

# EM Resident

Official Publication of the Emergency Medicine Residents' Association

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## *Turning the Tide* **Climate Change and Toxicology**



**Esophageal Rupture**

**Ultrasound for Botfly Removal**

**Award Winners and  
Leadership Recognition**

**Complicated Hematuria**

**Not all Halos are Glaucoma**

**Influencing the  
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# Building Trust Through Integrity

“It’s against my beliefs.” “Personal choice.” “I know what some of my friends went through and I decided not to.”

These are the vague, dismissive statements I hear when I ask my patients why they didn’t get vaccinated against COVID-19. In truth, the reasons so many of our patients deferred the vaccine are myriad and diverse. For some, the reason was a mistrust of physicians and our healthcare system, which has become more of a market than a system with every passing year.

I can’t say I blame them. Through the 2000s, my patients saw highly profitable “pill mill” pain clinics raking in the cash across Appalachia only to see their loved ones with back pain from manual labor jobs turn to street opioids when regulations tightened, clinics closed, and their oxycodone supply dwindled. Older patients may even remember when the U.S. Narcotic Farm, a prison for the rehabilitation of incarcerated people with drug addiction in Lexington, Ky., was exposed in the 1970s for the torturous experimentation physicians and scientists had imposed upon the incarcerated. For decades, these physicians had bribed the incarcerated with heroin to participate in experiments. They would push vulnerable people to the most brutal depths of withdrawal in order to document their deterioration and determine whether withdrawal could be fatal.

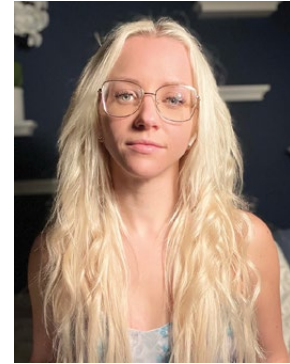
These atrocities and others like them (the Tuskegee untreated syphilis study, surgical experimentation on Black women throughout slavery and the Jim Crow era, and the lack of equitable healthcare provided to our patients who are incarcerated that continues to this day) have been committed by physicians who tell themselves they serve a greater good. Though we cannot directly compare the aforementioned tragedies with the conflicts we face in

EM today, we are confronting an ethical dilemma that has been brewing for years. It impacts patients’ health while business executives and shareholders, far-removed from the patients they impact, stand to make millions: the corporatization of emergency medicine.

The role of contract management groups backed by enormous private equity companies in the hiring, firing, and medical practice of EM physicians has become increasingly scrutinized. Little data is available to quantify the impact CMGs have on quality of care in the emergency department, but in a recent online survey of EMRA members many of you raised concerns that overreaching corporate interests by non-physicians may prioritize profits over patients. In the wake of the controversial predicted surplus of over 7,000 EM physicians by 2030, EMRA is joining this conversation. Based on member surveys, online member discussions, and meetings of the Board of Directors (composed of EM residents and fellows like myself) EMRA President Dr. Angela Cai has released EMRA’s EM workforce statement. Reproduced over the following pages, this living document is only one step in EMRA’s journey through the rapidly changing world of EM administration.

As Editor-in-Chief of *EM Resident*, I also must acknowledge the publication’s advertising relationship with CMGs. The other residents and myself who comprise the EMRA Board of Directors are perceptive to your concerns regarding these relationships, and also cognizant of the fact that many of you will go on to staff CMG-owned EDs. *EM Resident* will continue to examine our corporate relationships and serve as a platform for authentic discussion on this issue. I always welcome your letters to the editor on topics like this, sent to the email below.

What is most important to us as physicians must always be the patient.



**Jessica Adkins Murphy, MD**  
Editor-in-Chief, *EM Resident*  
University of Kentucky

This job does not exist for us to earn a salary. It certainly does not exist for shareholders to turn a profit. This job exists to help people who are suffering. Centering that humanity and compassion in the decisions we make is essential to making the right decisions for our patients and maintaining the public’s trust in our integrity. Our response to the difficult job market must be patient-centered. Reducing residency spots to alleviate saturated job markets in attractive cities is not patient-centered. On the other hand, enforcing the requirements of residency accreditation produces better physicians that meet our standards for patient care. Advocating for state and federal funding for emergency physician coverage in rural, lower-volume EDs would also alleviate urban job market saturation while better serving our rural patients currently seen by non-physicians and non-EM-trained physicians.

In the changes that we make in medicine, and the ones we passively allow to happen, it is our responsibility to constantly evaluate whether we are building a *compassionate* healthcare system or one driven by profit. We are trusting this system to heal the people we love most. We are trusting it to also heal the people who have no one else to care for them at all. ★

Let’s keep in touch!

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# TABLE OF CONTENTS

- 4** Influencing the Future of the EM Workforce  
LEADERSHIP SPECIAL REPORT
- 9** Turning the Tide  
TOXICOLOGY, SOCIAL EM
- 12** The Toxicology of COVID  
COVID-19, TOXICOLOGY, INFECTIOUS DISEASE
- 14** An Acutely Swollen and Cyanotic Leg  
CRITICAL CARE, NEPHROLOGY
- 16** Think Fast  
PEM, CARDIOLOGY
- 18** Hematuria in the Emergency Department: Evaluation and Management  
UROLOGY
- 22** Isolated Traumatic Gallbladder Hematoma Caused by Electric Scooter Accident  
TRAUMA
- 24** Uncontrolled Hyperglycemia as a Cause of Choreiform Movements  
ENDOCRINOLOGY
- 26** Use of Bedside Ultrasound in Diagnosing Boerhaave Syndrome  
ULTRASOUND, CRITICAL CARE
- 28** Bupropion Toxicity  
TOXICOLOGY
- 30** An Ultrasound Teaching Case  
INFECTIOUS DISEASE, ULTRASOUND
- 32** Torsades de Pointes after Olanzapine Use for Agitation  
CARDIOLOGY, TOXICOLOGY
- 34** Spinal Cord Ischemia in an Undifferentiated Patient  
NEUROLOGY
- 36** Not All Halos Are Glaucoma  
OPHTHALMOLOGY
- 38** Duty to Serve: Federal Law Enforcement and the Foundation for Emergency Medicine  
CAREER PLANNING
- 41** A Fellowship for the Future  
MEDICAL EDUCATION, CAREER PLANNING
- 42** COVID-19 Vaccine Equity and Distribution in EDs  
SOCIAL EM, COVID-19
- 44** EMRA @ ACEP22  
EVENTS
- 45** EMRA Honors 2022 Winter Award Recipients  
AWARDS
- 46** EM Leaders of Tomorrow  
LEADERSHIP
- 51** ECG Challenge  
CARDIOLOGY
- 53** Board Review Questions  
PEERPrep
- 54** Sponsor Thank You  
CORD AA

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# UNITY, PURPOSE, AND PASSION

## Influencing the Future of the EM Workforce

Angela Cai, MD, MBA  
EMRA President  
@angelagcai

**E**MRA has been hard at work ensuring that your voice, as the future of our specialty, drives solutions to the emergency medicine workforce in response to the multi-organizational workforce study. We would like to continue the dialogue by sharing what we've learned, what you've told us, and where EMRA stands. This is a living document being shaped by you.

The projection of a 9,000 EM physician surplus by 2030 has created a lot of angst and fear among all of us. But projections are just that — projections. As additional research continues, EMRA remains focused on solutions to design our futures.



### SUPPLY

#### **Residency Program Growth and Training Standards**

##### **EMRA's Stance**

EMRA is participating in a multi-organizational effort to propose recommendations to the ACGME for increasing residency standards. These recommendations must center on training quality rather than arbitrary training obstacles without educational merit.

EMRA continues to support both 3- and 4-year training programs and

individual resident decisions to choose their training format.

EMRA supports a broad definition of required resident scholarly activity, as this affords residents the opportunity to complete a project that is meaningful to their individual careers and best contributes to the advancement of our specialty.

##### **Background**

Over the past 6 years (2014-2020), the number of emergency medicine residency training positions

and programs has grown rapidly, making EM one of the fastest growing specialties. The number of training programs has increased by 15%. The number of training positions has grown even more, by 30%, which is a result of both new program growth and existing programs expanding their complement. Compared to prior years, the proportion of programs sponsored by for-profit institutions during this period has increased from 4% to 37%. All programs are sponsored by hospitals

or educational consortiums. No current programs are sponsored by physician staffing groups.<sup>1</sup> Hospitals that created Graduate Medical Education (GME) programs after 2015, known as “GME-naive,” have a strong incentive to increase the number of residents at their site within 5 years of starting because the Centers for Medicare and Medicaid Services (CMS) calculates their training cap after the fifth year.<sup>2</sup>

Residency programs are accredited by the Accreditation Council for Graduate Medical Education (ACGME) Emergency Medicine Review Committee based on compliance with minimum standards. Accreditation of ACGME programs is a voluntary process. ACGME policy states, “It is not the intent or purpose of the ACGME to establish numbers of physicians in any specialty.”<sup>3</sup>

## DEMAND

### Nonphysician Providers

#### EMRA’s Stance

Patients want and deserve emergency medical care that is provided and led by physicians.

EMRA has believed for decades that the only pathway to the independent practice of emergency medicine in the 21st century is completion of an ACGME/AOA accredited residency training program and ABEM/AOBEM board certification.

EMRA partners with ACEP in their advocacy against non-physician provider independent practice, including at the ACEP state chapter level and through the AMA Scope of Practice Partnership.

EMRA supports post-graduate training of nonphysician providers to ensure that patients receive the highest quality care. Nonphysician provider post-graduate training is not equivalent to residency training and should not interfere with resident education.

### Background

In 2012, the Affordable Care Act awarded up to \$200 million to educate and train nurse practitioners. Since then, the number of nurse practitioners practicing in the United States has nearly doubled, from 154,000 to over 290,000 as of March 2020. EM has seen a similar explosion of care being delivered by nonphysician providers (NPPs, ie nurse practitioners and physician assistants), with an estimated 15% of all patient encounters in emergency departments being seen by a NPP in 2015 (and that ratio is projected to grow to 20% by 2030).<sup>4,5</sup> The projected increase in proportion of visits seen by NPPs is notable given the projected oversupply of emergency physicians by 2030.

