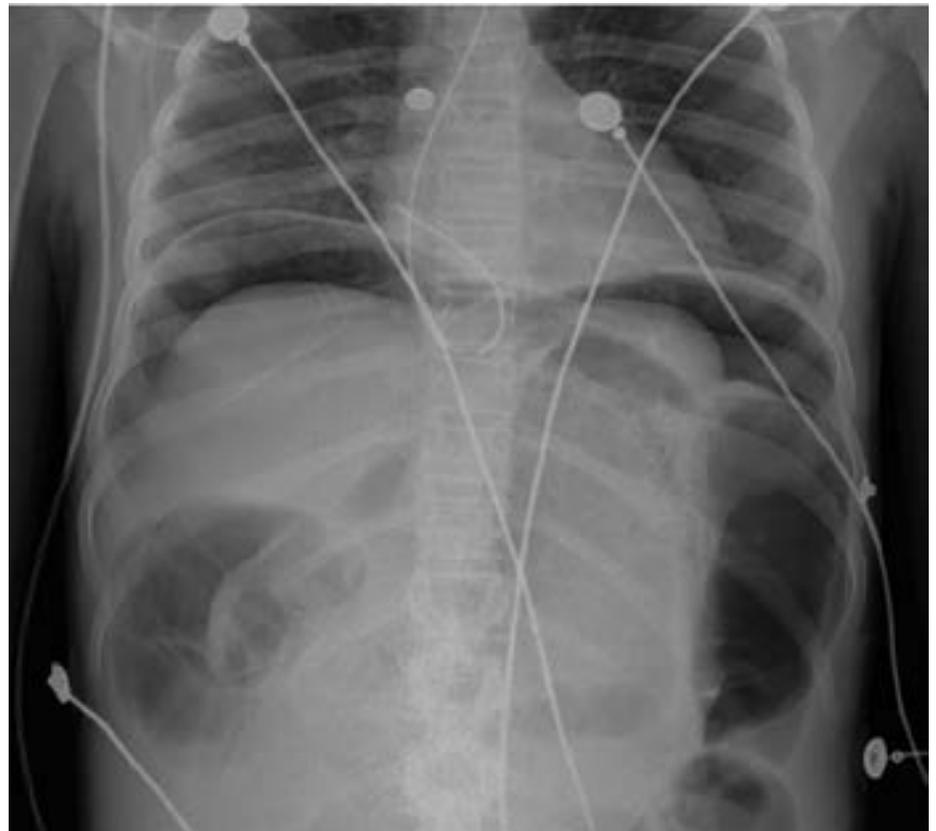


FIGURE 2: Follow-up CXR demonstrating worsening pneumoperitoneum

due to the accumulation of undigested material. Although not completely understood, trichobezoars, accounting for 50% of bezoars, are thought to form as the smooth, slippery, enzyme-resistant hair stays in the gastric mucosal folds

as it escapes the stomach's peristaltic propulsion,<sup>2-5</sup> eventually accumulating and taking shape of the stomach due to the peristalsis.<sup>3</sup> The bezoar is then covered in mucus and gives off a putrid smell due to the fermentation of fats,



potentially causing halitosis.<sup>4</sup> Although most trichobezoars are located in the stomach due to the pyloric sphincter preventing migration, small hair can migrate as a tail through the pylorus into the intestines and colon; this is called Rapunzel syndrome.<sup>3-4</sup>

As with our patient, many are misdiagnosed on initial presentation with generalized abdominal pain, possibly delaying patient care and leading to complications. Malhotra-Gupta noted in a case review that three of 18 pediatric patients had prior diagnoses before they were finally diagnosed with trichobezoar and gastric perforation.<sup>6</sup>

With delayed diagnosis, bezoars can increase in size. This reduces blood supply to the stomach mucosa, causing ischemia specifically to the limited blood supply watershed area of the lesser gastric curvature, thus causing erosion of the gastric mucosa and eventually leading to ulceration and perforation.<sup>3,5-9</sup>

Treatment includes removal of the trichobezoar, prevention of complications, and treatment of the

underlying cause. Removal includes endoscopy; however, surgery is indicated with perforation, hemorrhage, or Rapunzel syndrome.<sup>3</sup>

After treatment of the trichobezoar, a multimodal preventative therapy — specifically addressing the underlying cause — is needed to prevent recurrence. Fluoxetine and other SSRIs have been used to treat the underlying behavior of hair consumption, although no reproducible patient benefits have been seen.<sup>5</sup>

Since the start of the COVID-19 pandemic, there has been an increase in pediatric mental health visits both in the ED and outpatient, especially in females and adolescents.<sup>10-14</sup> Saunders et al. found that there was a 10-15% increase in outpatient mental health utilizations, with higher ED visits and hospitalizations in females, specifically 7 to 12 years old.<sup>10</sup> A global meta-analysis showed that specifically anxiety and depressive symptoms have doubled in prevalence since the pandemic and increased throughout the pandemic in

females.<sup>13</sup> Krass et al. even reported that 62.6% of mental health visits were female patients.<sup>14</sup> This adds to the growing body of evidence for increasing rates of pediatric mental health prevalence.<sup>11-14</sup>

Trichobezoar prior to the pandemic had an estimated incidence of 0.4 to 1% in the general population, mainly in women younger than 30 years old,<sup>2,3,5</sup> with most patients diagnosed having concurrent psychiatric or neurological disorders.<sup>4</sup> However, with the rise in pediatric mental health emergencies in the past two years, it is important to consider this diagnosis and screen all patients, especially adolescents, for anxiety and depressive symptoms, with subsequent habits such as hair swallowing.

**CONCLUSION**

Trichobezoars are an uncommon diagnosis but should be considered in the differential for patients presenting to the emergency department with abdominal pain. It is especially important not to overlook the possibility for this diagnosis in young female patients with underlying psychiatric disorders. ★

**TAKE-HOME POINTS**

- Consider broad differential diagnosis for pediatric patients with abdominal pain.
- Screen all patients for depression and anxiety.
- Consider obtaining preliminary X-rays in pediatric patients with abdominal pain.



FIGURE 3: Trichobezoar in the stomach



FIGURE 4: After removal



FIGURE 5: UGI series showing small leak of contrast from the lesser curvature



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# I Ordered the Urine Drug Screen, Now What?

## An Overview on Testing, Interference, and Utility of UDS in the ED

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### CASE

A 36-year-old woman presents to the ED with chief complaints of altered mental status, cough, fever, and runny nose. Her vitals are: BP 104/62; pulse 120; respiratory rate 28; SpO<sub>2</sub> 96% on room air; and temperature 100.5°F. Her

friend at bedside notes the patient has been taking acetaminophen cold and flu, pseudoephedrine, diphenhydramine, ibuprofen, and dextromethorphan to treat her current acute symptoms. The patient has a history of asthma and frequent marijuana use. Differential

diagnosis includes a polypharmacy overdose such as acetaminophen or diphenhydramine, illicit drug intoxication, viral encephalopathy, and electrolyte abnormality. You decide to order a head CT, CBC, CMP, TSH with reflex T<sub>4</sub>, EKG, aspirin and

acetaminophen levels, Flu/COVID/RSV swab, and a urine drug screen (UDS).

## INTRODUCTION

The UDS is a frequently ordered test in the emergency department as well as in inpatient services, psychiatry, pain medicine, and outpatient clinics. Nearly 30 million ED visits per year are associated with some form of drug use.<sup>1</sup>

In non-emergency medicine settings, these drug screens can be used to evaluate pharmacologic diversion, medication compliance, and the possibility of recent or current intoxicants.

In the ED, the UDS allows the emergency physician to ascertain the nature of a patient's current or recent toxicity, which can assist in the narrowing of a differential diagnosis. For example, the UDS may play a role in determining if a chest pain patient is suffering from an acute occlusive myocardial infarction or amphetamine-induced vasospasm, or if an unresponsive patient may have a substance-related cause for their obtundation.

Despite the ubiquitous nature of these tests — which are widely used as screening and diagnostic tools within medicine as well as in the sports industry and business sectors — many healthcare providers are skeptical to trust the results for a variety of reasons. To best understand the utility (or possible lack thereof) of the UDS, it is important to analyze how the test is actually performed.

## THE SCIENCE BEHIND THE TEST

Although hair, fingernails, toenails, and saliva can be used to detect the presence of drugs, hospitals most commonly employ the use of blood or urine assays. These assays take the form of an immunoassay or gas chromatography combined with mass spectrometry (GS-MS).<sup>2</sup> The latter is considered the gold standard of detection and has the capability to detect innumerable substances if the column used is calibrated appropriately.

Not all hospitals have access to GS-MS due to the cost and maintenance of the instrument or a lack of qualified

**TABLE 1. SUBSTANCE DETECTION TIME AND COMMON FALSE POSITIVES AND NEGATIVES.**<sup>4,7,8,9,10</sup>

SUBSTANCE (Tested Metabolite)	Detection start	Detection end	False positives	False negatives
Amphetamines (-amphetamine)	4-6h	2-3d	Pseudoephedrine (Sudafed), phenylephrine, amantadine, atomoxetine, bupropion, chloroquine, metformin, ranitidine, chlorpromazine, trazodone, promethazine, hemp-containing foods, proton pump inhibitors	MDA, MDMA
Benzodiazepines (oxazepam)	2-7h	Most 3-5d Diazepam - 10-30d	Coca tea, some forms of yerba mate, sertraline	Clonazepam, lorazepam, alprazolam, midazolam,
Phencyclidine	4-6h	7-14d	Dextromethorphan, venlafaxine, ibuprofen, thioridazine, diphenhydramine, tramadol, ketamine, lamotrigine, zolpidem	None
Opiates (morphine)	2-6h	1-3d	Levofloxacin, ofloxacin, imipramine, naltrexone, rifampin, dextromethorphan	Oxycodone, hydrocodone, tramadol, methadone, buprenorphine - synthetic opioids
Cocaine (benzoylecgonine)	2-6h	12h, possibly 2-4d	Coca leaves in tea drinks	None
Cannabinoids (11-carboxy-delta9-tetrahydrocannabinol)	1-3h	3-30d depending on chronicity of use	Promethazine, NSAIDs, pantoprazole	Synthetic/ designer cannabinoids
Tricyclics	8-12h	2-7d	Cyclobenzaprine, quetiapine, carbamazepine, cyproheptadine, hydroxyzine, cetirizine, diphenhydramine	None
Barbiturates	2-4h	Short-acting up to 24h, long-acting up to 3 weeks	Ibuprofen, naproxen	Sodium thiopental

personnel.<sup>2</sup> Regardless, it is the urine immunoassay that most emergency physicians are familiar with ordering. Immunoassays utilize pre-made antibodies to a particular substance of interest (e.g., cocaine metabolite) and the presence or absence of a color-producing enzyme as a surrogate method of detecting the presence of the substance. This is a qualitative process that generally answers the binary question of “drug or no drug” without providing details such as last time used or quantity.<sup>2</sup>

Unfortunately, immunoassays can be ripe with error. The antibodies may bind substances with a similar structure to the target of interest despite not actually being the drug in question, or in contrast, they may miss drugs because of chemical modifications or lack of a common metabolic pathway.<sup>2</sup> Due to this

cross reactivity of compounds with the immunoassay, confirmatory testing is required to ensure that the result is not a false positive.<sup>2</sup>

Herein lies the big controversy with the UDS: Can we trust it? Do the results matter?

#### THE LITERATURE

Most emergency departments use two separate types of drug screens: a narrow or five-panel screen, and an expanded panel screen. Typically, the five-panel screen evaluates for the presence of amphetamines, benzodiazepines, opiates, cocaine, and cannabinoids.<sup>3</sup> The expanded panel includes tricyclics, fentanyl, barbiturates, methadone, and phencyclidine (PCP), in addition to the five-panel substrates.<sup>3</sup> These tests are independent of specific urine tests that exist such as a urine fentanyl screen.

It is important to note that the UDS components vary from hospital to hospital.

In **Table 1**, we detail some common substrates and the intricacies around their testing.

Positive results in the UDS can be affected by many external factors including patient body mass, short-term versus long-term use of the drug, pH of the urine, and last ingestion time.<sup>4</sup> There are several common drugs that do not have any false negatives found in the literature. These include PCP, TCAs, cocaine, and LSD. Thus, if a patient admits to ingesting these drugs and tests negative, the level in their urine is likely below the cut-off value. In addition, patients can adulterate their urine through the addition of substances such as Visine drops, which frequently can



## The urine drug screen is a widely used, but imperfect, diagnostic test. Because of its potential flaws, it should not be used out of context or in isolation, but it remains a valuable test in the toolbox of the emergency physician.

cause false negatives.<sup>4</sup>

In a retrospective analysis of more than 8000 urine samples, Johnson-Davis and colleagues showed that there is a significant false positive rate depending on the immunoassay used for urine screening. With amphetamines, there were up to 14% false positives, and with opiates, a 34% false positive rate. With other drugs of abuse, the false positive rate was significantly lower, with 0.4% for benzodiazepines and 0.9% false positive for THC.<sup>5</sup>

Urine drug screens also fail to mirror emergency physician clinical gestalt. In a small single-center study, Gilfillan and colleagues used urine drug screening results and physician notes to determine if clinician suspicion was correct. In this study, 35 patients were suspected of drug use but only 14 tested positive, showing clinicians to be correct only 40% of the time. Of 58 patients the clinicians did not suspect, 18 — or 31% — tested positive.<sup>6</sup> This study did not use any confirmatory testing. It is certainly possible that clinician gestalt was inaccurate, but given the overwhelming data of UDS inaccuracies, it is likely that many of these drug screens had false positives or false negatives.

### DISCUSSION

With possible false positives and false negatives, emergency physicians must be cautious in using the UDS to inform and guide care in the ED. The UDS is severely limited given the wide variation in false positive and lack of sensitivity in identifying drugs of a particular class (e.g., benzodiazepines or opioids) as detailed in **Table 1**.<sup>4,7,8,9,10</sup>

Of significant clinical importance recently is the ability to detect the presence of opioids. As the opioid epidemic continues, EDs everywhere have been plagued by street adulterants

contaminated with fentanyl and analogs, none of which screen positive on a UDS given they are not metabolized through morphine like heroin. Additionally, emergency physicians must consider many other common sources of intoxication and overdose that are not typically included on urine drug screens, such as ketamine, mescaline, gamma-hydroxybutyrate (GHB), and synthetic cannabinoids.<sup>2</sup> Providing care to a trauma activation or sexual assault victim may require testing outside the UDS in specific circumstances, such as drug-facilitated sexual assault.