What factors contribute to the increased utilization of NPPs? First, NPPs are substantially cheaper to employ, while allowing hospitals and staffing groups to bill patients at the same rate and thus generate a higher profit. Second, there is a strong push for independent practice for NPPs, especially for nurse practitioners. To date, Full Practice Authority (FPA) legislation has now been passed in 23 states, granting nurse practitioners the full practicing rights of a physician after as little as 18 months of online classes and 500 clinical hours required for NP licensure.<sup>6</sup> In many other states, NPs are able to practice with minimal physician oversight. A recent analysis by a team of nurse researchers concluded that due to extensive variability across the academic preparation, licensure, and certification of NPs in EDs, NPs **should not** perform independent, unsupervised care in the ED, regardless of state law or hospital regulations, in order to protect patient safety.<sup>7</sup> Despite these concerns, with tremendous lobbying

effort by the American Association of Nurse Practitioners and strong financial backing from other supporters of FPA within health care, the FPA movement has advanced significantly over the past decade. Expanding NPP scope of practice affects patients across all specialties, which is why EMRA supports ACEP’s alliance with the AMA Scope of Practice Partnership to coalesce efforts across the house of medicine.

## RURAL MEDICINE

### EMRA’s Stance

EMRA advocates for increased elective opportunities for rural exposure in residency.

EMRA believes that every emergency patient deserves to see an emergency physician; however, given current economic and workforce constraints, rural patients are often evaluated by nonphysician providers without physician supervision. EMRA believes emergency physicians should direct care for these patients by setting training standards for nonphysician providers and supervising rural care onsite as much as possible with telemedicine as an adjunct.

### Background

Providing adequate access to emergency care to rural communities has been an ongoing challenge. Although 1 in 5 Americans live in a rural area, only 8% of all emergency physicians practiced in rural communities as of 2020 — and that number is shrinking.<sup>8</sup> In fact, over the past 4 years, 96% of EM graduates chose to practice in urban areas. The rural emergency physician workforce is also aging, with many now in mid-career or closing in on retirement.

In addition to a maldistribution of emergency physicians, rural hospitals are facing other significant challenges

**Several solutions have been proposed to help improve access to emergency care for rural communities including the expansion of telemedicine, post-graduate training in EM for NPPs.**



that make practicing rural EM uniquely challenging. These factors include the following:

- **Hospital Closures and Financial Strain:** Due to a combination of low patient volumes, especially in frontier departments, and a lower income and often uninsured payer mix, many rural facilities operate at a negative margin and require state and federal assistance to remain open.<sup>9,10</sup> However, this assistance is often not enough and over 136 rural hospitals have closed over the last decade, with another 21% of rural hospitals at high risk of closing even pre-pandemic.<sup>11</sup> The COVID-19 pandemic has now accelerated the rate of hospital closures with a record number of 73 hospitals closing over 2021.<sup>12</sup>
- **Limited Resources:** Practicing EM in a rural community also poses challenges for those with limited exposure to delivering care in an under-resourced setting. Because many services are often not directly available or require transfer, emergency physicians often have to make crucial decisions without access to this potentially care-altering information. If the patient is critically ill or specialty services are not available in the hospital, one of the most important decisions is when to admit versus when to transfer — many times the transferring hospital will be an hour or more away. This requires managing critically ill patients for longer periods of time, the ability to step into the role of a variety of subspecialists, and a mastery of a wide breadth of procedural skills.
- **Lack of Exposure to Rural Emergency Medicine:** The majority of EM residents train in tertiary care centers with 24/7 access to subspecialty care, with only a small subset of these programs requiring a rural rotation.<sup>13</sup> Thus, most emergency physicians will graduate residency without ever being exposed to rural EM. Other challenges that exist to exposing

residents to rural EM include ensuring appropriate supervision, adequate patient volume, and acuity at selected rural sites, as well as longitudinal education experiences. Rotating at rural sites may cause both financial strain to the resident and may pose a strain on the resident's family and relationships as well.

- **Lack of Incentives:** Incentives to recruit emergency physicians to rural areas, such as loan repayment programs and sign on bonuses, are not widely available.

Several solutions have been proposed to help improve access to emergency care for rural communities including the expansion of telemedicine, post-graduate training in EM for NPPs, as well as reimbursement reforms.

## EXPANDING THE SCOPE OF EMERGENCY MEDICINE

### EMRA's Stance

EMRA recognizes that most trainees want to work in the hospital-based emergency department. We also know that emergency medicine is changing with patient and workforce needs. Expanding the specialty's scope allows emergency physicians to have options for variety in practice which promotes career longevity. EMRA is committed to looking ahead at the future to ensure that residency curricula appropriately prepare trainees for their practice environment of choice.

EMRA supports telemedicine training opportunities, interstate licensure compacts, and reimbursement policies that promote current practice of and future innovations in telemedicine.

### Background

There are many opportunities for innovation in EM practice. While many trainees value the traditional model of providing acute unscheduled care in a hospital-based emergency room, emergency physicians are interested in non-traditional practices to fill gaps in the health care system and provide a diversity of practice, which can promote career satisfaction.<sup>14</sup> Patients are also interested in new delivery models such as telemedicine.<sup>15</sup>

All told, there are many reasons to expect that EM will not be practiced in the same way in 20 years, and we must prepare trainees for future models.<sup>16</sup> In addition to providing opportunities, this scope expansion can provide new job opportunities in the face of projected workforce shortages. Other specialties have evolved by expanding their practice such as the expansion of anesthesia into pain management.

## CORPORATE PRACTICE OF EMERGENCY MEDICINE

### EMRA's Stance

EMRA believes patient care and medical education must be evidence-based.

EMRA advocates for emergency physicians to have fair employment environments, including due process, billing transparency, and honoring of employment contracts.

EMRA is concerned that corporate-investors in emergency medicine practice and training create conflicts of interest and incentives that infringe upon physician and patient well-being. While all business models have a profit motive, the incentives are more powerful when outside stakeholders are invested.

EMRA supports objective research on the potential impact of corporate affiliated emergency residencies and the resulting hiring and training outcomes.

### Background

The Corporate Practice of Medicine doctrine (CPOM) is the term used for the general principle that limits the practice of medicine to licensed physicians and prohibits corporations from practicing medicine or directly employing a physician. Most, but not all, states have laws prohibiting the corporate practice of medicine. These laws can limit or prohibit non-physicians from owning, investing in, or otherwise controlling medical practices. Exceptions to CPOM are common and include professional corporations formed by physicians, hospitals, and health maintenance organizations (HMOs). Corporate generally refers to public shareholders, venture capital,



**The concern with corporate actors in medicine is the conflict of interest between financial performance versus the patient care, educational, and research missions of EM practice and training.**



private equity, insurance companies, and health systems.<sup>18</sup>

These laws are particularly pertinent when discussing physician practice management companies (PPMC), sometimes referred to as contract management groups (CMGs), which are corporate entities that contract with multiple hospitals to provide ED physician staffing. The PPMC often handles billing, scheduling, liability insurance, and other important (but often cumbersome) administrative tasks in exchange for overhead fees collected from the physician's reimbursement. It can also provide educational support, leadership training, and other advancement opportunities for physicians, but it cannot directly employ physicians or provide clinical care.<sup>19</sup> PPMCs have a wide variety of governance (eg democratic, partnership) and ownership structures that range in various combinations of physician

owners, corporate owners, and corporate investors.

Despite the existence of this doctrine, corporate interests have had increasing involvement in health care. In 2016, the percentage of physicians who do not have ownership in their practice across all specialties topped 50% for the first time. Physicians across all specialties including EM have questioned the role of corporate actors infringing on autonomous clinical practice and medical education.<sup>20</sup> Consolidation in health care across insurers, hospitals, and physician groups continues to increase the role of corporate actors in EM. Since their emergence in EM in the 1970s, PPMCs in 2013 contracted with over half of the EDs in the U.S.<sup>21</sup>

Fundamentally, the concern with corporate actors in medicine is the conflict of interest between financial performance versus the patient care, educational, and research missions of

EM practice and training. While all health care businesses must generate profit, certain corporate structures may incentivize business decisions at odds with physician or patient well-being. For example, the economics of private equity control might incentivize riskier, more aggressive strategies that are especially concerning given the lack of transparency in health care relative to other industries where private equity invests.<sup>22</sup>

While there is no objective data on the impact of private equity or other corporate investors in EM, their role in poor patient outcomes and increased spending in the nursing home industry has led the public and Congress to scrutinize their presence in health care overall.<sup>23</sup> Further complicating the understanding of corporate actors in EM is the wide variation in ownership and governance structures that utilize corporate investment. ★



# Penn State Health Emergency Medicine

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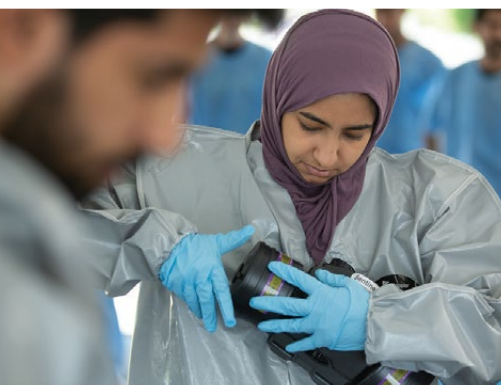
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# TURNING THE TIDE

## Harmful Algal Blooms and Climate Change



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I had never heard of red tides until moving to California for residency training. The first encounter I had with red tides evoked a moment of complete awe. I was certain I was witnessing a once-in-a-lifetime show of nature. With each crashing wave, a shock of light would travel through the water, brightening the night like a lightning bolt. It was magical, to say the least, but little did I know the evil nature hiding beneath these infamous coastal red tides.

Red tides are created by a rapid growth and accumulation of marine plankton. These organisms are photosynthetic, and some possess the remarkable ability of producing

bioluminescence, which explains the amazing phenomenon I witnessed that summer night in San Diego. These marine planktons are incredibly important for atmospheric carbon dioxide absorption and are the foundation of marine food chains. However, some species can also produce toxins that can cause significant harm to the health of coastal communities. These are called Harmful Algal Blooms (HABs).<sup>1</sup> HABs also occur in freshwater sources, such as the Great Lakes, with cyanobacteria that produce a liver toxin. The focus of this piece will be coastal marine HABs.

Climate change is altering aquatic ecosystems. As surface temperatures increase and oceans acidify, HABs become more prevalent. The 2021 Intergovernmental Panel on Climate Change's (IPCC) report, which is an aggregation of the leading climate science, concluded that HABs and their harmful health effects to coastal communities are directly linked to climate change.

Since the 1980s, coastal warming, deoxygenation, and eutrophication has caused overgrowth of harmful algal blooms (HABs) and pathogenic organisms such as *Vibrio*, impacting food supply, tourism, and human health, according to the IPCC.<sup>2</sup>

For clinicians, the science translates to bedside care and diagnoses. Given the unavailability of rapid assays, the diagnosis and management of poisoning from HABs is largely based on the emergency medicine clinician's ability to interpret symptoms and exposure history. Failure to consider HABs poisoning in the right clinical context can have a significant impact on outcomes, given their association with adverse chronic health effects and potential lethality.<sup>3</sup> As we continue to adapt to the detrimental health effect of climate change, it is our responsibility to continue to educate ourselves on the environmental health threats to the communities we serve. In this context, we will summarize the marine toxins presenting more frequently (as outlined in Goldfrank's *Toxicologic Emergencies*).<sup>4</sup>



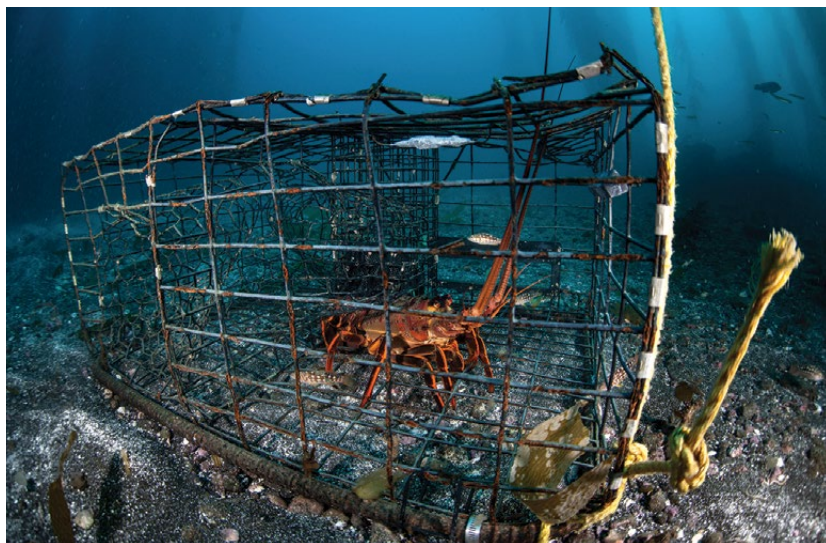


## Saxitoxin (Paralytic Shellfish Poisoning)

Saxitoxin is a neurotoxin that acts by blocking voltage-sensitive sodium channels and can cause rapid development of symptoms within 30 minutes of ingestion. Severity of symptoms typically correlates with higher numbers of contaminated shellfish consumed. Symptoms are mostly neurologic and include perioral paresthesia, headaches, numbness, cerebellar dysfunction, cranial nerve dysfunction, muscle weakness, and ultimately respiratory failure may occur. Various assays for saxitoxin exist, but are generally not readily available; thus, diagnosis is largely based on history and clinical presentation. Mortality due to respiratory failure has recently been reported to be as high as 15% depending on availability of emergency care<sup>5</sup> and typically occurs within the first 12 hours; however, with the appropriate supportive management, symptoms tend to resolve within a few days.

## Brevetoxin (Neurotoxic Shellfish Poisoning)

Brevetoxin is a heat-stable toxin that induces symptoms by stimulating sodium channels of nerves and muscles. Symptoms typically develop within 3 hours of ingestion. Classically, patients present with gastrointestinal symptoms and neurologic symptoms such as headaches, myalgias, tremors, ataxia, dizziness, and dysphagia. Patients have also reported temperature-related dysesthesias, which is the reversal of hot and cold perception. Unlike saxitoxin, brevetoxin does not cause paralysis. Interestingly, if present at high concentrations during red tides, brevetoxin can be inhaled and cause respiratory irritation and bronchospasm. As with saxitoxin, various assays that identify brevetoxin exist; however, given their unavailability, diagnosis is based on history and treatment is supportive. In patients with respiratory symptoms, bronchodilators can be used. No deaths have been reported and most patients recover within 72 hours.



## Domoic Acid (Amnesic Shellfish Poisoning)

Domoic acid is structurally similar to glutamate. It causes neuronal excitation which can lead to cellular death in the amygdala and hippocampus (hence the name *amnesic*). Symptoms typically develop at 5 hours after ingestion, but have been reported up to 38 hours after ingestion. GI symptoms such as nausea, vomiting, diarrhea, and abdominal cramps can occur. Neurologic symptoms include headaches, disorientation, chewing, grimacing, ophthalmoplegia, and in severe cases, anterograde memory loss, seizures, coma, and death. Cardiac and hemodynamic instability may occur. Treatment is largely supportive. While some deaths have been reported, mortality is less than 2% and typically occurs in older adults. Long-term memory deficits and neuropathies have been reported in up to 10% of patients.





### **Okadaic Acid (Diarrhetic Shellfish Poisoning)**

There are several types of toxins that cause diarrhetic shellfish poisoning; however, okadaic acid is the most well-known. It acts by inhibiting phosphatase 1A and 2A, which causes localized gastrointestinal symptoms such as vomiting, diarrhea, and abdominal cramps. Treatment is supportive and symptoms resolve within 3 days. Interestingly, these toxins have been shown to be tumor-promoting and may have an association with some gastrointestinal malignancies.<sup>6</sup>

### **Ciguatera Toxin (Ciguatera Fish Poisoning)**

Ciguatera toxin is a heat-stable, acid-stable, odorless, and tasteless toxin that acts by depolarizing voltage-dependent sodium channels and causing membrane depolarization. Illness typically develops after ingestions of a contaminated tropical reef fish, such as red snapper, barracuda, parrotfish, among others. Symptoms usually develop within 2-6 hours of ingestion of toxin, but can occur up to 72 hours after exposure.

Patients with ciguatera fish poisoning can present with a constellation of symptoms which typically involve the gastrointestinal, neurologic, and cardiovascular systems. Myalgias and arthralgias are commonly reported. GI symptoms usually include nausea, vomiting, diarrhea, and abdominal cramps. Neurologic symptoms can be substantial. These have been described as perioral paresthesia, metallic taste, blurry vision, sensation of loose or painful teeth, pruritus, temperature-related dysesthesias, peripheral paresthesias, and ataxia or other cerebellar dysfunction. Cardiovascular involvement may include bradycardia, heart-blocks, and hypotension. Life-threatening symptoms such as coma and respiratory distress are rare but have been reported. GI symptoms typically resolve in 1-2 days, but cardiovascular and neurological symptoms can persist for days to weeks.

The FDA is capable of testing fish for ciguatera toxin, but



this is not readily available to clinicians. Given the difficulty accessing laboratory testing, diagnosis is largely based on history and clinical features. Vomiting and diarrhea tend to accelerate the elimination of toxins, therefore activated charcoal can be considered in patients that do not have vomiting. Mannitol has been reported to improve neurologic symptoms yet a clear benefit has not been proven and caution should be used in patients with hypotension. Associations have been documented between ciguatera fish poisoning and climate variability.<sup>7</sup>

## **CONCLUSION**

In summary, climate change is altering the distribution and seasonality of coastal HABs via complex processes.<sup>8</sup> These single-celled organisms produce toxins, which are ingested by other marine organisms and can subsequently be ingested by humans. At high enough concentrations, even walking or swimming in contaminated waters can cause illness. Most toxins produce a combination of gastrointestinal and neurologic symptoms. Since the diagnosis is primarily based on exposure history, a high index of suspicion is needed. Given the unavailability of antidotes, treatment is largely supportive. Preventing and minimizing exposure is therefore one of our most important tools.

We depend on better research and public health interventions to reduce morbidity and mortality, which in turn depends on our diligence in reporting suspected cases to the Centers for Disease Control and Prevention.<sup>9</sup> Additionally, recognizing and addressing climate change as a public health issue will reduce future warming and health harms in our communities.

## **More on Climate Health**

Tune in to our EMRA\*Cast episode with Dr. Rublee to hear more about climate health, emergency medicine, and health policy. **Listen online or on your favorite podcast platform** at <https://www.emra.org/about-emra/publications/emra-cast/hot-topics-climate-change/#shownotes>. ★



## THE TOXICOLOGY OF COVID

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Since 2020, the COVID-19 pandemic has run rampant in the United States, significantly altering many facets of everyday life. Since its onset, the world has been looking for preventative measures and treatments. As emergency physicians, we have seen the changing landscape of treatment modalities used in the hospitals over time and have watched with excitement the rapid development of COVID-19 vaccines.

However, while we were watching the rapid scientific development, there was a concurrent wave of misguided therapeutic misadventures among the general population.

A quick internet search will reveal myriad therapies, all with varying degrees of potential risk. Many proposed therapies have been widely publicized by prominent public figures and news outlets and have gained widespread popularity without sufficient scientific evidence to demonstrate their efficacy and safety. Perhaps the most widely discussed treatments or preventative measures in the mainstream media were bleach, chloroquine/hydroxychloroquine, and, most recently, ivermectin.

## Bleach and Household Cleaners

During the COVID-19 pandemic, the use of disinfectants increased dramatically. There were many news stories describing people ingesting bleach or attempting other self-decontamination methods to help fight off the virus. How true were these stories? National Poison Center data demonstrate a 20.4% increase in calls about bleach and other disinfectants from January 2020 to March 2020 compared to the same time period the year prior.<sup>1</sup> One survey described 60% of respondents had increased household cleaning frequency due to the pandemic.<sup>2</sup> Of the respondents, 39% reported using household disinfectants, including bleach, inappropriately. Methods included ingestion, application on skin, inhalation, and cleaning produce with disinfectant or household cleaners. In addition to the adult misuse of these products, a large percentage of the calls to the poison centers during the Covid pandemic have been accidental exposures in children, as has been the case in years before the pandemic.<sup>1</sup>

Many household cleaners and disinfectants are caustic agents. Important considerations for risk of significant damage include the amount ingested and the pH of the substance. Initial management of a patient exposed to a caustic agent includes a rapid assessment of the patient's airway. If there is concern for potential airway compromise (significant swelling, stridor, inability to tolerate secretions), emergent intubation should be considered for airway protection. Once the patient is stabilized, the next question is whether the patient requires endoscopic evaluation.<sup>3</sup> As a rule, any intentional ingestion

requires endoscopy, as the volume in these ingestions is potentially larger. In addition, those with intentional ingestions are less likely to report the development of symptoms, including pain, which could indicate significant damage has occurred. In unintentional ingestions (i.e. children), endoscopy is indicated if the patient has stridor or two of the following: pain, vomiting, or drooling.<sup>3,4</sup> Patients with ingestions that meet the concerning parameters should be admitted for monitoring and GI should be consulted.