Many psychiatric patients are required to have a UDS result for placement to ensure that possible drug withdrawals or concomitant substance-use counseling can be included during their hospitalization. Multiple studies performed within the United States and abroad have found that there is no significant change in patient disposition, clinical outcome, or length of stay in a psychiatric facility based on the results of a urine drug screen.<sup>11,12,13</sup> Kagel evaluated the UDS in an otherwise healthy group of 682 patients at a U.S. military hospital; the study found no positive UDS, and patients had no change in disposition or care.<sup>11</sup>

Another study in a single-center ED in a state psychiatric hospital in Israel found no change in disposition of patients based on the UDS. Drug use as high as 20-30% was predicted, but only 9% positivity was found. The study determined that UDS use for psychiatric patients should be more focused on those suspected of drug-induced psychosis.<sup>12</sup>

And yet another study by Schiller and colleagues at a large urban psychiatric emergency center divided 392 patients into a usual care group and a mandatory urine drug screen group. Schiller determined that when left to clinician

gestalt versus testing of all psychiatric patients with urine drug screening, there was no change in disposition or treatment length between the mandatory screen versus the usual care group.<sup>13</sup>

Others argue the drug test can significantly improve accuracy of diagnosis and thus inform treatment after disposition. One study by Szuster and colleagues found a one-third increase in substance-induced organic mental disorders when a urine drug screen was used for confirmation of drug use instead of a questionnaire or direct patient inquiry.<sup>14</sup> Elangovan supported this finding through a high rate (26%) of cocaine use in the tested psychiatric patient population at a single center, but a low rate of admission of use (13%) in the tested population at a psychiatric facility.<sup>15</sup>

Many overdoses involve multiple medications. If the patient's clinical presentation does not match the UDS, consider the presence of coingestants and non-tested ingestions, and ensure the patient's medication list has been thoroughly reviewed for possible confounding agents.

Physicians should also remember that the effects of most of these substances wear off long before they are no longer detected in the urine. Be careful of anchoring bias, especially if a patient is forthcoming about when they ingested the drugs they tested positive for on a UDS.<sup>7</sup>

The UDS transcends bedside clinical care to the far reaches of healthcare policy. In the age of value-based care, physicians are rewarded for providing the highest quality care at the lowest cost.<sup>16</sup> As the Centers for Medicare & Medicaid Services (CMS) continues to expand the notion of value-based care, it is reasonable to think that ordering a test that rarely changes management

or disposition may not be reimbursed. Testing that is not reimbursed or not indicated by CMS may lead to a withholding of physician incentive payments.

**SUMMARY**

For emergency physicians, the urine drug screen offers a practical way to assess possible causes of altered mental status for obtunded patients and may aid our psychiatry colleagues in psychiatric placement. It may also serve a role in confirming potential substances taken in overdose (e.g., tricyclics and benzodiazepines). However, given the risk of false negatives, clinicians should rely on additional laboratory studies and physical examination to guide clinical management. Numerous studies have shown that the UDS does not impact changes in disposition or management. The urine drug screen is not a foolproof diagnostic test. However, it is another tool in the toolbox of the emergency physician. Because of its potential flaws, it should not be used out of context or in isolation.

**CASE CONCLUSION**

The UDS returns positive for amphetamines, PCP, THC, and TCA. The patient’s friend at bedside is adamant the patient could not have ingested any of these medications and that she had not taken anything other than what was originally listed. The patient’s symptoms also do not seem to match this array of drug ingestion. You consider that several of these results are likely false positives. You request that the lab performs confirmatory testing. In the meantime, the patient is treated for multi-med ingestion with normal saline and benzodiazepines, and is closely monitored in the ICU. The confirmatory testing returns positive only for THC and negative for amphetamines, PCP, and TCA. Given the patient’s co-ingestions, she likely tested positive for these additional substances due to her aggressive treatment of her cold symptoms, leading to her altered mental status. ★



**TAKE-HOME POINTS**

- The UDS is an imperfect screening test and has been shown to minimally impart any change in clinical management or disposition, so use with caution.
- When in doubt, order confirmatory testing. See what is available in-house at your hospital and what requires a sendout assay.
- Understand which adulterants are tested for in your institution-specific panels.
- Obtain careful history of recent medication use, both prescribed and over-the-counter.

**TIPS**

- The addition of glucuronidase to the UDS increases the sensitivity of benzodiazepine detection as it cleaves off the large glucuronide moiety which interferes with analysis of medications not metabolized through an oxazepam intermediate.<sup>17</sup>
- Delta 8 THC, the “legal” hemp derivative sweeping the country, leads to a positive test on the UDS for cannabinoids, given the overwhelming structural similarity.<sup>18</sup>



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# Warfarin-Induced Skin Necrosis: Not Your Typical Rash

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## CASE

An 81-year-old man with a past medical history of multiple deep venous thrombosis (DVT), pulmonary embolism (PE), and paroxysmal atrial fibrillation presented to the emergency department with an acute onset bilateral arm rash. Prior hypercoagulable workup was negative for factor V Leiden mutation, prothrombin gene mutation, protein C and S deficiency, and antiphospholipid syndrome.

The patient was recently transitioned to oral anticoagulation warfarin from subcutaneous weight-based enoxaparin.

The patient sought treatment two hours after performing yard work. He noticed a rash spreading on his left forearm with pain and paresthesia. He denied pruritus, trauma to the area, or prior history of rashes or allergic reactions. The patient admitted to holding a pesticide canister in the affected limb while he was spraying shrubbery.

His clotting history was significant for a provoked DVT with subsequent PE in 2006, requiring three months of anticoagulation with warfarin without any complications. The patient had a second unprovoked DVT in 2011 and was

placed on lifelong anticoagulation with warfarin. In 2019, anticoagulation was switched from warfarin to a direct oral anticoagulant with apixaban due to new onset paroxysmal atrial fibrillation.

In May 2022, the patient developed an unprovoked DVT of his left femoral vein with complete occlusion of the mid segment while on apixaban. The patient was transitioned to weight-based enoxaparin for anticoagulation. Due to injection site discomfort with enoxaparin, the patient opted to transition back to warfarin for anticoagulation with an enoxaparin bridge.

Physical exam was pertinent for purpura with ulceration and blistering on the dorsum aspects of the forearms bilaterally. The left forearm demonstrated a greater girth and more profound rash. No bullae were appreciated (**Image 1**). Basic laboratory workup was significant for a therapeutic INR of 2.8, a baseline hemoglobin level of 11.7 mg/dL, and platelets of  $215 \times 10^9/L$ . X-rays demonstrated no evidence of fracture or subcutaneous soft tissue calcium deposits.

As the rash was not characteristic of traumatic ecchymosis, hematology/oncology was consulted for suspected warfarin-induced skin necrosis (WISN).

Hematology/oncology recommended additional diagnostics with a DVT ultrasound of all extremities and a baseline protein C activity. The DVT ultrasound of upper extremities was negative for any thrombi, and the DVT ultrasound of lower extremities showed a partial occlusion of the left femoral vein, which was mildly improved from May 2022. Intravenous vitamin K 10 mg and 2 units of fresh frozen plasma (FFP) were administered, and continuous



IMAGE 1: Purpuric rash of the left forearm with several annular ecchymoses



intravenous heparin was started prior to hospital admission.

#### DISCUSSION

WISN is a rare complication occurring in 0.01-0.1% of patients taking warfarin, especially at higher doses.<sup>1</sup> It is most commonly seen in middle-aged women, occurring on the breast, buttocks, and thighs, but can be present in any location.<sup>1</sup> It most commonly occurs within 5-10 days, as it takes warfarin several days to have full effect.<sup>2</sup> The pathogenesis is believed to be related to the reduction of vitamin-K dependent anticoagulant factors, and proteins C and S. Vitamin-K dependent clotting factors II, VII, IX, and X are also reduced, but proteins C and S have significantly shorter half-lives compared to clotting factors II, VII, IX, and X.<sup>2</sup> This decrease in proteins C and S creates a paradoxical hypercoagulable state, which leads to thrombotic occlusion of the microvasculature and resulting necrosis.<sup>2</sup> Patients started on warfarin

are frequently bridged with a heparin product for at least five days of therapy or until therapeutic INR goal is achieved to prevent WISN.<sup>3</sup>

#### DIAGNOSIS

Warfarin-induced skin necrosis is a clinical diagnosis but is supported by predisposing hypercoagulable diseases to include protein C and S deficiency, antiphospholipid antibody syndrome, and antithrombin gene mutation.<sup>4</sup> The rash characteristically starts as pain and paresthesia with an initial central erythematous macule which, as edema develops, progresses to a central purpuric zone, then finally necrosis.<sup>4</sup>

Lab work can be beneficial for further management of the patient outside of the ED and is necessary to evaluate for other pathologies that can mimic WISN. The differential includes hematoma, disseminated intravascular coagulation, purpura fulminans, cellulitis, necrotizing fasciitis, and calciphylaxis.

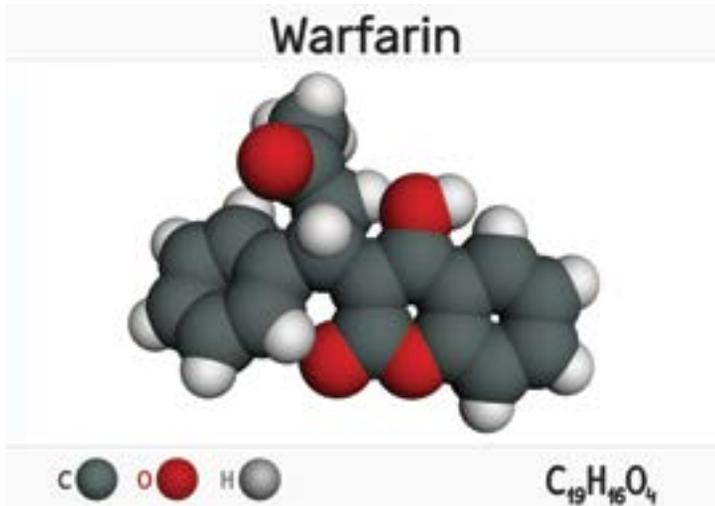
The patient was treated for WISN

and started on heparin-based therapy to prevent further microvascular thrombosis. He did well inpatient with improvement of his lesions while on heparin and was transitioned to subcutaneous enoxaparin. His lab work returned a protein C activity assay of 45%, which favors acquired protein C deficiency, likely secondary to diet lacking in vitamin K. Acquired protein C deficiency can also be seen in DIC, infection, and hepatic dysfunction; further investigation through alternative testing modalities is necessary for confirmation.<sup>5</sup>

Given suspicion of protein C deficiency, the patient was advised to complete a prolonged bridge with weight-based enoxaparin prior to transitioning back to a lower dose of warfarin.

#### MANAGEMENT

WISN management involves reversing warfarin and starting the patient on a heparin product to prevent further microvascular thrombi.<sup>3</sup> In this case,



warfarin was reversed utilizing 10 mg of vitamin K and two units of FFP.<sup>3</sup> Vitamin K is used to provide enough substrate to overcome the inhibition of vitamin K epoxidase inhibition by warfarin. FFP contains all of the vitamin K-dependent coagulation factors for reversal of coagulopathy.<sup>6,7</sup>

Other reversal options for patients with fluid overload include 4-factor

prothrombin complex concentrate (PCC), which has purified concentrates of factors II, VII, IX, and X for reversal of coagulopathy.<sup>6,7</sup> However, PCC can contain varying protein S and C concentrates depending on the manufacturer.<sup>7</sup>

#### CASE CONCLUSION

The patient was concomitantly

transitioned to an unfractionated heparin drip. Low molecular weight heparin is another reasonable option; however, we opted for a titratable option in the event of complications for this patient who required admission.

#### CONCLUSION

Rash and skin lesion differentials are vast and often subtle. Varying clinical scenarios present a diagnostic challenge for emergency physicians at any stage of their career. Warfarin-induced skin necrosis is a rare condition thought to be due to microvascular thrombosis and ischemia and is commonly related to protein C deficiency. Recognition of WISN is critical to reduce morbidity and mortality. The level of protein C is not necessary for diagnosis. Principles of management include reversing warfarin therapy with vitamin K and FFP as well as starting the patient on therapeutic heparin with hematology/oncology consultation. ★

# Case-con

Medical Student and Resident Case Study Contest

Sign up and start prepping for  
**Case-Con at ACEP23 in Philadelphia!**  
 You'll present interesting and notable  
 EM cases in this modern take on  
 poster sessions.  
 Three winners per category!



More info & registration  
 details coming soon



Welcome Aboard!

# EMRA 2023-24 Medical Student Council

We'd like to say **CONGRATULATIONS** to our newly appointed Medical Student Council. EMRA is grateful for your leadership!

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EMRA's Medical Student Council is the voice of medical students pursuing emergency medicine — in other words, the voice of the future of the specialty. Applications for MSC leadership positions are due Nov. 1 of each year. Each spring, we welcome new MSC leaders, who serve a one-year term beginning March 1 and ending March 31 the following year.

EMRA would like to extend a big, heartfelt welcome and congratulations to our incoming MSC leaders for 2023-24!



[more info](#)



## Congratulations

# EMRA 2023-24 Committee Leaders

We'd like to say **CONGRATULATIONS** and welcome aboard to our newly appointed committee chairs, chairs-elect, vice chairs, and assistant vice chairs. EMRA is happy to have you at the helm!