## Chloroquine/Hydroxychloroquine

Early in the pandemic, hydroxychloroquine was used for some COVID-19 patients based on early data. However, further studies demonstrated its lack of benefit and potential harm.<sup>5,6</sup> Despite the changing science, the general population continued to ask for and search out hydroxychloroquine or its counterpart, chloroquine. One high profile case involved a husband and wife who ingested aquarium cleaner containing chloroquine. Sadly, the husband died, and the wife became critically ill.<sup>7</sup>

Of the proposed COVID-19 treatments, chloroquine or hydroxychloroquine are perhaps the most likely to cause life-threatening toxicity in overdose. These drugs are best known as malarial and rheumatologic treatments.<sup>8</sup> In overdose, patients can develop toxicity quickly. They can develop significant hypokalemia, QRS prolongation, QTc prolongation, hypotension, respiratory depression, seizures, and coma. Management of these patients is complicated and requires early aggressive intervention.<sup>9</sup> This can include gastrointestinal decontamination if appropriate, early

intubation, and treatment with high dose diazepam and epinephrine, in addition to supportive care. For chloroquine and hydroxychloroquine toxicity, epinephrine is the vasopressor of choice as it is the best studied. Interestingly, high dose diazepam is also shown to be effective (2 mg/kg IV over 30 minutes then 1-2 mg/kg/day for 2-4 days).<sup>8</sup> The mechanism is yet to be elucidated, but several theories have been proposed that are beyond the scope of this short review.

## Ivermectin

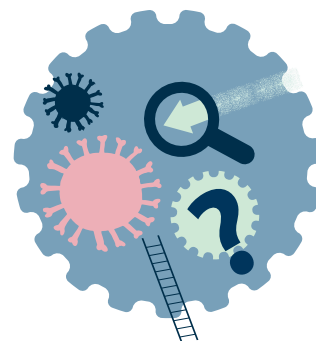
A more recent drug discussed in the media for potential use against COVID-19 is the anthelmintic ivermectin. Much of the recent fanfare resulted from an Egyptian study that was purported to show a significant mortality benefit.<sup>10</sup> However, this preprint article was retracted due to concerns about its data.<sup>11</sup> Despite the retraction, the article has been widely viewed, and ivermectin is being discussed regularly in the media. This has led to an uptick in calls to Poison Centers, as noted by the Mississippi State Department of Health.<sup>12</sup>

Although ivermectin has not been shown to be effective against COVID-19, it is generally well-tolerated in overdose. There are scant case reports of significant toxicity. Therapeutic use can result in gastrointestinal symptoms.<sup>13</sup> Overdose can cause encephalopathy and CNS depression.<sup>13,14</sup> Management is supportive. Many news articles describe individuals purchasing veterinary preparations of ivermectin, some of which are higher concentrations. We may learn more about this drug in overdose because of this recent misuse by the public.

## Conclusion

The COVID-19 pandemic and the myriad questions surrounding it have led individuals to search for prevention and treatment modalities with questionable scientific backing. It is important for emergency medicine providers to be aware of the potential toxicity from exposure to these xenobiotics.

**Poison Control Centers are available 24/7 to assist in the management of poisoned patients. Please do not hesitate to call us: (800) 222-1222. \***





# An Acutely Swollen and Cyanotic Leg

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A 44-year-old female presents to the emergency department with acute left leg pain that began one hour prior to arrival. The patient states that she was bending over and felt a pop in her left leg. On exam, the left leg is discolored (Fig. 1), warm, swollen, and exquisitely tender to palpation. There is decreased sensation distal to the left knee. 1+ dorsalis pedis artery and posterior tibialis artery pulses are palpated on the left, 2+ on the right. A point-of-care ultrasound (POCUS) was performed (Fig. 2, Fig. 3, Fig. 4), which confirmed the diagnosis.

## QUESTION. What is the next best step in management from the ED?

- IV antibiotics and hospital admission
- Systemic fibrinolytics
- Discharge with oral antibiotics
- Urgent vascular imaging

## ANSWER

The patient in the clinical vignette is suspicious for phlegmasia cerulea dolens (PCD). The correct answer is D: urgent vascular imaging. Point-of-care ultrasound (POCUS) of the left leg (Fig. 2 – Fig. 4) demonstrated a non-compressible thrombus extending from the left common femoral region to below the left popliteal region. Vascular surgery was consulted, and the patient was promptly taken to the interventional radiology suite for concurrent mechanical and chemical thrombolysis.

## Definition

PCD is a rare but emergent condition of massive venous thrombosis that can



**FIGURE 1.** Lower extremity physical exam findings

rapidly produce irreversible vascular gangrene.<sup>1,3</sup> First described by Gregoire in 1934, PCD is recognized by the classic triad of extremity pain, swelling, and blue discoloration secondary to venous occlusion.<sup>1</sup> Most cases of PCD are due to a DVT of an iliac or femoral vein<sup>4</sup>, and only 2-5% of PCD cases are caused by an upper extremity DVT.<sup>5</sup> This blue discoloration differentiates PCD from phlegmasia alba dolens, which presents as a pale limb due to arterial spasm and may precede PCD.

## Staging

In general, acute limb ischemia is classified by stages I-III: I (Viable), IIa (Marginally Threatened), IIb (Immediately Threatened), and III (Irreversible). Stage I (Viable) has no sensory loss, no muscle weakness, and audible doppler signals of the artery and vein. Stage IIa (Marginally Threatened) has sensory loss of the toes or no sensory loss, no muscle weakness, sometimes inaudible doppler signal of the artery, and audible doppler signal of the vein; the limb is salvageable with treatment. Stage IIb (Immediately Threatened) has sensory loss of more than the toes, mild to moderate muscle weakness, inaudible doppler signal of the artery, and audible doppler signal of the vein; the limb is salvageable with

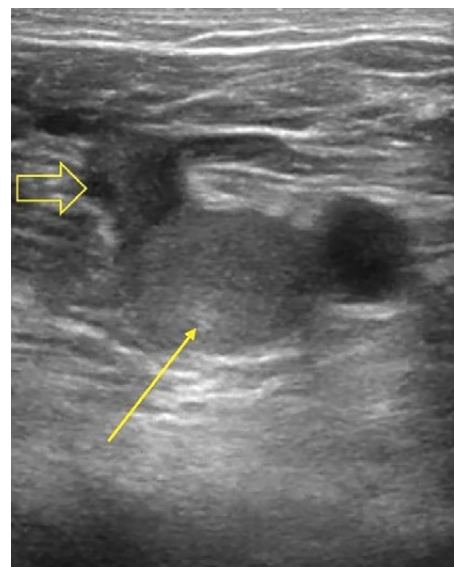
immediate revascularization. Stage III (Irreversible) has a profound sensory loss, muscle paralysis, and inaudible doppler signals of the artery and vein; this often requires amputation.<sup>6</sup>

## Risk Factors

Risk factors for PCD include malignancy, hypercoagulable disorders, venous stasis, oral contraceptives, and a history of previous DVT. In a systematic review, over one-third of patients with PCD had a malignancy. In contrast, two-thirds of patients with PCD had a history of hypercoagulable disorder, venous stasis, oral contraceptive use, or previous DVT. However, in 16% of patients, there was no history of disease (n=62).<sup>1</sup>

## Diagnosis

PCD diagnosis can be accomplished clinically with visualization of a swollen and cyanotic limb combined with positive DVT in the iliac or femoral veins confirmed by duplex ultrasonography,<sup>2</sup> which uses both B-mode and doppler features. Contrast venography is considered the gold standard, but is less commonly utilized due to ultrasound's comparative safety, accessibility, and portability.



**FIGURE 2.** POCUS showing non-compressible, echogenic, occlusive thrombus at the junction of the left femoral vein (solid arrow) and left greater saphenous vein (open arrow).



## Complications

The major complications of PCD include pulmonary embolism, myonephrotic metabolic syndrome, and compartment syndrome.<sup>1</sup>

**Pulmonary Embolism (PE):** In patients with PCD, 29% had PE, resulting in a 50% mortality rate.<sup>1</sup> PE is a life-threatening complication for 50% of untreated DVTs and contributes to 10-15% of all hospital deaths.<sup>7</sup> All patients suspected of having a PE should be on telemetry, have frequent blood pressure monitoring and maintain oxygen saturation greater than 95%. IV crystalloid fluids are given to increase preload. These patients should be given therapeutic dosing of unfractionated heparin (preferred for renal insufficiency or increased subcutaneous tissue thickness) or low molecular weight heparin. Unfractionated heparin is given as an initial bolus of 80 units per kilogram, then 18 units per kilogram per hour with a goal activated partial thromboplastin time between 55 and 80 seconds.<sup>4</sup> Depending on the disease burden, catheter directed thrombolytics or systemic thrombolytics may be indicated.

**Myonephrotic Metabolic Syndrome (MNMS):** MNMS is a type of ischemia-reperfusion injury caused by metabolites of muscle cells that have undergone liquefactive necrosis. When the clot is destroyed, reperfusion occurs, and the accumulated metabolites circulate in the body and can cause fatal hyperkalemia, pulmonary edema, metabolic acidosis, and myoglobinuria. MNMS can be prevented with aggressive fluid resuscitation and, if severe, hemodialysis.<sup>6</sup>

**Compartment Syndrome:** Similar to MNMS, compartment syndrome is caused by ischemic reperfusion leading to increased capillary permeability. The increased permeability results in edema which is contained in a compartment of the limb that is surrounded by inflexible fascia. A compartment pressure greater than 30mmHg may necessitate a fasciotomy to avoid permanent damage to arteries, veins, and nerves in the area.<sup>6</sup>

## Treatment & Disposition

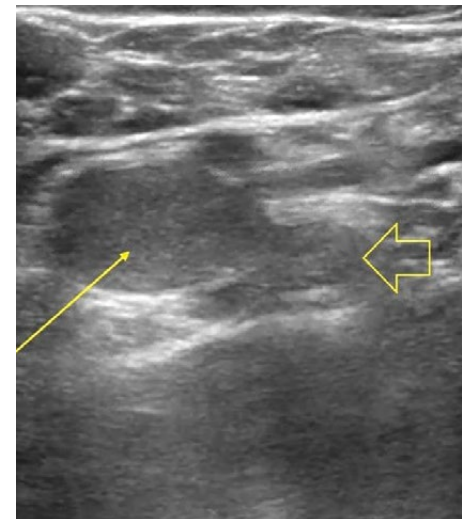
To treat PCD, immediate steps include neutral positioning of the limb, removal of constrictive clothing or dressings, fluid resuscitation with crystalloid

fluids, initiation of anticoagulation, and immediate consultation for thrombolysis or thrombectomy from vascular surgery, interventional radiology, or peripheral interventional cardiology. If none of these specialists are available, consider 50 to 100 mg intravenous alteplase (tPA) infused over four hours, keeping the contraindications of intravenous thrombolysis in consideration. Of note, there is limited data to support a particular alteplase dosing regimen.<sup>4</sup>

Acute limb ischemia stages I (Viable) and IIa (Marginally Threatened) are initially treated endovascularly due to less ischemic burden and less time constraint, while stage IIb (Immediately Threatened) is treated with immediate surgical thromboembolectomy.<sup>6</sup> Although many treatment options exist, urgent revascularization is universally required to prevent irreversible ischemia, amputation, and possibly death from cardiovascular collapse.<sup>1-3</sup> Regardless of the revascularization method, all patients require DVT prophylaxis and close follow-up to elucidate inciting factors.<sup>1,3</sup> Importantly, any patient undergoing thrombolytic therapy should be admitted to an intensive care unit.<sup>4</sup>

## Case Conclusion

After the patient underwent concurrent mechanical thrombectomy and thrombolysis, an inferior vena cava filter was placed and she was discharged with anticoagulation. Of note, she had no known risk factors, was not pregnant nor on oral contraceptives, and had no prior thrombotic events. Her one-month follow-up appointment for further hypercoagulability workup was negative. ★



**FIGURE 3.** POCUS showing non-compressible, echogenic, occlusive thrombus at the junction of the left femoral vein (solid arrow) and left deep femoral vein (open arrow).



**FIGURE 4.** POCUS showing non-compressible, echogenic, occlusive thrombus within the left popliteal vein (solid arrow).



## PEARLS

- **Identification:** If your patient has a swollen and cyanotic limb, use duplex ultrasound to look for a DVT in the iliac or femoral veins. Always monitor peripheral pulses.
- **Initial treatment:** Put the affected leg in a neutral position, remove constrictive clothing or dressings, give intravenous crystalloid fluids, initiate anticoagulation, and immediately consult an interventional vascular team for thrombolysis or thrombectomy. If none of these specialists are available, consider administration of alteplase (tPA).
- **Complications:** Look out for pulmonary embolism, myonephrotic metabolic syndrome and resulting arrhythmias, or compartment syndrome.



# THINK FAST

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Supraventricular tachycardia (SVT) remains the most common tachyarrhythmia in children, occurring in 1 in 250 to 1 in 1000 children.<sup>1</sup> Although common, the presenting symptoms of SVT can vary dramatically, even within similar age groups, posing a tremendous challenge to quick and accurate diagnosis.

With non-specific complaints such as fussiness and irritability in infants as well as chest pain and trouble breathing in children and adolescents, it is important for physicians to keep SVT in the list of differentials when caring for a pediatric patient.

This article aims to provide an overview of the pathophysiology of SVT, common presenting symptoms, approach to emergent management, and post-intervention care.

## Etiology

SVT is defined as a narrow complex tachycardia with a heart rate greater than 180 bpm in children and adolescents and greater than 220 bpm in infants.<sup>2</sup>

Only 15% of children will have spontaneous resolution of SVT.<sup>3</sup>

SVT demonstrates a bimodal incidence, occurring most often in children aged 6-9, and then again peaking in adolescence.<sup>3</sup> There are typically no congenital associations associated with SVT; however, Wolff-Parkinson-White syndrome (WPW), a subset of SVT, has been shown to have an association with Ebstein anomaly.<sup>3</sup>

## Pathophysiology

SVT is divided into three categories based on their cause, however there are essentially no differences in their emergency management:

1. Reentrant tachycardias with an accessory pathway: Atrioventricular reciprocating tachycardia (AVRT) involves an accessory pathway outside of the SA or AV node. Is it the most common type of SVT seen in children,

representing 82% of arrhythmias occurring during infancy. WPW is an example of an accessory pathway resulting in a pre-excitation syndrome.

2. Reentrant tachycardias without an accessory pathway: This is the most common cause of SVT in adults; it accounts for ~15% of SVT in the pediatric age group, increases with age, and is rarely seen in infants.
3. Ectopic tachycardias: An ectopic focus outpaces the SA node.

## Clinical Presentation

Clinical presentation of SVT can vary greatly, especially between age groups. Infants will typically present with fussiness, irritability, restlessness, tachypnea, and ashen appearance, especially if the episode has lasted longer than a few hours.

Older children may present with complaints of fast heart rate, chest pain, shortness of breath, dizziness, lightheadedness, or syncope, or even just a “funny feeling” in their chest.<sup>4</sup>

Fortunately, most children compensate for SVT very well, notably

with episodes of SVT that are fast on and off. Their ability to compensate can lead to delays in seeking medical attention. It is not uncommon for healthy infants to tolerate SVT for 24 hours before being recognized. However, infants with underlying congenital heart disease lack cardiac reserve and may present earlier and be more critical upon presentation.

Unrecognized SVT can progress to congestive heart failure in 50% of children with structurally normal hearts. Therefore, it is of extreme importance for clinicians to consider SVT as complaints may be so nonspecific and general that children may present in florid heart failure if symptoms go unrecognized.

### Diagnosis

Diagnosis of SVT is made with a 12-lead EKG. Typically, an EKG will reveal a narrow complex (QRS duration <0.09 seconds) tachycardia with a rate over 180 bpm in children and adolescents and over 220 bpm in infants.<sup>2</sup> Roughly 50% of infants will present with a visible P-wave on EKG.<sup>4</sup>

It is important to distinguish sinus tachycardia from SVT, especially in infants and younger children. Sinus tachycardia will typically have a varying heart rate, while SVT is generally steady and unvarying. Another clue to differentiation is if P-waves are visible on EKG, they will be normal and upright in lead I and aVF, while they will be abnormal in SVT.<sup>4</sup> Due to baseline elevated heart rates in children, it can often be challenging to appreciate P-waves prior to QRS complexes. A good trick is to increase the speed of the EKG to 50mm/s to reveal P-waves that can get buried in QRS complexes.

### Management

As always, start with the ABCs, obtain access and obtain a rhythm strip as soon as possible. Due to the fast rate, consider speeding up the pace of the paper to help distinguish any P-waves. Per the PALS SVT algorithm, SVT should be quickly classified into stable or unstable SVT.

#### Approach to Unstable SVT

Although unstable SVT is relatively uncommon, the management is different than in a child with stable SVT. Unstable SVT is defined as the presence of

hypotension, altered mental status, or evidence of shock during an episode of SVT. Monitor for prolonged capillary refill, lethargy, mottled skin.<sup>5</sup> If a patient presents in unstable SVT, immediate synchronized cardioversion is required. Initial energy required for the first round of synchronized cardioversion is 0.5-1 J/kg. If unsuccessful, increase the energy to 2 J/kg. If a child continues with unstable SVT after this point, expert consultation is strongly advised to discuss alternative or additional antiarrhythmic medications.<sup>1</sup>

#### Approach to Stable SVT

Stable SVT is much more common. You may try vagal maneuvers while preparing for an IV. Valsalva, straining, breath-holding, blowing through a narrow straw (for older children), or asking children to attempt to blow the plunger in an empty syringe can be attempted. For infants, an ice slurry applied to the face, covering the eyes and bridge of the nose, is an acceptable method as an alternative to vagal maneuvers.

Orbital pressure should never be performed, as it is often performed incorrectly and can cause facial or orbital trauma and possible retinal detachment. Carotid massage is rarely effective.<sup>6</sup>

While vagal maneuvers are trialed, IV or IO access should be established, preferably in the right upper extremity—the rationale being that it is the closest vein to the heart. In the case of vagal maneuver failure, adenosine should be pushed through the IV or IO with an initial dose of 0.1 mg/kg (maximum dose 6 mg). If a second dose is required, increase the dose to 0.2 mg/kg (maximum dose 12 mg). Adenosine is an amino acid that is rapidly metabolized, with a half-life of 9 seconds. Therefore, adenosine must be delivered via a large-bore IV placed as close to the heart as

possible and should quickly be followed by a normal saline flush. This is best accomplished by using a 3-way stop cock. Adenosine briefly blocks conduction in the AV node causing disruption of any tachycardia circuit that relies on the AV nodal conduction

Recent studies have suggested that a higher initial dose of adenosine (0.2 mg/kg) in both infants and children reduces the risk of unsuccessful cardioversion by 35% with a NNT: 3 (Quail 2012). However, the most recent PALS update still recommends a stratified dose. During adenosine administration, a rhythm strip should be running to capture termination of SVT and return to normal sinus rhythm or to reveal the underlying dysrhythmia.

If adenosine fails, consult pediatric cardiology. SVT refractory to adenosine usually either abates with procainamide or synchronized cardioversion. IV procainamide has been found to be more effective than IV amiodarone for refractory SVT.<sup>10</sup> If requiring synchronized cardioversion, consider sedation and pain control as this is a very painful procedure.

#### Post-Resuscitation Management

If the clinician is successful in terminating an SVT, a decision must be made as to the next steps in management, specifically whether the patient is admitted to the telemetry unit or pediatric intensive care unit (PICU). Typically, patients with newly-diagnosed SVT should be admitted to the PICU for close observation. Those with known SVT may be able to be admitted to the telemetry unit, although consultation with a pediatric cardiologist is recommended. Although out of the scope of this review, long-term medical management of pediatric SVT may require daily antidysrhythmic medications. ★

### TAKE-HOME POINTS

- SVT is the most common pediatric arrhythmia, with a bimodal incidence peak in ages 6-9 and adolescents.
- SVT presentation varies greatly, especially between age groups. It is of vital importance for the clinician to always consider SVT with generalized, vague complaints.
- Rapid assessment of the patient is crucial, as is differentiating between stable and unstable SVT. Vagal maneuvers and adenosine are the initial resuscitative measures for patients in stable SVT, while synchronized cardioversion without delay is necessary for patients in unstable SVT.