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Medical University of South Carolina

**March 21-24, 2023 CORD Academic Assembly in Las Vegas, NV**





## A Case of Constipation ... or Not

### Karly Lebherz, MD

Resident, PGY-2  
Emergency Medicine  
Ochsner Health/Ochsner Hospital for Children

### Anna Suessman, DO, MEd

Director of Education  
Dept. of Pediatric EM  
Ochsner Health

#### CASE

A 2-year-old male born at 37 weeks' gestation presented to the emergency department for abdominal distension and constipation. He was discharged from the hospital three days ago after a three-day admission for acute respiratory failure with hypoxia secondary to COVID-19. His last normal bowel movement was four days prior, despite home use of stool softeners and enemas. His mom denied fevers, vomiting, and anorexia, but did report intermittent periorbital edema for an unclear amount of time. During his recent hospitalization, he was not noted to have edema or signs of fluid overload.

The patient's vital signs in the ED were as follows: HR, 100-128; RR, 22-28; temperature, 37°C; SpO<sub>2</sub>, 97-98%RA;

and weight, 12.7kg (11.3kg at most recent discharge).

Physical exam showed an alert child crying while examined and nontoxic in appearance. The patient's abdomen was distended and full, but not tense. Bowel sounds were present. Discomfort with palpation, but no focal tenderness/rebound/guarding. No stool in the rectal vault.

Pertinent lab results: (CBC) WBC 25, Platelets 940; (CMP) Potassium 3.0, Albumin 0.7, Alkaline Phosphatase 104; (Urinalysis) Protein 3+, Occult Blood 2+.

#### DIFFERENTIAL DIAGNOSIS, NEXT STEPS

Differential diagnoses: nephrotic syndrome (minimal change disease, focal segmental glomerulosclerosis,

membranous nephropathy), glomerulonephritis, liver disease (cirrhosis, Alpha-1 antitrypsin deficiency), protein-losing enteropathy, and malnutrition.

ED workup continued: CT abdomen and pelvis showed small pleural effusions, large amount of ascites, dense liver (consider hepatic etiology for ascites), mildly atrophic left kidney.

The patient was admitted for further management including electrolyte replacement and consultation by pediatric nephrology and gastroenterology. Paracentesis was considered but deemed unnecessary from a therapeutic standpoint due to stable respiratory status. Gastroenterology recommended stool alpha one antitrypsin testing. Nephrology agreed

with the suspected diagnosis of nephrotic syndrome versus minimal change disease and recommended a low-sodium diet with fluid restriction and 1mg/kg steroids twice daily. The patient has not yet returned for follow-up.

## DISCUSSION

Nephrotic syndrome (NS) is diagnosed by the presence of proteinuria (protein excretion greater than 50 mg/kg/day or greater than or equal to 3+ proteins on urine dipstick), hypoalbuminemia (less than 2.5 g/dL), and peripheral edema. Hyperlipidemia and thrombotic disease are also frequently observed.<sup>1,2</sup>

Additional workup should include a lipid panel, ANA, C3, C4, viral hepatitis, and chest x-ray to evaluate for possible pleural effusions if suspected, and renal US if renal vein thrombosis is suspected.<sup>2</sup>

Most cases of NS in pediatric patients are secondary to minimal change disease (MCD). A presumptive diagnosis of MCD can be made without a renal biopsy based on clinical findings (e.g., age 1-9 years, absence of HTN/hematuria, normal complement levels and renal function). The most common risk factor is recent illness, particularly upper respiratory tract infection. Presenting symptoms commonly include periorbital/genital or, less commonly, lower extremities edema, ascites/pleural effusions, and fatigue with or without weight gain.<sup>4</sup>

There have been a few recent cases in the pediatric community with COVID-19



infection preceding NS. To date, there has been one published case of new onset NS reported with COVID-19 infection. Recurrence of nephrotic syndrome has also been reported in a pediatric patient ill with COVID-19.<sup>5</sup> More than one-third of patients admitted to the hospital for COVID-19 infection also develop acute kidney injury (AKI), with many requiring kidney-replacement therapy.<sup>7</sup> Histopathologic kidney findings of patients who died with COVID-19 have been most remarkable for acute tubular injury and are generally lacking classic viral nephropathy, diffuse thrombotic microangiopathy, or glomerulonephritis.<sup>8</sup>

As a reminder, nephritic syndrome

also presents with edema with or without proteinuria, but more often is associated with elevated blood pressure and decreased GFR. The most significant differentiation is that nephritic syndrome manifests with hematuria (either gross or microscopic) while nephrotic syndrome does not.

## TREATMENT

Hospital admission is necessary for severe edema, symptomatic fluid overload, infection, or thrombosis. Cases of mild to moderate edema can be discharged with outpatient management, including nephrology follow-up.

Mainstay treatment for nephrotic syndrome includes diuresis and a low-salt diet. Intravenous fluids should be administered, even if edema is severe, if the patient is in hypovolemic shock. Furosemide (1-2 mg/kg) is recommended for volume overload. Correct hypoalbuminemia if <2 g/dL with a dose of 0.5-1 g/kg. Finally, the need for immunosuppressive therapy depends on the etiology of the nephrotic syndrome. In pediatrics, 90% of NS cases are due to MCD. In these cases, a six-week course of prednisone (2 mg/kg a day taken BID or TID) should be administered. Despite the known association with thrombotic disease, prophylactic anticoagulation is not recommended unless the patient has a history of known thromboembolism. ★



*Announcing our...*

# EMRA Winter Awards Winners!

EMRA would like to extend a big and heartfelt CONGRATULATIONS to our 2023 Winter Awards Winners. These honorees exemplify excellence within the EM specialty.

*Chair of the Year*

**Lisa Clayton, DO**

Florida Atlantic University

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**Katey Osborne, MD**

Madigan Army Medical Center

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Saint Agnes Medical Center

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Dartmouth Hitchcock Medical Center

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**Adedamola Ogunniyi, MD**

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**Jennifer Williams**

University of Rochester

Department of Emergency Medicine

*ACEP/CORD Teaching Fellowship Scholarship*

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University of Pennsylvania

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Emory University School of Medicine

*Travel Scholarships*

*ACEP Scientific Assembly*

**Andrew Namep, MD**

Central Michigan University

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*Subcommittee Appointment*

**Sophia Gorgens, MD**

Zucker-Northwell NS/LIJ

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**Hope Taitt, MD**

Kings County Hospital Center

*Dr. Alexandra Greene Medical Student*

*of the Year Award*

**Josue Minaya**

Donald and Barbara Zucker

School of Medicine at Hofstra/Northwell

**Anthony Martella, MD**

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*SAEM Travel Scholarship*

**Ogechukwu Agatha Oforjebe, MD**

LAC+USC Medical Center

*Chief Resident(s) of the Year*

**Sarah Lee, MD**

**Milton Rahman, DO**

**Adil Husain, MD**

Staten Island University Hospital



#EMRAWinterAwards2022

*A Call for Nominations...*

# EMRA Summer Awards

Recognize excellence in emergency medicine by nominating yourself or colleagues for a coveted EMRA Award. Winners are selected for the following awards:

**CORD Academic Assembly Travel Scholarship**

**LAC Travel Scholarship**

**Augustine D'Orta Humanism Award**

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

**EMRA/ACEP Medical Student Elective in Health Policy**

**Stephen Tantama, MD, Military Excellence Award**

**EMRA/ACEP EDDA (Emergency Department Director's Academy) Travel Scholarship**

**EMRA/ACEP EMBRS (Emergency Medicine Basic Research Skills) Scholarship**

**Faculty Teaching Excellence Award**

**Faculty Mentor of the Year Award**

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

For application forms, nomination deadlines, and details about the EMRA Summer Awards, visit

[emra.org/awards](http://emra.org/awards)



**#EMRASummerAwards**

# Thyrotoxicosis: A Case of Storming Hormones

## Nhat Chau, MD

Resident, PGY-2  
Emergency Medicine  
Ochsner Health

## Anna Suessman, DO, MEd

Director of Education  
Dept. of Pediatric EM  
Ochsner Health

### CASE INTRODUCTION

A previously healthy 9-year-old female presented to the emergency department with a 10-day history of progressive left-eye bulging. The child and her family denied eye pain, vision changes, recent falls, injury to the eye, or insect bites. Her parents also denied any recent headache, blurry vision, photophobia, fever, cough, runny nose, vomiting, chest pain, palpitations, diarrhea, constipation, or rash. Mom reported that her child had weight loss and cold sensitivity in the past couple of months. She also reported that 10 days ago, she also noticed that

her child's neck looked larger. There were no known sick contacts, including COVID-19 exposures.

The patient's vital signs were: BP 120/76 mmHg; pulse 144 bpm; RR 24 breaths/min; temperature 36.8 C; and SpO<sub>2</sub> 97% on room air.

On physical examination, the patient was well-appearing; head normocephalic and atraumatic; left eye proptosis present, normal conjunctiva, EOM, PERRL, unremarkable fluorescein exam; neck with full range-of-motion; no palpable mass tachycardia without murmurs; lungs clear to auscultation.

Workup showed: Normal CBC, BMP; TSH low < 0.01; T<sub>3</sub> 336; Free T<sub>4</sub> 2.71.

Differential diagnoses: Graves' disease versus thyrotoxicosis, orbital cellulitis, Cushing's syndrome, orbital myositis, myasthenia gravis, or orbital tumors.

ED management:

- Ultrasound of soft tissue head, neck, and thyroid
- Propranolol 20 mg PO and methimazole 10 mg PO
- Pediatric ophthalmology and endocrinology consultation
- Admission for observation and further endocrinology workup

### DIAGNOSIS: THYROTOXICOSIS

Hyperthyroidism is defined as excessive thyroid hormone synthesis and secretion from the thyroid gland. The most common cause is Graves' disease, followed by toxic nodular goiter.<sup>1</sup>

Thyrotoxicosis is characterized by the clinical manifestations of excess circulating thyroid hormones, regardless of the source. In addition to Graves' disease, thyrotoxicosis often occurs in thyroiditis, iodine-induced and drug-induced thyroid dysfunction, and factitious ingestion of excess thyroid hormones.<sup>1</sup>

Thyroid storm is an acute life-threatening condition of hyperthyroidism, characterized by high fever, dehydration, tachycardia or tachyarrhythmias, tremor, nausea and vomiting, diarrhea,



restlessness, extreme agitation, or signs and symptoms of multiorgan failure, inclusive of heart failure, hepatomegaly, respiratory distress, abdominal pain, delirium or coma, and possible seizure.<sup>2</sup>

While thyroid storm is rare in the pediatric age group, the peak incidence is between 11 and 15 years of age. Females are five times more likely to have thyrotoxicosis than males. Graves' disease is the most common cause of thyrotoxicosis in the pediatric population. Risk factors include a history of Down syndrome, Turner syndrome, stress, high iodine intake, tobacco use, and family history of autoimmune thyroid disease.<sup>4</sup>

### SYMPTOMS AND SIGNS

Thyroid-associated ophthalmopathy is the main and most common extrathyroidal manifestation of Graves' disease. Proptosis or exophthalmos refers to the forward displacement of the globe due to an increase in retro-ocular fibroadipose tissue and swelling of extraocular muscles. Proptosis may be symmetric but is often asymmetric.<sup>3</sup> Complications include diplopia, strabismus, periorbital edema, conjunctival hyperemia and chemosis, corneal ulceration from lagophthalmos (incomplete eyelid closure), and optic nerve compression.

Cardiovascular manifestations include palpitations, widened pulse pressure, low peripheral vascular resistance, chest pain, dyspnea, cough, orthopnea, displaced apex, and murmur. Hyperthyroidism causes an increased cardiac output due to both increased peripheral oxygen demands and increased cardiac contractility. Atrial fibrillation occurs more frequently in older patients, but mitral valve prolapse is common in both adults and children.<sup>2</sup>

Gastrointestinal symptoms include failure to thrive and weight loss, even with an increase in appetite. Weight loss occurs due to increased calorogenesis, gut motility, hyper-defecation, and malabsorption.<sup>5</sup>

Behavioral symptoms include irritability, nervousness, anxiety, emotional lability, and fatigability.<sup>4</sup>



### MANAGEMENT

Multidisciplinary approaches involving care plan coordination between endocrinology, ophthalmology, orbital surgery, and radiology typically serve patients best.<sup>2</sup>

Beta-adrenergic blockers are recommended for all symptomatic patients before definitive treatments, especially those with tachycardia, tremor, and anxiety. Recommended dose of propranolol is 1 to 2 mg/kg/day divided into 3 to 4 doses, and atenolol 1 to 2 mg/kg once a day. Atenolol is preferred in reactive airway disease because of its cardioselective characteristic.

Definitive treatments range from antithyroid medications, to radioactive iodine, to surgery:

- Antithyroid medications are the initial treatment of choice in the pediatric age group. Methimazole 0.25 to 1 mg/kg/day daily or propylthiouracil (PTU) 5 to 10 mg/kg/day twice daily are commonly used. Methimazole is preferred in children because of its convenient once-daily dosing and its less adverse effect profile. PTU is preferred in pregnancy, especially during the first trimester,

because of teratogenic effects associated with methimazole.

- Radioactive iodine therapy is an option for children with adherence difficulties, children who fail medications after 1 to 2 years, and children with a major adverse reaction to medications. Absolute contraindications include pregnancy and lactation; relative contraindications include moderate to severe Graves' ophthalmopathy and children younger than 5 years of age.
- Surgery is second line, considered for children younger than 5 years who do not respond to or experience a major adverse reaction to oral medications. Surgery also should be considered in those with a very large goiter, severe ophthalmopathy, pregnancy, persistent hyperthyroidism despite use of oral medication and radioactive iodine, and personal preference.
- If thyroid storm is suspected, treat first with beta-blocker and propylthiouracil (preferred over methimazole), then add inorganic iodine and hydrocortisone 1 to 2 mg/kg IV every eight hours. It is important to fluid resuscitate and correct any deranged electrolytes.<sup>4</sup> ★



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# Emergent Diving Injuries: A Standardized Approach to Assessment

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This article aims to provide a standardized approach to assessing diving-related injuries. We'll first review the unique aspects of a history critical to diagnosing diving-related injuries. We'll then present two very general chief complaints and work through a list of differentials to consider, each associated with a specific phase of a dive.



by stage of the dive.

### **Descent**

The differential for a diver experiencing dizziness during descent is limited. A simple cause may be **caloric vertigo**, which occurs when cold water enters the ear unilaterally and stimulates the vestibular system. The temperature inequality may be caused by hoods or earplugs that alter the normal flow of cold water into the outer ear. This condition is transient and self-limited.