# Hematuria in the ED

## EVALUATION AND MANAGEMENT

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### Case

A 64-year-old male presents to the ED for blood in his urine, which started this morning, followed by inability to urinate spontaneously in the afternoon. He complains of lower abdominal pain and fullness. He has a history of kidney stones more than 30 years ago, hypertension, diabetes mellitus (type 2), and chronic kidney disease stage 5 (CKD) for which he has been undergoing assessment for renal replacement therapy by outpatient nephrology for the past 4 months. He takes aspirin 81mg daily for CAD prevention; otherwise, he is not on blood thinners. On exam, he is hypertensive with otherwise normal vital signs. He appears uncomfortable, with distention and tenderness of the lower abdomen upon palpation. His genitourinary exam is normal. During the initial assessment, the nurse obtains a urinalysis and culture from a straight in-and-out catheter to drain his bladder, which provides immediate relief of his pain. The urine is grossly bloody upon inspection.

Urinalysis demonstrates 3+ blood and >50 RBC/hpf, but negative leukocyte esterase, negative nitrates, and no bacteria. His hemoglobin is at baseline, as are his BUN and creatinine, consistent with stage 3 CKD. A CT abdomen/pelvis without contrast reveals a large heterogeneous right renal mass with surrounding perinephric stranding, concerning for primary renal malignancy. There is also hyperdense material in a moderately distended bladder most likely reflecting blood clots, as well as scattered periaortic adenopathy which, given



**FIGURE 1.** Coronal CT of bladder with retained blood clots.

the context, raises concern for locally metastatic disease. He is unable to void spontaneously again, even after a fluid bolus.

### Introduction

Hematuria is a common complaint among patients presenting to the emergency department. Hematuria is an understandably distressing symptom. Thankfully, the distress experienced by patients with hematuria is often worse than the conditions diagnosed within the ED. Nonetheless, there are several emergent conditions the emergency physician should consider when evaluating a patient with hematuria.

Hematuria is defined by the American Urologic Association (AUA) as greater than three red blood cells (RBCs) visible under a microscope under a high-powered field (hpf) on a standard urinalysis (i.e., greater than 3 RBC/hpf).<sup>1</sup> In practice, most labs running urine specimens utilize flow cytometry rather than microscopy to automate the counting of RBCs for standard urinalyses. Hematuria is divided into two types: microscopic hematuria and macroscopic (or gross) hematuria. Microscopic hematuria can only be diagnosed by urinalysis. Macroscopic hematuria is any urine with visible blood

or blood-tinged coloring observed with the naked eye. Microscopic hematuria is a common incidental finding in the ED. Macroscopic hematuria is more often why a patient presents to the ED for evaluation.

### Microscopic Hematuria

There are few immediately life-threatening causes of microscopic hematuria, but the finding itself, especially in isolation, often warrants outpatient evaluation. Common causes of microscopic hematuria include urinary stones, infection, viral illnesses, trauma, vigorous exercise, sexual intercourse, menstruation, renal disease, or recent genitourinary instrumentation.<sup>1,2</sup> Microscopic hematuria in an ED patient experiencing pain or weakness rarely establishes a specific diagnosis by itself, but is often a guidepost in identifying those who will go on to be diagnosed with infection, stone, or even renal failure. Disposition of these patients will depend on the clinical context.

For adult patients with asymptomatic microscopic hematuria, only about 2% will go on to be diagnosed with serious urologic disease such as renal cell cancer, bladder cancer, or prostate cancer.<sup>3</sup> In the absence of any other identifiable

causes of microscopic hematuria, a patient should receive a referral to their primary care doctor for repeat testing of their urine. Persistent microscopic hematuria on two or more urinalyses when benign causes (such as sexual intercourse or exercise) are excluded warrants an outpatient urology referral for advanced imaging and cystoscopy (especially for patients over 35 years-old or with risk factors for urological malignancies such as smoking, chronic UTIs, occupational exposure to aniline dyes or benzenes). A nephrology referral is additionally recommended if significant proteinuria, cellular casts, or dysmorphic red cells are identified, all of which may suggest a glomerular etiology of hematuria.<sup>1, 4</sup>

***Discharged patients with asymptomatic microscopic hematuria should be referred to their primary care doctor for repeat urinalysis and subsequent referrals to urology and/or nephrology if the microscopic hematuria persists.***

### **Macroscopic Hematuria**

Macroscopic (or gross) hematuria is visible to the naked eye. Only 1 mL of blood is required to make a liter of fluid appear grossly hemorrhagic, so even a small amount of blood mixed into the urine can yield alarming results.<sup>4</sup> Conversely, the urinary tract can be a

source of life-threatening hemorrhage. Initial management of the unstable patient with gross hematuria is, like all critical patients, focused on the ABCs of resuscitation. Unstable patients with gross hematuria should be treated like other patients in hemorrhagic shock with early and emergent hemostatic transfusion. Causes of such life-threatening bleeding include renal tumors, arteriovenous malformations (AVMs), aortocaval fistulas, complicated ureterolithiasis and trauma, each of which are discussed below.

### **Emergent Causes of Gross Hematuria**

Renal tumors such as renal cell carcinomas (the most common renal cancer) or other malignancies with a predilection for renal metastasis (such as melanomas, lung, breast, gastric, and pancreatic cancers) may present with gross hematuria.<sup>5</sup> Such renal tumors with disordered angiogenesis may lead to uncontrolled hemorrhage should a vascular bed rupture into the renal collection system. Along with rapid urological consultation and evaluation, these patients may also benefit from a vascular surgery or interventional radiology consult for trans-arterial embolization of the tumor or even the entire kidney.<sup>6, 7</sup> Renal cell carcinoma is primarily treated with nephrectomy, which is usually curative when

performed prior to tumor metastasis.<sup>5</sup>

Renal arteriovenous malformations within the kidney can cause gross hematuria, and may be treated with a vascular or interventional radiology consultation for IR embolization.<sup>8</sup>

An aortocaval fistula is an abnormal connection between an aneurysmal abdominal aorta and the inferior vena cava (IVC). Increased arterial-level pressures in the low-pressure IVC causes increased venous congestion in the pelvic organs, including along the bladder wall. These bladder wall varices can then rupture, leading to gross hematuria.<sup>9</sup> Although rare, aortocaval fistulas are usually detected by CT, and warrant emergent vascular surgery consultation.<sup>8</sup>

Gross hematuria is present in up to 30% of patients suffering from a kidney stone.<sup>10</sup> Nephrolithiasis causing obstruction, especially when signs of infection are present on urinalysis, requires emergent urological evaluation and treatment. Additionally, urine and blood cultures should be obtained promptly and broad-spectrum antibiotics rapidly initiated for the unstable patient presenting with sepsis secondary to an infected kidney stone. Patients with suspected obstruction or infection should receive CT imaging for urological planning purposes, as should patients with suspected first-time kidney stones. CT can also be a helpful tool in considering alternative diagnosis, particularly in the sick or elderly patient presenting with undifferentiated abdominal or flank pain.<sup>10</sup> Rapid point of care ultrasound may also be used to determine the laterality of the stone causing obstruction by evaluating for hydroureter and/or hydronephrosis.<sup>11</sup> The sensitivity and specificity of ultrasound are 84% and 53% respectively, compared to CT, which has a sensitivity and specificity of 95% and 98% respectively.<sup>11</sup>

Traumatic causes of hematuria should be evaluated with a retrograde urethrogram to rule out urethral injury, usually followed by CT with IV contrast to evaluate for other injuries.<sup>12</sup>



**FIGURE 2.** Sagittal CT of bladder with clots and distention.



**FIGURE 3.** Axial CT of right renal mass.

**After relieving an obstruction, patients may have post-obstructive diuresis and, in rare cases, this can progress to hypovolemic shock.**

### Urinary Obstruction

Hemorrhagic debris in the dependent portions of the bladder will eventually clot and obstruct the bladder neck, leading to acute urinary retention. Placement of a large-diameter three-way foley will provide immediate relief to the obstructed patient. Following placement of a three-way catheter, continuous bladder irrigation (CBI) should be initiated to wash out residual clot from the bladder to avoid further obstruction. The decision to initiate CBI is typically done at the recommendation of a urologist. In addition, strictly measuring net urine output in a patient treated for urinary retention is necessary to identify post-obstructive diuresis, a known and potentially fatal complication following relief of urinary obstruction.<sup>13</sup> Post-obstructive diuresis is an abnormal amount of urinary output following bladder decompression (usually over 200ml in the first two hours) which can lead to electrolyte derangements, dehydration, hypotension, and even shock.

### Non-emergent Causes of Gross Hematuria

Other causes of gross hematuria include urinary tract infection (UTI), uncomplicated nephrolithiasis, urethritis/sexually transmitted infection, schistosomiasis (the most common cause of gross hematuria in underdeveloped countries), and iatrogenic causes.<sup>8</sup> A 1987 study demonstrated foley catheterization only induced hematuria in 17% of patients, and even then the

amount of hematuria never exceeded 3 RBCs/hpf.<sup>14</sup> Conditions which may be mistaken for hematuria by patients include menstruation as well as the ingestion of substances which may turn the urine red such as beets, rhubarb, berries, food colorings, and medications including nitrofurantoin, rifampin, phenazopyridine (commonly sold over-the-counter under the brand names Azo or Pyridium for the treatment of suspected UTI), hydroxychloroquine, and iodine.<sup>4</sup> Careful history-taking can help identify these less emergent (or entirely benign) causes, thus guiding efficient management and disposition of the stable patient presenting with hematuria. Laboratory evaluation of hematuria should include complete metabolic panel (CMP) including blood urea nitrogen (BUN) and creatinine, complete blood count (CBC), coagulation studies if the patient is on blood thinners, and of course urinalysis to evaluate for the presence of infection, proteinuria, and casts.

### Imaging

The decision to image an otherwise stable patient with hematuria in the ED is made on a case-by-case basis. Interestingly, the American College of Radiology (ACR) Appropriateness Criteria for hematuria states that a CT urogram is “usually appropriate” for any adult patient presenting with gross hematuria.<sup>15</sup> The ACR’s criteria does not qualify this recommendation or attempt to sub-classify which patients with hematuria may benefit from advanced imaging more than others. There is no unifying consensus regarding the most appropriate imaging criteria or even

### TAKE-HOME POINTS

- Gross hematuria in the unstable patient can be a life-threatening cause of hemorrhagic shock.
- Urinary obstruction should be treated with urology consult and placement of a three-way catheter for decompression and continuous bladder irrigation (CBI). Watch out for post-obstructive diuresis.
- For imaging, CT urogram is appropriate for the otherwise stable patient with macroscopic hematuria—otherwise consider CT abdomen/pelvis with IV contrast.
- Ill patients or those with significant renal impairment or anemia require admission for further work-up.
- Patients otherwise appropriate for discharge should receive urology referral for follow-up as a high percentage of patients will go on to have malignancy.



the most appropriate modality.<sup>16</sup> Most ED physicians choosing to image their patient with hematuria opt for a CT of the abdomen/pelvis with IV contrast to evaluate for a broader array of diagnoses, that is, if they are not suspecting kidney stone, in which case a CT renal protocol without contrast is the study of choice. However, in the otherwise stable patient with gross hematuria in which there is low suspicion for other intra-abdominal pathology (such as an abdominal aortic aneurysm), a CT urogram will provide better resolution of the renal and urothelial tissues to evaluate for malignancy, although the sensitivity is higher for renal and other upper urinary tract tumors than compared to lower tract tumors such as bladder cancer.<sup>16</sup>

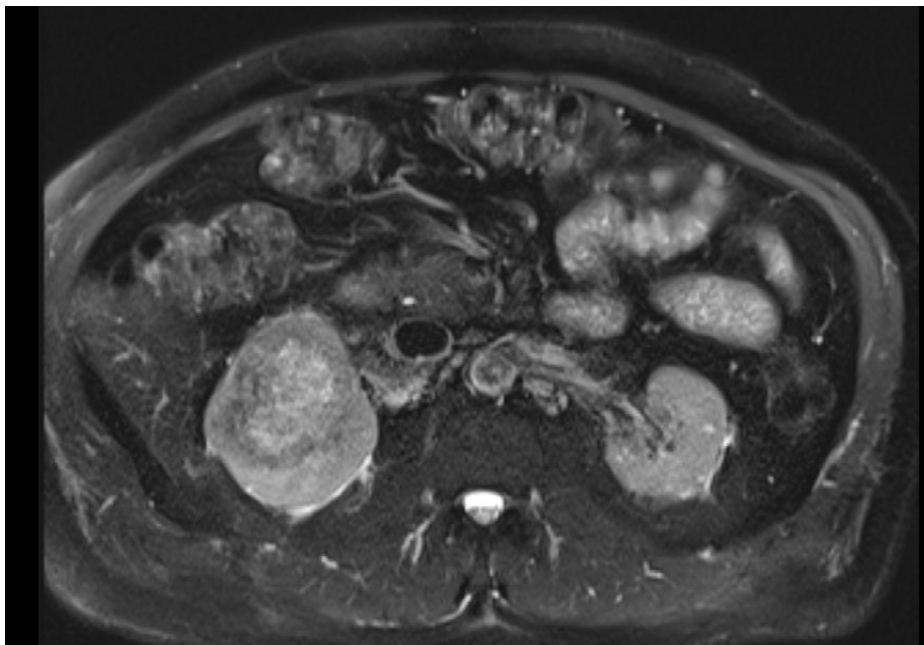
### Disposition

Symptomatic patients with a significant decline in their renal function, decreases in their hemoglobin or hematocrit, proteinuria, red blood cell casts, or muddy brown casts should prompt admission for further work-up. The stable patient who presents due to a limited number of episodes of hematuria, with stable renal function and blood counts, and who can void spontaneously can be discharged to home with outpatient urology referral. A similar patient found to have a UTI can

be discharged home with oral antibiotics, but should be instructed to follow up with their primary care provider to ensure resolution of the hematuria after successful treatment of their UTI. In one study of over 4000 patients presenting for evaluation of hematuria, 18% of patients with gross hematuria were found to have malignancy.<sup>17</sup> Therefore, if benign causes have been ruled out or treated, referral to urological specialist is needed for further imaging or cystoscopy to rule-out malignancy.

### Case Resolution

*Urology is consulted. A 3-way Foley catheter is inserted, and continuous bladder irrigation is started. The patient is admitted for acute urinary obstruction and continued hematuria. He undergoes MRI for evaluation of the renal mass, which reveals 7.7cm right renal cell carcinoma, but fortunately no evidence of locally metastasis. Several days later he undergoes radical nephrectomy of the right kidney and pathology confirms diagnosis of clear cell carcinoma. Due to his CKD, he requires initiation of hemodialysis and receives several sessions before being discharged four days post-operatively. He continues on hemodialysis, grateful for his care, and has returned to managing his construction business. ★*



**FIGURE 4.** MRI follow-up of right renal mass.

## A WORD REGARDING PEDIATRICS

A urinalysis is often the only laboratory test initially obtained during the evaluation of a child in the ED, but it can provide an important window into a child's renal function.

Children presenting with hypertension (above the 95th percentile for age), decreased urine output, and edema who are found to have proteinuria or cellular casts in combination with hematuria on a UA should receive a robust work-up including complete blood count, complete metabolic panel with renal profile, renal ultrasound, pediatric nephrology consult, and admission.<sup>18,19</sup>

The differential diagnosis for such a presentation contains significant, life-threatening diseases: post-infectious glomerulonephritis, Henoch-Schonlein purpura (HSP), hemolytic-uremic syndrome (HUS), membranoproliferative glomerulonephritis, IgA nephropathy, and focal segmental glomerulosclerosis (FSGS).

These conditions affect adults as well, but in the pediatric population where less laboratory evaluation is usually performed, an abnormal urinalysis in isolation could alter the entire diagnostic work-up in the right clinical context. However, an otherwise well-appearing child with normal vital signs with isolated microscopic hematuria should receive a referral to their pediatrician to have repeat urinalysis performed, similar to the recommendation from the AUA for adults.<sup>18</sup>

# Isolated Traumatic Gallbladder Hematoma Caused by Electric Scooter Accident

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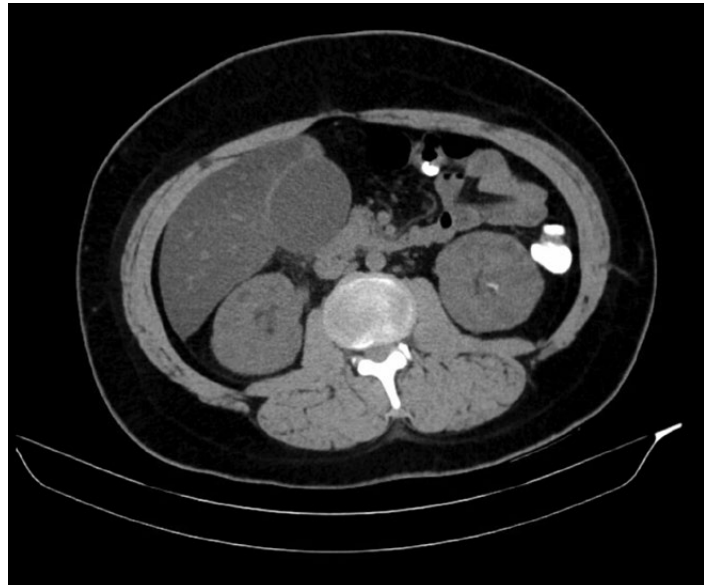
## Case

A 22-year-old male presented to the emergency department after an accidental fall from a motorized scooter going 20 miles per hour without wearing a helmet. The patient states he hit a pothole, was thrown over the handlebars of his scooter, and had loss of consciousness after hitting the pavement.

On arrival, his vital signs were normal and he appeared alert and oriented to person, place, time, situation. He denied any alcohol or drug use. He appeared uncomfortable and was actively vomiting while sitting on the stretcher. The vomitus was yellow in color. A cervical collar was immediately placed due to the reported mechanism of injury. The patient was moving all four of his extremities with no difficulty and following commands. He had a right forehead abrasion, no external chest trauma, lungs clear to auscultation bilaterally, abdomen soft with no evidence of Cullen or Grey Turner sign. Elicited on exam were a positive Murphy sign and epigastric tenderness. A bedside eFAST exam was performed and was negative. All other systems were unremarkable.

Laboratory values showed hemoglobin 13.4, creatinine 2.02, sodium 159, potassium 4.1, lactic acid 2.4, ALT 52, AST 71, total bilirubin 0.4, lipase 19, troponin < 0.01, and a small amount of blood in his urine. CT abdomen with contrast showed a dilated gallbladder with wall thickening and possible cholecystic fluid versus hemorrhage (Figure 1). CT head revealed a small isolated subarachnoid hemorrhage (Figure 2). A follow-up right upper quadrant ultrasound showed a heterogenous intramural fluid collection with associated wall thickening consistent with post-traumatic gallbladder hematoma (Figure 4).

General surgery was consulted and recommended admission to their service for repeat CBC, repeat imaging, and serial abdominal examinations. On hospital day 2 abdominal tenderness resolved and diet was advanced as tolerated. Repeat CT abdomen (Figure 3) and CT head showed no interval changes. On hospital day 3 liver functions tests increased to



**FIGURE 1.**



**FIGURE 2.**

ALT 76, AST 92, total bilirubin 1.5. On hospital day 4 liver function tests and bilirubin levels normalized. Hemoglobin and creatinine remained stable throughout the hospital course, and the patient was discharged with outpatient general surgery clinic follow-up.



FIGURE 3.



FIGURE 4.