**Ear barotrauma** comes in two forms, the much more common **middle ear barotrauma** and the less common **inner ear barotrauma**. In both conditions, an inability to equalize the middle ear air space during descent leads to significant negative pressure in relation to the outside environment resulting in distortion and damage to tissues. The tympanic membrane (TM) is most commonly damaged. This damage initially presents as pain and bleeding within the layers of the TM and may lead to rupture. Divers can experience vertigo and hearing loss after TM rupture. Discontinuation of the dive and cessation of diving until the TM heals is recommended. The same mechanism causes inner ear barotrauma, but the damage is to the round or oval windows of the inner ear. This disruption can cause a leakage of endolymph from the inner ear. Symptoms include severe vertigo, nausea/vomiting, and hearing loss.<sup>2</sup> Referral to ENT is recommended for patients who sustain inner ear barotrauma. (See Middle Ear diagram, page 41.)

### **Bottom**

Dalton's Law of Partial Pressures states that the partial pressure of any gas is the product of the percentage of that gas in the mixture multiplied by the depth. At sea level, atmospheric pressure is 1 atmosphere (ata). Oxygen is 21% at sea level, therefore ppO<sub>2</sub> at sea level = 0.21 ata. In seawater, every 33 feet of depth adds an additional atmosphere of pressure. Thus, the partial pressure of oxygen in a standard air mixture at 66 feet is calculated below:

$$66 \text{ ft}/33 = 2 \text{ atm.}$$

$$2 \text{ atm} + 1 \text{ ata (1 ata @ sea level)} = 3 \text{ ata}$$

$$0.21 \text{ FiO}_2 \times 3 \text{ ata} = 0.63 \text{ ata ppO}_2$$

*Atm* represents the number of atmospheres measured over surface pressure. *Ata* presents absolute atmospheres, which includes the pressure from the atmosphere above our heads at sea level. This concept is important because the effects of any gas are directly related to its partial pressure.

The bottom phase of the dive presents a set of new differentials. We'll start with incorrect gas mixtures.

**Hypoxia** would likely begin to manifest a few minutes into a dive when the diver first reaches the bottom. Hypoxia can occur in rebreather diving, where exhaled air rich in CO<sub>2</sub> and low in O<sub>2</sub> is recycled within the diving rig using a chemical-based scrubbing system to remove the CO<sub>2</sub> and an oxygen source to add additional oxygen. Improper addition of oxygen to the gas mixture by the rig can rapidly lead to hypoxia. Hypoxia can also occur in self-contained underwater breathing apparatus (SCUBA) or surface-supplied diving when gasses are mixed incorrectly. In both SCUBA and surface-supplied diving, exhaled gasses are released from the rig and into the water as bubbles. Low percentages of oxygen are used when diving to extreme depths. For example, a 3% oxygen mixture could be used when diving to depths of around 200 feet. This mixture would lead to a ppO<sub>2</sub> of approximately .21 ata (.03 FiO<sub>2</sub> x 7 ata [198ft] = .21 ata O<sub>2</sub>), equal to surface air. However, using that gas mixture at a depth of 66ft would be deadly as the ppO<sub>2</sub> would only be .09 ata (.03 FiO<sub>2</sub> x 3 ata = .09 ata O<sub>2</sub>), essentially equivalent to breathing 9% oxygen at the surface. The primary mechanism driving respiration is carbon dioxide. Low oxygen alone does not provide a robust respiratory drive; thus, hypoxia commonly presents late as confusion, dizziness, or nausea. Treatment is simple: Provide more oxygen!

**Carbon monoxide (CO)** poisoning is also likely to manifest during this period. CO poisoning may be caused by gas bottles pressurized with a petroleum-based fuel-burning air compressor. If the air being compressed into a tank

is in close proximity to the exhaust of the compression system, CO will be pressurized into the air canister. Like all other gasses, the partial pressure of CO is multiplied by depth. CO poisoning commonly presents with headaches, nausea, vomiting, confusion, or lethargy. CO monoxide is treated with 100% oxygen, and hyperbaric therapy is indicated in patients with a CO > 25% or 20% if pregnant, loss of consciousness, severe metabolic acidosis (pH < 7.1), or evidence of end-organ ischemia.<sup>3</sup> A Navy Treatment Table 6 is recommended.<sup>1</sup>

As depth surpasses 100 ft, or more specifically, as the partial pressure of nitrogen exceeds 4 ata, the risk of **nitrogen narcosis** increases. Nitrogen at high partial pressures has a narcotic effect, simulating drug or alcohol intoxication. Divers expecting to dive to significant depths generally use helium instead of oxygen, as helium does not have the same narcotic effects as nitrogen. Nitrogen narcosis manifests as giddiness, poor decision-making, loss of skill, tingling, inappropriate laughter, and lack of concern for safety. Nitrogen narcosis in itself is not dangerous, but divers can quickly become tangled or lose their air source in their confusion. There are sea stories of divers cutting their own air lines due to the narcotic effect of nitrogen. Treatment is simple: Ascent to a shallower depth rapidly corrects the problem with no sequelae.

**CNS oxygen toxicity** is another condition with which risk increases with increasing depth. High partial pressures of oxygen have a toxic effect on the central nervous system. CNS oxygen toxicity occurs at partial pressures of Oxygen over 1.3 ata (USN) -1.4 ata (PADI).<sup>4</sup> For a diver breathing air, oxygen toxicity would not be a risk until a diver reached 187 ft. However, divers breathing NITROX or another form of mixed gas, oxygen toxicity can occur at much shallower depths. A commonly used NITROX mixture in recreational diving is 32% oxygen. With this mixture, a diver would reach a ppO<sub>2</sub> of 1.4 ata at 111ft. Oxygen toxicity presents as a group of possible symptoms known as VENTID-C. This acronym stands for visual symptoms (tunnel vision or



blurry vision), ear symptoms (tinnitus), nausea, vomiting, twitching and tingling, irritability, dizziness, and convulsions.<sup>1</sup> O<sub>2</sub> toxicity is easily treated by ascent to a shallower depth, although aborting the dive is recommended. The most significant risk with CNS O<sub>2</sub> toxicity is convulsions, which could quickly lead to drowning.

**Ascent/Post Dive**

**Decompression sickness** (DCS), also known as “the bends,” and **arterial gas embolisms** (AGE) are the most common injuries during ascent or after a dive. We’ll cover **arterial gas embolism** first as it is more likely to occur chronologically before DCS. An AGE occurs due to overexpansion of gas in the lungs, a result of Boyle’s Law. Boyle’s law states that the pressure and

volume of a gas are inversely related. As a diver ascends to the surface, air-filled structures throughout the body will expand (lungs, sinuses, middle ear, dental cavities). Rapid ascent with a closed glottis does not allow expanding air in the lungs to escape. This air must go somewhere, which can result in mediastinal emphysema, pneumothorax, or subcutaneous emphysema, listed in descending order of likelihood. These injuries are collectively known as pulmonary overinflation syndrome (POIS). This damage can also result in air bubbles directly entering the arterial system, leading to deadly consequences. These bubbles rise against gravity through the arterial vascular system to the most superior portion of the body, the brain. Once these gas emboli lodge in smaller arterioles, they cause

damage similar to thrombotic clots, preventing blood flow and damaging endothelium. AGEs present within the **first 10 minutes** of surfacing from a dive and are an immediate emergency. AGEs present with a wide range of symptoms related to the location of the brain in which they damage and can present as severely as immediate unconsciousness upon surfacing. When symptoms correlate with both AGE and DCS, if they develop within ten minutes of ascent, **they are treated as AGE until proven otherwise.** Treatment is recompression with Navy Treatment Table 6.<sup>1</sup>

**Decompression sickness** results from the rules of Henry’s Law. This law states that the amount of dissolved gas in a liquid is proportional to its partial pressure above the liquid. In diving,

the human body can be substituted for the liquid mentioned above. As a diver descends deeper, more of whatever gas mixture they breathe will dissolve in their tissues. This becomes more of a problem on ascent, as the diver's tissues have a saturation point directly related to depth. If a diver ascends too quickly after a long dive, the amount of gas dissolved in their tissues becomes greater than the maximum saturation point, and the gas bubbles out of solution. DCS is further categorized into two types, DCS Type I, in which involvement is limited to a single joint, the skin, or the lymph system, and DCS Type II, which can involve the nervous system, inner ear, or pulmonary system. Gas bubbles formed in DCS are generally caused by helium or nitrogen since these gasses are inert and cannot be metabolized. The body can metabolize and break down oxygen, so it is less likely to cause issues, even if it exceeds its saturation point.

DCS can cause neurologic symptoms through several different mechanisms. Direct or "de novo" formation of bubbles within the tissues of the brain or spinal cord will cause local damage to these organs. Symptoms will be directly related to the structure in which the bubbles are found. Neurologic DCS has a predilection for the spinal cord, leading to paresthesias, paralysis, incontinence, or any number of peripheral neurologic complaints.<sup>5</sup>

Bubbles from decompression sickness can also occur in the venous system and are known as venous gas emboli or VGEs. VGEs can cross through cardiac or pulmonary shunts to the arterial system. At this point, they become AGEs and can have all of the effects of previously discussed AGEs. However, the term AGE in diving medicine is almost always used to describe AGEs caused by pulmonary overinflation. MRI can locate ischemia and edema from DCS in both the brain and spinal cord, but obtaining advanced imaging should not delay treatment.

The final neurologic possibility resulting from DCS is that of inner ear DCS. This is a relatively rare cause of DCS, more likely to be seen in saturation

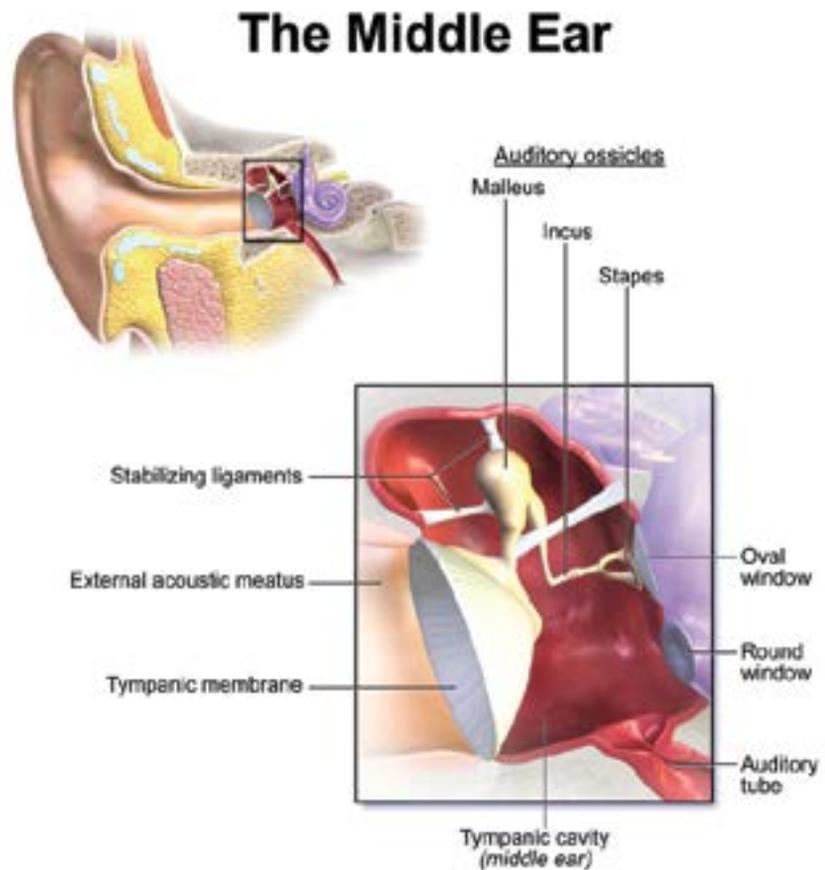
divers breathing helium. In inner ear DCS, bubbles form de novo in the inner ear structures, leading to dizziness similar to that of an otolith. Of course, immediate differentiation between inner ear DCS and central intracranial DCS is challenging. Still, EM physicians should utilize the same strategy for differentiating central versus peripheral vertigo symptoms in non-diving patients, with a diving history leading to consideration of inner ear DCS as an additional peripheral cause.

All patients with neurologic, pulmonary, or inner ear DCS should be treated with a Navy Treatment Table 6,<sup>1</sup> with neurology follow-up. For both AGE and DCS, supplemental oxygen should be initiated as soon as possible if a recompression chamber is not immediately available. (See Treatment Table 6, page 42.)

It is important to note that while we typically associate the bubbles of DCS

with occlusive mechanisms associated with the presentation of an ischemic cerebrovascular accident, DCS symptoms do not always localize to a specific neurologic lesion or territorial infarct. Newer research is beginning to shed light on the multifaceted etiology of DCS, with bubble vascular occlusion not being the only element of the complex presentation.<sup>6,7</sup>

**Alternobaric vertigo** can cause vertigo or dizziness during ascent, although it can also occur during descent. The mechanism involved is an imbalance in pressures between the middle ear spaces. On ascent, the middle ear pressures increase, and the eustachian tube releases this excess gas into the nasopharynx. A pressure imbalance is created if one of these tubes is blocked. Treatment is to attempt the diver's preferred equalization strategy and ascend slowly.



Ear Anatomy, Middle Ear<sup>10</sup> (From Wikimedia Commons, the free media repository. File: Blausen 0330 EarAnatomy MiddleEar. See emresident.org for full citation.)

### Treatment Table 6

1. Descent rate - 20 ft/min.
2. Ascent rate - Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption (see paragraph 17-8.10.1.1).
5. Table 6 can be lengthened up to 2 additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to 2 additional 75-minute periods at 30 feet (15 minutes on air and 60 minutes on oxygen), or both.
6. Tender breathes 100 percent O<sub>2</sub> during the last 30 min. at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 feet. If there has been more than one extension, the O<sub>2</sub> breathing at 30 feet is increased to 60 minutes. If the tender had a hyperbaric exposure within the past 18 hours an additional 60-minute O<sub>2</sub> period is taken at 30 feet.

### Treatment Table 6 Depth/Time Profile

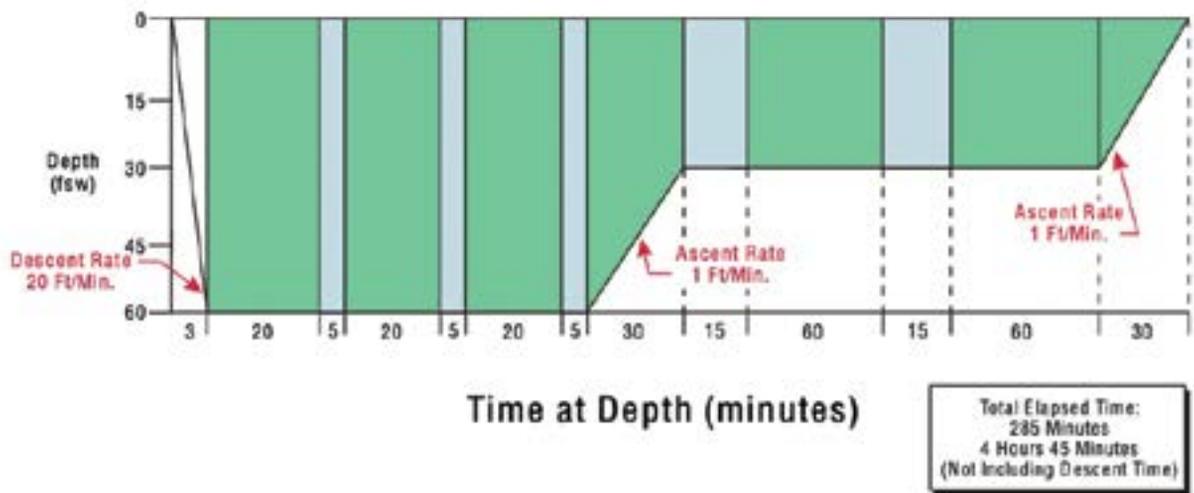


Figure 17-5. Treatment Table 6.

Treatment Table 6' (See emresident.org for full citation.)

**CASE 2**  
**CARDIOPULMONARY PRESENTATION:**  
**SHORTNESS OF BREATH**

The next diving-related chief complaint that emergency physicians should be familiar with is shortness of breath and/or chest pain.

**Descent/Bottom**

A diver experiencing shortness of breath early in a dive may be suffering from **hypercapnia**. As discussed earlier, CO<sub>2</sub>

is the primary driver of respiration, and elevated levels of CO<sub>2</sub> will consistently result in an increased respiratory rate and feelings of dyspnea. Hypercapnia is less likely to occur with SCUBA divers, as it would be challenging to contaminate a SCUBA bottle with high levels of CO<sub>2</sub>. It can happen in rebreather diving if the rig is not functioning correctly or if the CO<sub>2</sub> absorbent is no longer pulling CO<sub>2</sub> from the system as it should. Hypercapnia can also occur when diving with full

face mask or hard hat systems, as these devices include areas of stagnant air or dead spots, which allow for the build-up of CO<sub>2</sub> in a localized area. This problem can easily be remedied by venting the space with fresh gas. Elevated CO<sub>2</sub> also increases the risk of CNS oxygen toxicity, has an additive effect with nitrogen narcosis, and may increase DCS risk.

Diving with a rebreather poses additional risks as the chemicals used to scrub CO<sub>2</sub> are highly toxic when they

come into contact with water. Ingestion of these caustic alkaline chemicals, known as a **caustic cocktail**, can lead to severe damage to the airway. Symptoms include choking, gagging, foul taste, and burning. Initial management should include a fresh water rinse of the mouth. These patients may require intubation, decontamination, bronchoscopy, and endoscopy.

### **Ascent/Post Dive**

As discussed in the section on AGEs, overexpansion of air in the lungs forces gas into nearby structures. Thus, **pneumothorax (PTX)** should be considered in any patient with chest pain or SOB. If a concurrent diving injury requiring recompression is present, a PTX should be addressed before the patient enters the recompression chamber. There is little risk with recompression, but during ascent in the chamber at the end of treatment, the air in pleural space will begin to expand again, causing further damage. Standard

treatment for a PTX is appropriate to include needle decompression, chest tube, and inspired oxygen.

**Pulmonary decompression sickness** is colloquially referred to as “the chokes” and is the final form of Type II or emergent DCS we will discuss today. In the prior section, we discussed VGEs, which form de novo in the venous system in patients with DCS. In patients without a cardiopulmonary shunt, most of these gas emboli are filtered out of the vascular system by the lungs. Pulmonary DCS can occur in patients with an overwhelming burden of VGEs. Initial symptoms are cough, shortness of breath, burning substernal chest pain, pain with respirations, and increased respiratory rate. Radiographic findings are similar to that of pulmonary edema. Treatment is inspired oxygen, and a Navy Treatment Table 6.<sup>1</sup>

### **CONCLUSION**

Although most diving-related injuries are not life-threatening, it is imperative

to promptly establish contact with your on-call hyperbaric physician when a serious diving-related injury is identified. Your hyperbaric physician will guide you in your approach to treatment and may help you with the transportation of your patient to the nearest recompression chamber. If you do not have a readily accessible hyperbaric specialist, the Divers Alert Network (DAN) has a 24/7 hotline which you can contact at **+1 (919) 684-9111**. This number will connect you to a DAN medic who will further advise and help you locate the nearest recompression chamber.<sup>8</sup>

Finally, while we discussed in depth the differential diagnosis of diving injuries, divers also suffer from the same injury patterns as the general population. A diver who has multiple cardiac risk factors and presents with chest pain should be evaluated for acute coronary syndrome the same as every other patient with chest pain, with the diving injury differentials being considered in addition. ★



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# The Ethics of Caring for an Unfavorable Patient

Adiba Matin, MD  
Mayo Clinic

**In light of the recent uptick in gun violence and the controversy regarding legislation, we must be prepared to care for victims of mass casualty scenarios — but also for the alleged culprit of the crime. In many of these attacks, the shooter survives and is treated in an emergency department following these heinous incidents.**

**A**s a new senior resident, I imagine myself in the position of the emergency physicians who treat alleged attackers. These physicians have to grapple with so many things

simultaneously: maintaining composure, keeping the environment calm, dealing with concerns for their own personal safety, and managing the mountain of visceral, natural biases against the

culprit, all while attempting to provide the alleged attacker with the same care provided to all other patients.

As providers, this is just one example of the many situations we face in the





emergency department when our patients challenge us. We are inevitably put in situations where emotions of frustration, anger, anxiety, dread, and guilt are invoked — whether it be a rude patient, an ungrateful one, a demanding one, a racist one, or possibly one who tries to dismantle the sanctity of our society.

What is expected of us in these circumstances? The implications of the bioethical principle of justice means that we should strive to be fair and equal to all people.<sup>1</sup> But in these difficult circumstances, is it possible to abstain from making moral judgments on patients?

Social scientists have found that moral judgment of patients is pervasive. It occurs not only when taking care of a patient who allegedly committed a criminal act, but also during daily scenarios when the appraisal of a patient's social worth and culpability are routine. There is limited literature, however, on the impact of moral judgment in healthcare. Evidence suggests that moral appraisals are the result of a multiplex of variations in patient characteristics, physician characteristics, situational factors, and organizational factors.<sup>2</sup>

Based on research, it is extremely

difficult to completely eliminate all biases — both implicit and explicit — from daily life, let alone patient care. But this is not to say you must treat every unfavorable patient. An article published in the *New England Journal of Medicine* provides a flowchart to help assess whether it is appropriate for patients to be reassigned per their request.<sup>3</sup> The article recommends evaluating for stability first. If the patient is unstable, you must treat to the best of your ability. If the patient is stable, then it is possible that you can ask more questions about why the patient would like a reassignment. If the rationale may be rooted in bigotry, then one might explore options for the patient and recognize the impact this may have on the physician. At that point, it may be appropriate to negotiate, offer transfer, accommodate, or limit unacceptable conduct.

In terms of the physician's requests, ethically, it is clear that when biases and conflict interfere with providing neutral care to the patient, and the patient is stable, then it may be appropriate to defer care to another party. With alleged shooters, the Emergency Medical Treatment and Labor Act (EMTALA) protects their care, ensuring public access to emergency services.<sup>4</sup> When the option of transfer is not available,

particularly in emergent situations, the circumstances are tough. However, there are steps that can be taken to ensure that tough situations are handled in an ethical manner. An important step is acknowledging the bias. This is difficult. It requires honesty and practice.

Although more literature in this space is necessary, research by *The Journal of Philosophy, Ethics, and Humanities in Medicine* shows that “cognitive reappraisal is shown to have far healthier personal and interpersonal consequences than emotion suppression strategies.”<sup>2</sup> This means that understanding the bias or negative reaction is important, but also shifting the focus to something else, like the professional commitment or the ethical standard that is required for a physician, can help make an otherwise dreadful situation more bearable.

Debriefing, leaning on peers, and seeking mental health help are all important steps in caring for unfavorable patients. Taking a pause to acknowledge the emotional toll that this may take on you, approaching the situation in a systematic format, and defining your role as a physician may help mitigate the psychological impact on the provider and ultimately promote ethical care. ★

# Not Just a URI: Serious Complications With Sinusitis

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### CASE PRESENTATION

A 7-year-old male with a history of eczema presented to the emergency department with persistent fever, cough, and nasal congestion of 2 weeks' duration that had worsened over the last three days. An initial ED visit had occurred 10 days earlier for congestion, cough, and fever resulting in a diagnosis of viral syndrome. During the second ED evaluation, the patient's symptoms improved with administration of IV fluids and antipyretic. A chest X-ray, labs, and nasopharyngeal Covid-19 PCR test were unremarkable. No antibiotics were prescribed.

The patient returned to the ED the following day with fatigue, persistent high fever, and orbital swelling. Initial vital signs included BP 111/61, pulse

154, temp 39.3 °C (102.7 °F), resp 22, and SpO<sub>2</sub> 97%. Physical exam revealed left orbital swelling, tenderness to palpation, yellow/green nasal discharge, dry oral mucous membranes, clear breath sounds, and a soft and nontender abdomen. Extraocular movements were documented to be intact with normal conjunctiva. The initial differential diagnosis included sepsis with bacterial sinusitis with osteal, orbital, or intracranial involvement including abscess.

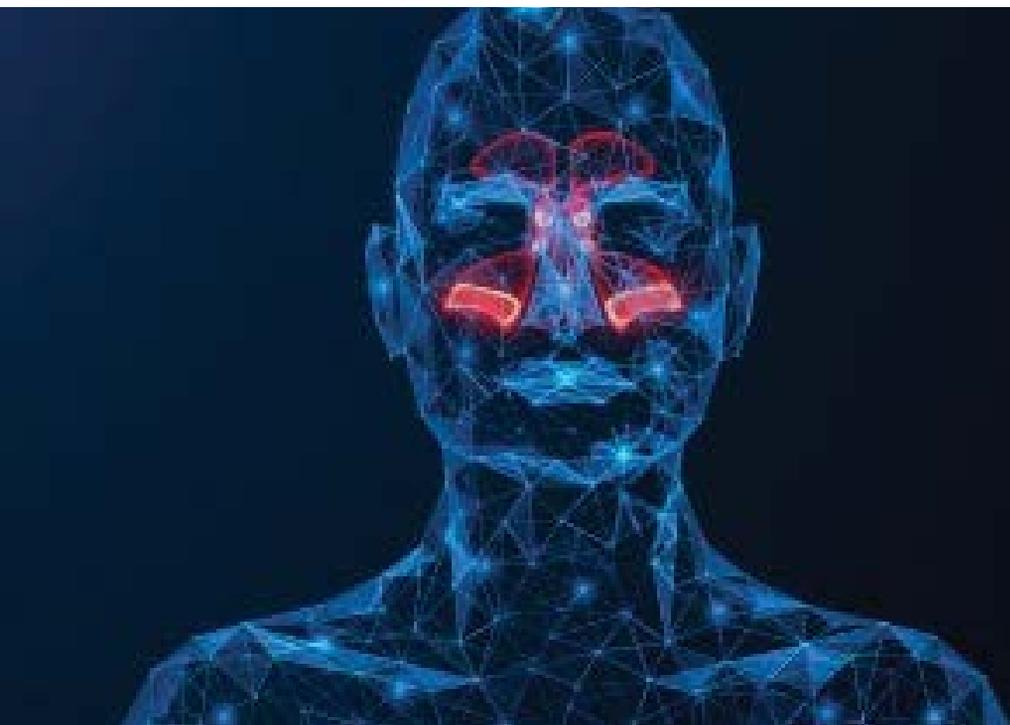
A CT scan revealed pansinusitis including opacification of the frontal, right and left maxillary, and right ethmoid sinuses. In addition, left superior ophthalmic vein thrombosis, sagittal sinus septic thrombosis, and orbital cellulitis were noted. The patient

received broad spectrum IV antibiotics including ceftriaxone, vancomycin, and metronidazole. He was admitted to the PICU for further workup and management.

During PICU admission, the patient had an MRI/MRV that showed subdural empyema and superior ophthalmic vein thrombosis. He received an emergent craniotomy with subdural effusion evacuation. Operative cultures grew *methicillin-resistant Staphylococcus aureus*, *coagulase-negative Staphylococcus species*, *Streptococcus constellatus*, and *Prevotella Intermedia*. A PICC line was placed for provision of long-term broad spectrum antibiotics. Ultimately, the patient developed subdural empyema of the right hemispheric and required bifrontal craniotomy drainage. After 6 weeks in the PICU, his clinical status improved, and he was discharged on a further 4-week outpatient course of linezolid, levofloxacin, and metronidazole.

### DISCUSSION

This case demonstrates the serious complications that can accompany sinusitis, including orbital cellulitis and septic thromboembolism with intracranial abscess. Rhinosinusitis, defined as inflammation of the mucosal lining of the nasopharynx and sinuses, is most commonly caused by a viral infection. Acute bacterial rhinosinusitis (ABRS) most commonly occurs when there is a secondary infection of the sinuses. It is most common in ages 4-7 years but can occur at any age. Patients will present with symptoms consistent with upper respiratory illness (URI) for a protracted duration, usually for more than 10 but fewer than 30 days. There





can also be a biphasic presentation, such as in this patient, in which symptoms improve for a period of time but then exacerbate and the patient's condition acutely worsens.<sup>1-4</sup>

When evaluating a patient with suspected bacterial rhinosinusitis, it is not always necessary to obtain radiologic imaging. If symptoms have lasted fewer than 7 days, then supportive care with nasal wash, decongestants, and intranasal corticosteroids can be trialed. For sinusitis lasting more than 7 days, high-dose amoxicillin with clavulanate can be prescribed.<sup>2,5</sup> Failure to improve within 72 hours should prompt further investigation, with consideration of performing advanced imaging and possible escalation to parenteral antibiotic therapy. CT scans of sinuses and the brain should be obtained if there are concerning symptoms suggesting disease extension, such as periorbital/orbital swelling, vomiting, altered mental

status, neck stiffness, neurological deficit, or high fevers lasting for more than 3 days.<sup>1,6</sup>

It is estimated that anywhere from 0.5% to 24% of children are hospitalized with sinusitis, likely because of the risk of severe complications ranging from orbital cellulitis to intracranial infection.<sup>7</sup> The cavernous sinus collects blood from various sites including the face, ears, nose, pharynx, sinuses, and cerebral cortex. The valveless flow of blood within the cavernous sinus increases the risk for spread of infection as the blood can flow in any direction. The sinuses are particularly vulnerable to the spread of infection and formation of septic thrombosis because of this blood flow.

Approximately 5% of children hospitalized with ABRs develop intracranial complications such as septic dural sinus thrombosis, subdural empyema, epidural abscess, meningitis, cerebritis, brain abscess, or cavernous

sinus thrombosis.<sup>8,9</sup> In a retrospective study of hospitalized children with intracranial complications of ABRs, 89% had visited a health care provider prior to their admission, but only half were prescribed antibiotics.<sup>7</sup> Of those prescribed antibiotics, 44% had been receiving antibiotics for more than 7 days before developing intracranial complications — signifying the potential complexity of treating the infection with oral antibiotics on an outpatient basis.<sup>7</sup>

Despite the severity of the intracranial complications of sinusitis (ICS), most children make a good neurologic recovery. In a sample of 25 children with ICS, 40% experienced neurologic deficits, but most resolved within 2 months. Only 2 patients had permanent neurologic sequelae.<sup>8</sup>

At a 6-month follow-up exam with neurosurgery, the young boy in our case was noted to have made a full recovery without any deficits. ★

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(10 Days Prior to the Representative Council Meeting)

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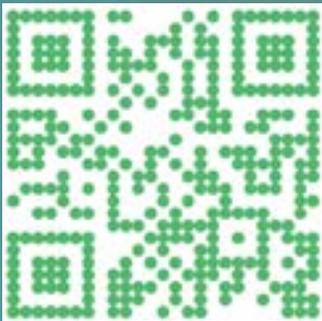
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# VF Arrest: Prolonged CPR in an Electrical Storm

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## A CASE REPORT

A 44-year-old man presented to the emergency department with chest pain for 3 days and collapsed while undergoing triage. The patient was found to be pulseless; he was immediately placed on a stretcher, ACLS was initiated, and he was wheeled into the resuscitation room with compressions in progress. The patient was placed on the monitor, with end-tidal CO<sub>2</sub> measuring at 15mmHg. He was given 1 dose of epinephrine, and at the initial pulse check, was found to be in

pulseless ventricular fibrillation (VF).

The patient was defibrillated at 200 joules (J) and placed on a mechanical CPR machine. During mechanical compressions, the patient woke up, stated he was okay, and started fighting with staff and the machine. Staff paused for pulse check and the patient again became unresponsive, was in VFib, and ACLS was continued. The patient again returned fighting staff during compressions and was unable to be verbally de-escalated. The patient was

given 150mg ketamine intravenous for sedation while preparing for intubation. He was defibrillated 3 more times prior to obtaining return of spontaneous circulation (ROSC). During this time, the patient was placed on dual pads for dual defibrillation, and advanced cardiovascular life support (ACLS) was continued.

After the patient was intubated successfully with rocuronium and etomidate, ketamine was started for post-intubation sedation. The patient

was dual-defibrillated 3 more times until initial ROSC was obtained. At that time cardiology was at bedside, an ECG demonstrated STEMI in V1-V3, and the decision was made to transfer to a tertiary site with STEMI lab, cardiothoracic surgery, and extracorporeal membrane oxygenation (ECMO).

Minutes after acceptance to a tertiary care center, the patient again went into pulseless VF, and ACLS was reinitiated. ROSC was again obtained after a total of 75 minutes and included 15 defibrillations (at least 5 dual defibrillators) and a total of 13 mg epinephrine, 8mg magnesium, 450 mg

of heart failure or cardiovascular event. The definitive treatment for VF is defibrillation at 120-200 J, many times requiring dual defibrillators, as it has a higher success rate.<sup>4</sup> VF is usually a short-lived arrhythmia and results in SCD, unless met with chest compressions within 3 minutes of arrest to maintain myocardial perfusion. Mechanical compression devices have shown better survival rates in prolonged resuscitations, but worse survival when thrombolytics were used.<sup>5</sup> A study from Milwaukee in 1983 demonstrated a 46% survival rate to discharge in VFib arrest patients with a mean time of resuscitation of 12.6 minutes.<sup>6</sup> Reports

Impella, ECMO, intra-aortic balloon pump, Centrimag, and TandemHeart. Each device differs in its capabilities, time to placement, time allowed to be in place, and complications profile.

#### IMPORTANCE OF AWARENESS

VF arrests can occur after days of symptoms and proceed into a ventricular storm. This may require prolonged resuscitation with defibrillators, the definitive therapy of VF. Early dual sequence defibrillation has been shown to increase outcomes, while treating other underlying causes. These patients may require intubation for further sedation, if

**VF arrests can occur after days of symptoms and can proceed into a ventricular storm. This may require prolonged resuscitation with defibrillators, the definitive therapy of VF. Early dual sequence defibrillation has been shown to increase outcomes while treating other underlying causes.**

amiodarone, 200mg lidocaine HCl, 1g calcium chloride, and 3 amps of sodium bicarbonate.

The patient was transferred to a tertiary care site via ambulance along with two senior residents and an attending physician on board in case there was a need for prolonged CPR (due to the distance of the facility and unpredictable traffic of New York City). No interventions were required en route. The patient was taken immediately to the catheterization lab, impella placed, and was found to have a 100% occlusion of the left anterior descending artery that responded to mechanical thrombectomy and stenting. The patient had post-perfusion runs of VFib and defibrillated successfully. At this time, the patient was hemodynamically stable on vasopressors and did not require ECMO. That evening, the patient was weaned from sedation and was able to follow commands in his native language and acknowledge his family members in his room.

#### DISCUSSION

Ventricular fibrillation is the most common cause of sudden cardiac death (SCD), usually preceded by 48 to 72 hours of symptoms prior to occurrence.<sup>3</sup> VF is a manifestation of ischemia, thus more likely in patients with a history

demonstrate resuscitations can be successful after multiple hours, and even days. One case report demonstrated a patient was able to sustain shock-resistant VF for 10 days, as he had cardiac perfusion via LVAD.<sup>7</sup>

Once the patient has been resuscitated to sinus rhythm, reperfusion therapy must occur to maintain myocardial perfusion. If cardiogenic shock is present, an assistive device must be placed. Several devices exist to aid in circulatory support and allow the patient time to recover to have further intervention. These options include

this occurs, propofol will help reduce the sympathetic storm. Patients that regain ROSC after VF arrest may need emergent cardiac intervention due to its high association with ischemia. If your facility does not have catheterization abilities, it may require transfer to a tertiary care center to include interventional cardiology, cardiothoracic surgery, and ECMO capabilities. Be considerate in placement of IV lines using mostly large bore peripherals, as large veins and arteries should be saved for cardiac intervention including impella, ECMO, and stenting. ★





# Mystery Bruising: A Case of Post-Viral ITP

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### CASE INTRODUCTION

A 7-year-old male with no past significant medical history presented to the emergency department, accompanied by his mother, for evaluation of bruising to the lower extremities that had been observed for the past week. The mother reported she was initially not concerned about the bruising because the patient had started karate lessons. However, the patient reported to mom that he was not getting kicked in the legs.

A few days prior to presentation, the patient developed a large bruise to the right abdomen just above the hip. All of these bruises reportedly occurred without any trauma. This was when he was having a cough and rhinorrhea, which mom thought may have been allergies. The patient was reported to have an occasional nosebleed about once

every other month, including one just in the previous week. Both the patient and mom denied fever, sore throat, myalgias, arthralgias, night sweats, weight loss, hemoptysis, hematuria, melena or hematochezia, bleeding gums, abdominal pain, nausea, vomiting, chest pain, shortness of breath, abnormal breath sounds, ear pain or discharge, foamy or frothy urine, changes in color of urine, changes in frequency of urination, rashes, or family history of bruising disorders. There was no recent antibiotic use or travel, and no recent known tick, mosquito, or other parasitic exposure. No new foods had been introduced to the patient's diet. No new pets or chemical exposures were reported.

Nursing reported that petechiae appeared at the site of the blood pressure (BP) cuff after the BP was obtained.

The patient's vital signs were stable. There were no labs drawn at the time of the initial evaluation.

A physical exam revealed a patient who appeared in no acute distress. He was attentive and interactive. Heart sounds were normal rate and regular without murmurs. Lungs were clear to auscultation with good chest expansion. The abdomen was soft and nontender. The spleen was not enlarged. There was no edema. The eyes had normal conjunctiva bilaterally. There were 6 small petechiae noted within the oropharynx. The patient had small areas of petechiae noted on bilateral anterior upper arms. Scattered petechiae were noted across the upper chest anteriorly. There was a large area of ecchymosis on the lower right side of the abdomen. He had innumerable ecchymotic lesions over

the bilateral lower extremities, especially below the knees and anteriorly. There were no rashes on the palms or soles.

## DISCUSSION

In this case, what would be your initial workup? The differential here should include malignancies such as myelodysplastic syndromes, leukemias, lymphomas; infections such as post-viral, HIV, Hepatitis C; drug-induced such as many antibiotics, seizure medications; and primary hematologic processes such as thrombotic thrombocytopenic purpura and disseminated intravascular coagulation. In this case, post-viral platelet disruption was thought to be the leading diagnosis.

Initial workup should include CBC, reticulocyte count, blood smear, fibrinogen, Von willebrand factor (VWF) activity, and a coagulation panel. In this case, the reticulocyte count was not ordered.

Our patient's labs are shown (**Table 1**). A smear was not readily available but would typically be normal with variable platelet size, large platelets, and some giant platelets.

A diagnosis of virus-associated idiopathic thrombocytopenic purpura (ITP) was made.

The incidence of post-viral ITP is about 1 per 20,000 children a year. Two-thirds of children who develop ITP have had a recent viral illness. Occasionally, a virus such as EBV, varicella, or HIV (testing is not usually indicated) can be detected. The mechanism is not fully understood but typically results from increased platelet destruction and/or decreased production. A majority of children only develop minor bleeding, such as our patient with scattered ecchymosis, petechiae, and mucosal bleeding.

Treatment will usually be outpatient. The American Society of Hematology (ASH) suggests any patient with no or only minor bleeding be observed, plus/minus steroids or 2nd line medications, with hematology follow-up within 48 hours. This differs from the adult disposition. Any significant bleeding, of course, requires immediate inpatient

WBC	5.62
RBC	4.03 L
HGB	11.5
HCT	31.7 L
MCV	78.7
MCH	28.5
MCHC	36.3 H
PLT	1 LLL
RDW	12.3
Fibrinogen	180 L
PT	11.1
INR	1.0
COVID	Not detected
VWF activity	Send out

TABLE 1

treatment.

In patients with no or minor bleeding, ASH recommends observation over steroids or IVIG. If a decision is made to give steroids, ASH suggests a course of 2-4 mg/kg/day for 5 to 7 days. If there is a contraindication to steroids, then IVIG 1 g/kg x 1 can be given with the possibility of repeating dosing once. If there is severe, life-threatening bleeding, then IVIG with above dosing is indicated. In the event that symptoms persist for longer than 3 months, second-line agents — including thrombopoietin receptor agonists, rituximab, and/or splenectomy — may be indicated. All patients and parents should be given strict precautions to not participate in contact sports, collision activities, trampoline use, or any activity where there is higher risk for developing bleeding, especially intracranial bleeds. Children prone to falls and trauma, such as toddlers, may require treatment since they may not be able to follow these precautions.

## CASE RESOLUTION

Our patient had non-severe bleeding and was discharged from the ED after a discussion with pediatric hematology. Guidance was given to the patient and



Bruising, lower extremities

mother to avoid activities that could lead to injury. Return precautions — including epistaxis, hematemesis, hemoptysis, hematochezia, and neurologic changes suggesting intracranial bleeding — were discussed. The patient followed up with hematology the next day, and a decision was made to administer a short course of oral steroids. ★



# HOW TO CARE

In my final year of residency, I am struck with an urgency to cram as much learning as possible into the short time remaining. I'm sure that senior residents across the country are doing the same. I have also taken a few steps into the role of teacher, offering what few pearls of wisdom I can to the new interns familiarizing themselves with the department. It is during these

moments that I can truly appreciate the breadth of what I've learned in residency. The realization comes as a refreshing break, however short, from the anxiety and uncertainty that inevitably hits when I step back into the chaos.

There was a time when I was seeing all my patients in rooms. Now I see more patients in chairs in the waiting room than in beds, while all around me

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there are staffing shortages, excessive bed holds, wave after wave of Covid, and increasingly grumpy and occasionally violent patients and family members.

I've learned a lot in residency, even from "waiting room medicine" — it teaches prioritization and self-sufficiency, if nothing else. I have learned to care for all sorts of patients with all sorts of conditions.

But the new, seemingly endless shift in the landscape of emergency medicine and the health care system overall has highlighted that while I can care for, residency has failed to teach me how to care about.

How do I care about the patient who comes in with the same neck pain she has had for the past 15 years, who only came in tonight because she “had some time,” when I just left a family in a back room with a chaplain, coming to terms with the loss of their loved one?

How do I care about the unvaccinated patient who has a fever but refuses a Covid test when I know that upstairs there are still other Covid-positive patients dying on ventilators?

How do I care about the consultant who berates me for calling them to see a patient still in the waiting room, when people are dying waiting for a room upstairs?

How do I care about the next patient I need to see, when I feel like I have used up all my “caring” on the man whose wife died the day before their 60th wedding anniversary, the woman whose son died of a drug overdose, the woman who withdrew care for her father after four hours of resuscitation?

How do I care about doing my best, when even at my best I am told by the patient’s family that my hospital should be shut down, that we should be reported, that I should be ashamed of how long it takes to be seen, to get a room, for labs to result, for papers to discharge?

How do I care about my future, when I hear that it will be difficult to find a job in just a few years, when my attendings cry in the hallways, when I hear all the reasons that wellness is pivotal and burnout is a critical finding, but see no evidence of wellness in my studies?

How do I care about my husband, who has supported me through my entire career so far, when I’ve heard people talking all day and just want to sit in silence?

How do I care about myself?

When do I schedule a doctor/dentist/therapy appointment? Do I pick up my medication from the pharmacy and be late to my shift, or do I withdraw from my SSRI on my string of nights? Do I sleep for an extra hour, or go to the gym? Socialize with friends, or clean my house?

How do I even care about caring? And how do I know when to stop? How do I reset to be able to care tomorrow when I go home and still think of all the people I cared about today?

I will graduate soon, and I am fully confident that I will graduate as a competent physician, able to manage a breadth of clinical presentations. I will know what to do, or I will know how to figure it out. Rather, I fear that the biggest failure of my training will be what I should do after the patients leave my care: when I go to the next room, to the next shift.

This is not a criticism of where I’ve trained; I love my residency and my co-residents, and I have endless respect and admiration for my mentors and our faculty. But I worry that there is no evidence-based answer for my dilemma,

and that this is a deficit in residency training overall. As I read and hear about other physicians in our specialty working through the same emotional burnout, I think that there must be something more we as a specialty can do for the next group of new physicians. Built-in “wellness time” and recommending meditation are small steps, but we can only meditate on the state of the system for so long before the weight of carrying the safety net drags us down. Dealing with the emotional and mental repercussions of our work should be integrated as part of our training earlier in residency, to give us the skills to answer these questions both during our training and long after we have left.

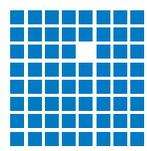
I don’t have the answer. I have no panacea for this pandemic of burnout. I worry that even my admirable mentors don’t have the answers or know the appropriate remedy. I am still trying to pluck out some pearls of wisdom from all of this to pass along to a new intern or medical student. But I hope that by the time someone looks up to me for guidance, I will have found it. ★



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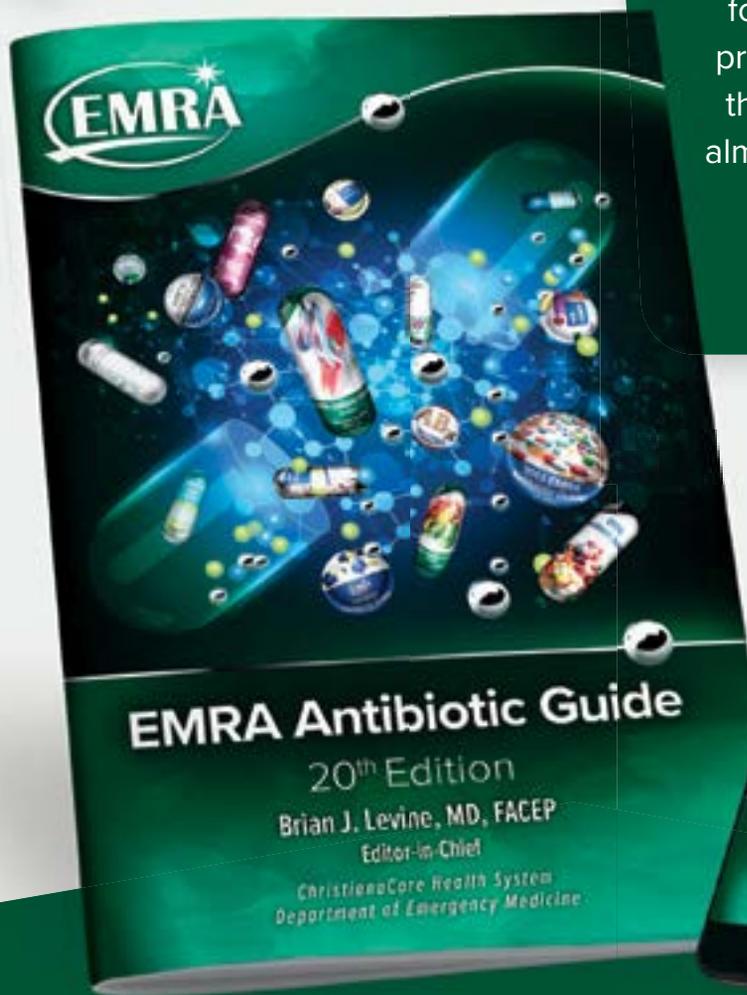
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# Cauda Equina or Just HSV? Don't Forget About Elsberg Syndrome

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**C**auda equina syndrome (CES) is a “can’t miss” diagnosis in the emergency department characterized by lower back pain.

It is associated with red flag symptoms such as urinary retention or incontinence, saddle anesthesia, and weakness or sensation changes in the

lower extremities.

Most providers consider the common textbook differential diagnoses for CES such as spinal epidural abscess, multiple



sclerosis, and local spinal tumors, but many are unaware of another rare cause of CES: herpes simplex virus (HSV), also known as Elsberg syndrome.

### CASE REPORT

A 39-year-old female with a past medical history of a recent genital herpes infection in the past month treated with oral valacyclovir presented to the Emergency Department (ED) with chief complaints of lower back pain, weakness and sensation changes in the right leg, and urinary retention. The back pain was described as a burning and tingling sensation in her sacral region that had been progressively getting worse over the past week, with radiation to the lower extremities. Urinary retention began on the day of presentation. She denied any recent travel, intravenous (IV) drug use or smoking history.

On physical examination, the patient's vitals were all within normal limits without a fever or tachycardia. Her examination was significant for 4/5 strength, decreased sensation to light touch, and pain in the left lower extremity.

The patient was started on broad spectrum anti-infectives including vancomycin, ceftriaxone, and acyclovir despite being afebrile because there was significant concern for a potential infectious etiology of her symptoms, with HSV highest on the differential.

ED workup consisted of basic laboratory values including complete blood count (CBC), comprehensive metabolic profile (CMP), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP), as well as magnetic resonance imaging (MRI) of the full spine and a lumbar puncture (LP) with cell count, cultures, HSV polymerase chain reaction (PCR), and opening pressure. Basic laboratory values were unremarkable, and the MRI of the full spine was within normal limits. The LP was significant for lymphocytic pleocytosis with 85% lymphocytes and negative for HSV.

Given the patient's history and lymphocytic pleocytosis, a clinical diagnosis of Elsberg syndrome was made, and the patient was admitted to

the floor and started on IV acyclovir every eight hours, later switching to oral valacyclovir at a dose of 1 gram three times per day. After 21 days, she was discharged after clinical improvement in her urinary retention.

### DISCUSSION

Elsberg syndrome is a clinical diagnosis that is a presumed infectious syndrome most commonly secondary to herpes simplex virus 2. It consists of acute or subacute lumbosacral radiculitis that must be considered in all patients presenting to the ED with CES.<sup>1</sup> The exact pathophysiology of Elsberg syndrome is not completely understood. However, it is hypothesized that the initial viral infection causes an inflammatory reaction that results in myelitis and radiculitis. Literature review reveals some case reports of Elsberg syndrome published over the years, but only one study from the Mayo Clinic retroactively found 30 patients suspected of having Elsberg syndrome through chart review from 2000 to 2016. In this study, urinary retention and weakness in the lower limbs were the most common clinical presentations (77% and 40% of patients, respectively).<sup>1</sup> Furthermore, most patients tested positive for HSV; however, other viruses such as varicella-zoster, cytomegalovirus, West Nile, and COVID-19 have been associated with the syndrome.<sup>2-5</sup>

Recorded cases of Elsberg syndrome show that it tends to present within a week of the initial viral infection, but this may be confounded by missed diagnoses in the subacute or late stages.<sup>1</sup> Furthermore, our patient presented subacutely, almost an entire month after initial HSV-2 infection, although this may be attributable to the oral valacyclovir she had been taking.

When it comes to diagnostic criteria and findings, the data is also limited and scarce. On spine MRI, there are no pathognomonic findings. MRI can either be negative or there can be findings of spinal cord lesions that are commonly multiple, discontinuous, and centrally or ventrally positioned on the cord.<sup>1</sup> Cerebrospinal fluid (CSF)

examination tends to show lymphocytic pleocytosis; however, CSF PCR detection of specific viruses can be negative and cannot be used to exclude presence of an infection.<sup>6</sup> Due to the wide range of diagnostic findings, a broad workup is recommended.

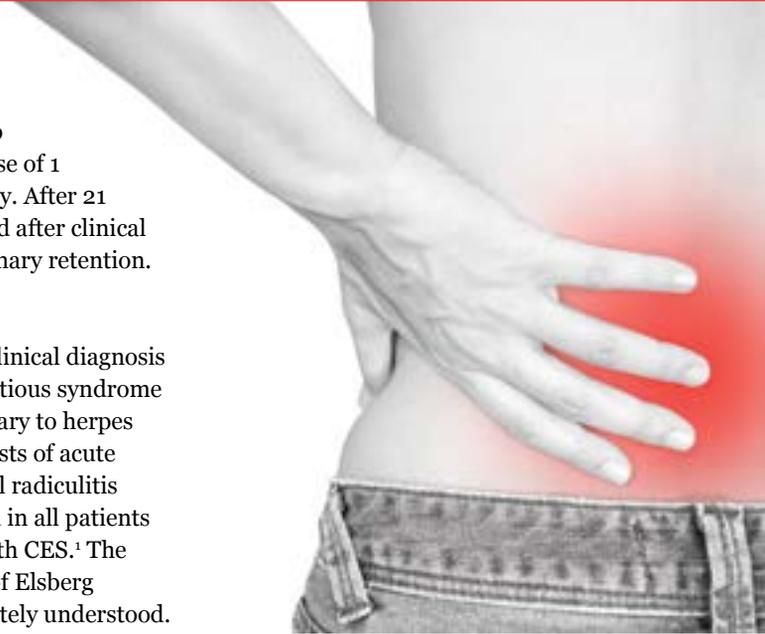
Lastly, in terms of treatment, there is no documentation regarding the best window for treatment or efficacy of different treatments. Most prior cases either turn to intravenous high-dose steroids or intravenous acyclovir to treat the inflammatory and viral components, respectively.<sup>1</sup>

Our patient did not receive steroids inpatient and had a slow resolution of symptoms with only IV acyclovir, whereas in other case reports, some patients received IV steroids and no antivirals.<sup>1</sup>

Nevertheless, the current recommendation is to proceed with these treatments, as both treatments are relatively well tolerated and there have been several case reports of Elsberg syndrome causing permanent neurological deficits in patients.<sup>7</sup>

### IMPORTANCE OF AWARENESS

Emergency physicians must consider Elsberg syndrome in their workup of CES because the condition is treatable, and discharge of these patients without full workup and treatment with acyclovir can result in potentially devastating permanent neurologic deficits. ★





# Wilderness on the Water: MedWAR 2022

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**E**MRA members met at Lake Chabot Regional Park near San Francisco on Oct. 4 to compete in MedWAR, the annual test of medical knowledge, wilderness skills, and endurance. The sunny temperate climate of the West Coast was certainly a welcome relief compared to the blustery conditions during MedWAR 2021 in Boston. The roughly 11-mile course circled the lake, taking competitors from scenarios on the water to panoramic views of the City by the Bay.

#### WHAT IS MEDWAR?

MedWAR, short for Medical Wilderness Adventure Race, is a competition concept developed in Augusta, Ga., and created by Drs. Michael Caudell and David Ledrick. Teams race through an orienteering course peppered with medical quizzes and proctored wilderness medicine simulation challenges. It's a test of strength, endurance, adaptability, and medical knowledge — all rolled into one.

Since its inception in 2000, MedWAR has been adopted internationally, with versions hosted by multiple medical schools and residency programs. EMRA MedWAR debuted in 2016, led by the Wilderness Committee's Carrie Jurkiewicz, MD, and Geoff Comp, DO, with faculty advisors Drs. Caudell, Taylor Haston, and Paul Auerbach, MD, MS. It has become one of EMRA's most dynamic annual competitions.

#### ESCAPE FROM ALCATRAZ

Before daybreak, more than 30 volunteers gathered their supplies and dispersed to six stations throughout the park. Once competitors arrived, EMRA Wilderness Committee Chair Katie Kammert, DO, reviewed race details and rules while everyone fueled up for the day. At 9 am, racers began their trek by "escaping Alcatraz" through a series of wilderness medicine matching cards and scrambled letters that eventually led them to tying the "King of Knots," the bowline.

#### CIRCUMNAVIGATING LAKE CHABOT

After escaping the notorious, cold, shark-infested waters, teams encountered a

diver in distress who was covered in jellyfish stings. Thankfully, there was a research team onboard that had an ultrasound graciously provided by Butterfly to diagnose a pneumothorax. From the Florida Keys, competitors climbed more than 1,000 feet where they embarked on a search-and-rescue mission from Denali basecamp. Teammates worked together to recover a climber suffering from High Altitude Cerebral Edema (HACE) and a femur fracture that required improvisational skills to make a splint and hypothermia wrap for transport.

Their journey took them downhill to the swamps of Georgia and some drunken "noodlers" who found the wrong end of a cottonmouth. A representative from BTG (CroFab creator) was able to play the victim and help racers think of resources that might be available inside and outside their ED. Further down the trail, an explosion occurred at a local meth house resulting in a mass casualty and pandemonium. While trying to treat an opioid overdose and blast injuries, racers had to ameliorate the agitated delirium of actor Michael Nguyen, MD, in order to perform a lateral canthotomy





for his retrobulbar hematoma. Wild boars of Zakopane, Poland, attacked local hikers, transpiring in one patient suffering from an evisceration and open chest wound while the other victim distracted rescuers with his screams about his problem “down there.” A priapism reduction in the wild was a first for MedWAR and certainly drew attention to the course. On the final stretch, racers returned to the beach where a surfer was found down after a lightning strike. A nearby shark wasn’t

deterred from this opportunity, so the victim required a tourniquet and CPR to help the victim “hang ten” again.

**TREKKING FOR TACOS**

With five minutes to spare, all teams finished the race and celebrated their success over tacos. Last year’s champions from Albany, Receptaculum Ignis, placed third, while Stanford, with three minutes to spare, took the victory from the Turkey Sandwich Titans of Riverside. The Spirit Award honoring Dr. Paul Auerbach

went to Miles of Smiles from Rutgers Medical School for their enthusiasm and embodiment of wilderness medicine. Team members received an autographed copy of Dr. Auerbach’s book, Wilderness Medicine, courtesy of Elsevier. Acting awards went to Michael Nguyen, MD, and Drake Johnson, MS-3, from UCSF for both distracting racers yet also teaching them valuable procedure skills. Many thanks to Elsevier, Butterfly, BTG Specialty Pharmaceuticals, and Zanfel for their generous contributions that helped this race become a reality.

EMRA members met at Lake Chabot Regional Park near San Francisco on Oct. 4 to compete in MedWAR, the annual test of medical knowledge, wilderness skills, and endurance. The sunny temperate climate of the West Coast was certainly a welcome relief compared to the blustery conditions during MedWAR 2021 in Boston. The roughly 11-mile course circled the lake, taking competitors from scenarios on the water to panoramic views of the City by the Bay.

**EMRA WILDERNESS LEADERS**

This event would not have been possible without the hardworking members of the Wilderness Committee: Chair Katie Kammert, DO; Chair-Elect Matt Basinger, MD; EMRA Board Liaison and Past Chair Yev Maksimenko, MD; Vice Chairs Kara Hatlevoll, DO, FAWM, Tyler Rigdon, MD, and Kaitlyn Votta, MD; and Assistant Vice Chairs Joseph Mueller, MD Candidate Class of 2023, Danusha Sanchez MD, MIS, PMP, NREMT, Sasha Selby, MD, and Sarah Spelsperg, PA-C, FAWM, FEWM, MD Candidate Class of 2023. Special thank you to Medical Director Lainey Yu, DO, MS, FAWM, and MedWAR faculty advisors Michael Caudell, MD, FACEP, and Taylor Haston, DO, FACEP. Finally, we greatly appreciate the staff at EMRA including Heather Deja, Candice Grantham, and Leah Stefanini.

If you are interested in joining the committee for MedWAR at ACEP23 in Philadelphia this October, please email [emrawildernessctte@emra.org](mailto:emrawildernessctte@emra.org). ★



MedWAR participants in action



**MedWAR is back Oct. 12 at ACEP23 in Philly!** Get your teams organized and your volunteers together **NOW!**



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# ECG Challenge

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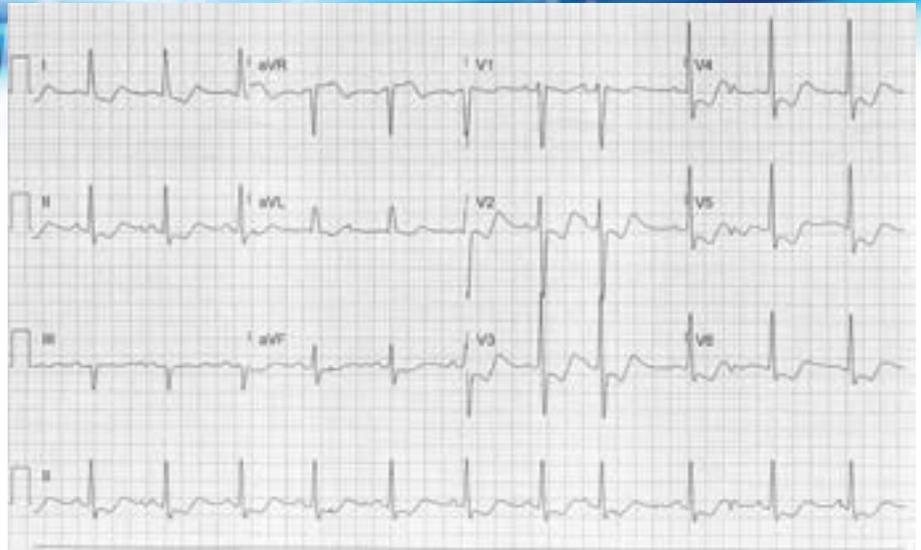


**CASE**

A 58-year-old male with a PMH of alcohol use disorder presents to the ED due to multiple falls.

**What is your interpretation of his ECG?**

**See the ANSWER on page 68.**



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# ECG Challenge

## ANSWER

**T**his ECG shows normal sinus rhythm at 70 bpm, normal axis, prolonged QRS complex duration with an IVCD, prolonged QTc interval, and STE in lead aVR. There are also STD, TWI, and U-waves in leads I, II, and V2-V6, all of which are seen with hypokalemia.

Potassium is primarily an intracellular ion and is critical in maintaining the resting membrane potential of the myocardium and is crucial during phases 2 and 3 of the cardiac action potential (AP). Hypokalemia leads to an increased risk of early afterdepolarizations (EADs) which are defined as a slowing or reversal of the normal repolarization during phase 2 or 3 of the cardiac AP. These EADs can trigger ventricular dysrhythmias such as torsades de pointes, polymorphic VT, and ventricular fibrillation.

The classic ECG finding seen in hypokalemia is the U-wave which is defined as a positive deflection after the T-wave, best seen in the anterior precordial leads at slower heart rates. U-waves are not specific for hypokalemia and can be due to hypomagnesemia, hypercalcemia, hypothermia, LVH, and medications. Other ECG findings in hypokalemia include increased amplitude and duration of the P-wave and QRS complex and PR interval prolongation.

The classic triad of ECG changes associated with hypokalemia includes TWI, STD, and U-waves, but all three findings are not always present, even with severe hypokalemia. When all three findings are present, it creates a “reverse Wellens” pattern of a downward deflection followed by an upward deflection following the QRS complex as seen in the case ECG. This pattern also resembles the graph of  $y = -\sin(x)$  (see **Figure 1**), so the authors propose calling this the “negative sine sign”. [Editor’s note: Dr. Schenker would like the readers to know that he had nothing to do with this and vehemently opposes the use of trigonometry in ECG education.]

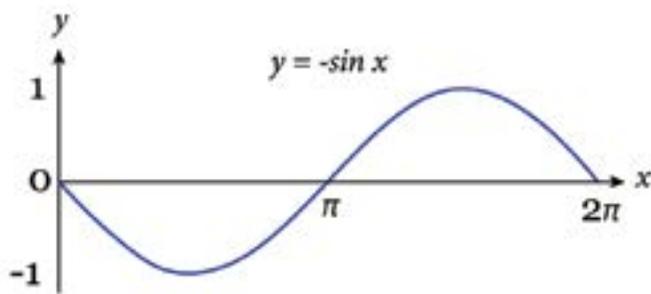


FIGURE 1: The “negative sine sign”

There is controversy regarding whether hypokalemia truly causes a prolonged QT interval or just the appearance of a prolonged QT interval due to the presence of U-waves (i.e., should the U-wave be included when measuring the QT interval). Regardless, hypokalemia that produces the appearance of a prolonged QT interval, TU complexes, or significant U-waves, increases the risk for ventricular dysrhythmias, in particular torsades de pointes.

The challenging aspect of treating hypokalemia-induced ventricular dysrhythmias is knowing how much potassium can be administered emergently and how quickly can it be given. Institutional protocols typically limit potassium replacement to 10 mEq/hour via peripheral access and 20 mEq/hour via central access, both of which may be insufficient to stabilize a patient with a ventricular dysrhythmia due to severe hypokalemia. The 2000 AHA ACLS guidelines state “If cardiac arrest from hypokalemia is imminent (ie, malignant ventricular arrhythmias), rapid replacement of potassium is required. Give an initial infusion of 2 mEq/min, followed by another 10 mEq IV over 5 to 10 minutes... Once the patient is stabilized, reduce the infusion to continue potassium replacement more gradually.”<sup>1</sup> The 2005 AHA CPR guidelines state, “If cardiac arrest from hypokalemia is imminent (i.e., malignant ventricular arrhythmias are present), rapid replacement of potassium is required. Give an initial infusion of 10 mEq IV over 5 minutes; repeat once if needed.”<sup>2</sup>

## CASE CONCLUSION

This patient’s workup was notable for a serum potassium level of 1.7 mEq/L. He was treated with IV and oral potassium and admitted to the internal medicine service.

## HYPOKALEMIA LEARNING POINTS

General Features:

- ECG triad of decreased TWI, STD, and U-waves (the “negative sine sign”)

EKG Features:

- T-wave flattening or inversion
- U-waves
- Slight STD
- STE in aVR
- Increased P-wave and QRS complex amplitude and duration
- PR interval prolongation
- QTc prolongation
- Increased ectopy (e.g., premature beats) and tachydysrhythmias

Clinical Significance:

- Hypokalemia changes can be masked by tachycardia
- Concurrent hypomagnesemia can precipitate ventricular dysrhythmias
- Prolonged QTc = increased risk of torsades de pointes

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- 1. A 19-year-old man with a history of developmental delay is brought in by his mother after she saw him drink a small amount of household bleach. The patient denies any complaints, including difficult or painful swallowing, shortness of breath, or pain. The results of the physical examination, including evaluation of the airway and examination of the oropharynx, are unremarkable. What is the most appropriate next step in the management of this patient?**

  - A. Administer a neutralizing agent
  - B. Arrange for an emergency department endoscopy
  - C. Discharge to home with outpatient follow-up
  - D. Perform gastric decontamination
- 2. A 64-year-old woman presents with blisters on her skin that slough when palpated. She appears well and has no other symptoms. Her medical records reveal that she was treated for oral ulcers several weeks ago. What is the most likely diagnosis?**

  - A. Bullous pemphigoid
  - B. Erythroderma
  - C. Pemphigus vulgaris
  - D. Toxic epidermal necrolysis
- 3. A 20-year-old woman presents with malaise and a rash. Her vital signs include BP 110/70, P 90, and T 38.3°C (100.9°F). The physical examination reveals tonsillar swelling with exudates, posterior cervical lymphadenopathy, splenomegaly, and a morbilliform rash. She says she has been taking antibiotics for a sore throat but is otherwise "never sick." What is the most likely diagnosis?**

  - A. Anaphylactic drug allergy
  - B. Coxsackievirus
  - C. Epstein-Barr virus
  - D. Streptococcal pharyngitis
- 4. What is one advantage of using atypical antipsychotic medications instead of typical antipsychotic medications in the treatment of psychosis?**

  - A. Decreased incidence of hypotension
  - B. Increased efficacy in treating negative symptoms
  - C. Less likely to cause neuroleptic malignant syndrome
  - D. More helpful in managing agitation in elderly patients with dementia
- 5. In which condition can intraosseous line placement be performed most safely?**

  - A. Fracture in the distal aspect of the same bone
  - B. History of osteogenesis imperfecta
  - C. Overlying cellulitis
  - D. Poorly controlled diabetes



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



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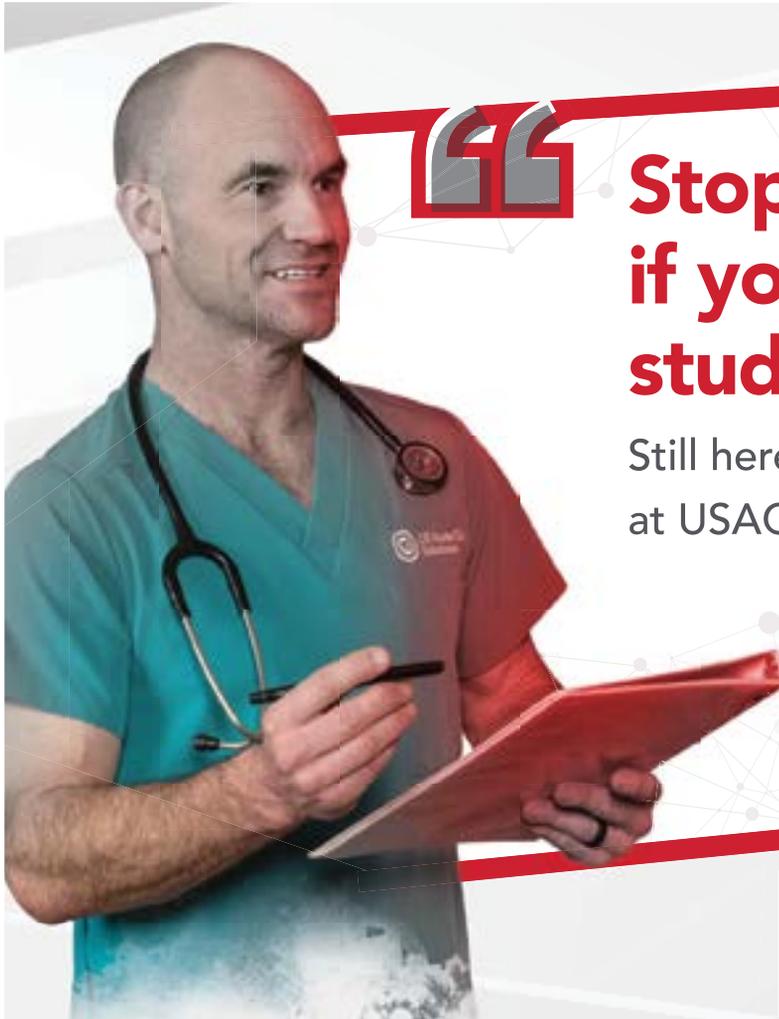
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