## Case Discussion

Gallbladder injuries are most commonly caused by penetrating trauma as opposed to blunt trauma. Only 2% of gallbladder injuries are isolated, while 49% are associated with additional injuries with secondary hemodynamic instability. Traumatic isolated gallbladder hematomas are rare, with only five previously reported cases found on review of the available literature.<sup>1-5</sup> The case above is the first reported to have been caused by an electric scooter accident. Gallbladder hematoma diagnosis is suggested by CT or right upper quadrant ultrasound. Definitive diagnosis is made surgically.<sup>1</sup>

Again, due to the presentation with isolated gallbladder hematoma being rarely reported, no definitive management guidelines exist. The cases discovered in our literature review and the case above were managed by serial abdominal examinations, serial imaging with CT or ultrasound, and ruling out possible complications of gallbladder injury. More than 90% of gallbladder trauma is associated with additional visceral injury. The common complications associated with gallbladder hematoma are duodenal hematoma and associated abdominal traumatic injuries. Duodenal hematoma must be ruled out when evaluating a patient for gallbladder hematoma because duodenal hematoma can lead to perforation and gastric outlet obstruction.<sup>2</sup>

## Conclusion

Isolated traumatic gallbladder injury is extremely rare and must be thoroughly evaluated due to the complications associated with traumatic gallbladder injury. Complications such as duodenal hematoma and associated traumatic abdominal injuries must be ruled out. No definitive treatment guidelines exist but previous case reports recommend serial abdominal examinations and repeat imaging with CT and ultrasound.<sup>1,4</sup> If the patient is hemodynamically unstable or shows no improvement in their condition, the definitive treatment is cholecystectomy or percutaneous drainage.<sup>1</sup>

Our patient had a 4-day hospital course. Hemoglobin remained at admission baseline throughout the hospital stay. Repeat CT abdomen did not reveal any significant changes in gallbladder appearance, and the patient was discharged in stable condition without complaint of abdominal pain. The patient was discharged home with outpatient surgery follow-up. ★

## TAKE-HOME POINTS

- Isolated gallbladder injuries are incredibly rare, so high suspicion must be maintained for additional injuries.
- The most common injury accompanying a gallbladder hematoma is a duodenal hematoma.
- Duodenal hematoma can result in small bowel obstruction.
- The imaging studies of choice for diagnosis of gallbladder hematoma are CT or right upper quadrant ultrasound.



# Uncontrolled Hyperglycemia as a Cause of Choreiform Movements

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Although rare, chorea-hyperglycemia-basal ganglia syndrome is a presenting symptom of uncontrolled diabetes mellitus. This condition is seen in elderly women, typically of Asian descent, with uncontrolled diabetes mellitus. Chorea-type movements have a broad differential diagnosis, including emergent and life-threatening conditions such as hemorrhage, infarction, malignancy, and infection. Less common causes of chorea movements include SLE, Huntington's chorea, and Wilson's disease. Patients classically present with hemichorea; however, bilateral chorea is also possible. The majority of patients have upper and lower extremities affected, with a smaller group having facial involvement. The treatment is decreasing the hyperglycemia and administering antipsychotics or benzodiazepines.

## Case

A 61-year-old female with no previous past medical history presented to the emergency department with the chief complaint of involuntary movements. The patient reported 2 days of uncontrollable movements over the right side of her body. There was no associated numbness, focal weakness, visual changes, headaches, or speech difficulties.

Physical examination revealed choreiform movements over the right upper and lower extremity. Neurological examination was otherwise benign without focal neurological deficit or sensation deficits. Vital signs were unremarkable. Laboratory workup was significant for a glucose of 396 without an anion gap metabolic acidosis and a sodium of 130. CT of the head showed hyperdensities in the basal ganglia bilaterally but greater on the left, compatible with calcifications.

The patient was admitted for further evaluation by neurology including an MRI, which showed chronic ischemic changes, most pronounced in the region of the

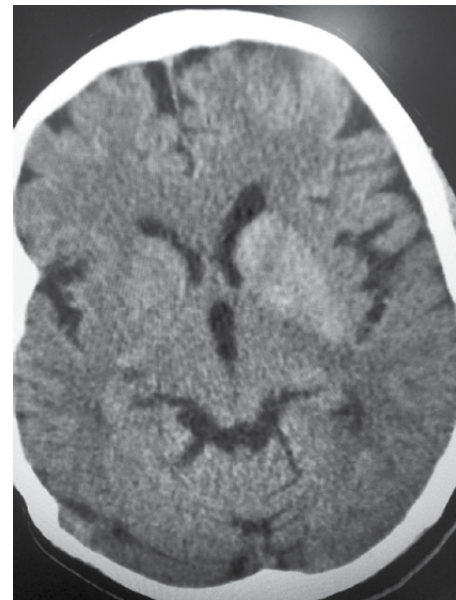
left basal ganglia. Further laboratory workup showed an HbA1c of 14%. Neurology and endocrinology were consulted, who provided the patient with the diagnosis of chorea-hyperglycemia-basal ganglia syndrome (C-H-BG) caused by uncontrolled diabetes mellitus. The patient was started on insulin, valium, and was discharged in stable condition with neurology follow up.

## Discussion

Chorea is defined as irregular, unpredictable, and brief jerky involuntary movements. Hemiballismus is defined as ballistic flinging movements. Both can be the result of central nervous system infections, neoplasm, intracranial hemorrhage, neurodegenerative disorders, drugs, toxins, and metabolic imbalances. The mechanism behind the cause of C-H-BG is unknown, although some theories have been postulated that include dopamine hypersensitivity or hyperactive dopaminergic states in postmenopausal women. Some debate that the cause is a direct effect of hyperglycemia on metabolism in the cerebrum, with hypoperfusion in the corpus in the striatum. Hyperglycemia is thought to cause favoring of anaerobic metabolism at a cellular level, which depletes gamma-aminobutyric acid (GABA) due to a substrate of GABA being quickly depleted in an anaerobic state. GABA functions to inhibit dopaminergic neurons in the nigrostriatal system and therefore a hyperglycemia-induced inhibition of GABA may lead to a hyperactive dopaminergic state, leading to the onset of chorea movements.

CT imaging shows hyperdensities in the striatal region of the caudate nuclei and putamen contralateral to the extremities affected by chorea. In cases of bilateral chorea movements, bilateral hyperdensities are seen. MRI classically shows high signal intensity of the basal ganglia lesion on T1 weighted MRI.

Treatment surrounds normalization of blood glucose. Patients can also be treated



with antipsychotics such as clozapine, risperidone, and olanzapine or with medications with GABAergic properties such as benzodiazepines. In one study, a group of 16 patients were treated only with blood sugar control and had complete resolution of their chorea. In another case series, 74% of patients had complete resolution of their chorea within 10 months. A majority of patients reach full recovery within 6 months.

## Case Resolution

The patient was discharged with outpatient follow-up. At follow-up with neurology, the patient's choreiform movements had nearly resolved. ★

## TAKE-HOME POINTS

- Uncontrolled diabetes mellitus can present with abnormal choreiform movements.
- The differential diagnosis for choreiform movements includes Huntington's disease, Sydenham chorea, Wilson's disease, malignancy, SLE, hemorrhage, thyroid dysfunction, and drug-induced chorea.
- Patients affected by C-H-BG are predominantly elderly women with poorly controlled diabetes.

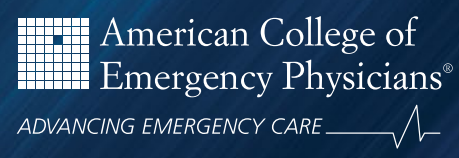
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# Use of Bedside Ultrasound in Diagnosing Boerhaave Syndrome

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Esophageal rupture is a rare condition that is recognized as one of the most fatal gastrointestinal injuries. Time to diagnosis is an independent predictor of mortality in acute esophageal rupture making this a can't miss diagnosis for emergency physicians. Here, we describe a rare presentation of Boerhaave syndrome and the utility of ultrasound in the diagnosis.

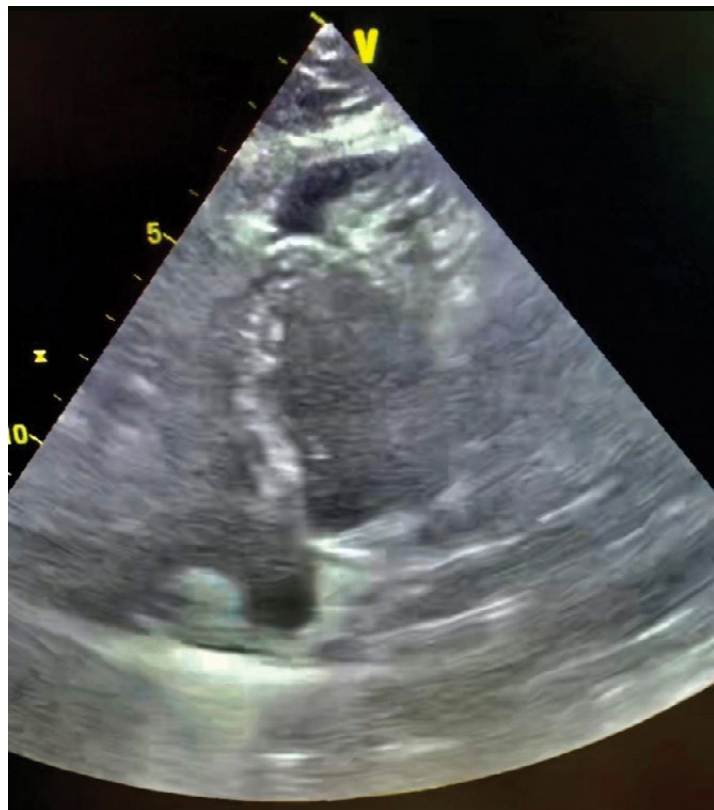
## Case

A 41-year-old female with a history of congenital CMV, developmental delay, VSD, esophageal stricture, and episodes of food impaction presented to the emergency department with symptoms of abdominal pain and nausea. The patient reportedly complained of achy non-radiating epigastric pain and nausea after choking on mashed potatoes the night before. Overnight her symptoms persisted gradually involving upper back pain and chest pain, prompting ED evaluation. The patient denied fever, chills, or vomiting.

On arrival, the patient was afebrile with a BP 96/60, HR 134, RR 24, and oxygen saturation 94% on room air. Chest X-ray was concerning for bilateral pleural effusions and possible infiltrate. Labs showed Na of 134 mmol/L, CO<sub>2</sub> of 18 mmol/L, anion gap of 19 mmol/L, lactic acid of 7.2 mmol/L, troponin of .046 ng/mL, WBC 29.3 K/uL, and hemoglobin of 10.8 g/dL. The patient was given ampicillin/sulbactam and 2 L of normal saline.

She was transferred to the ICU with a working diagnosis of aspiration pneumonia.

**FIGURE 1.** Point-of-care cardiac ultrasound demonstrated a large pericardial effusion with a hyperechoic fluctuant mass on the pericardium.



**Boerhaave syndrome, first described by Herman Boerhaave in 1724, is a spontaneous transmural perforation of the esophagus usually due to forceful emesis.**

On arrival to the ICU, the patient was ill-appearing with improved blood pressure but worsened dyspnea and hypoxemia. Physical exam was significant for increased work of breathing, diaphoresis, cold extremities, and delayed capillary refill. Chest X-ray was unchanged from earlier. Antibiotics were broadened and BiPAP initiated. Due to concern for cardiogenic shock a bedside ultrasound was performed. The cardiac ultrasound appeared distorted: a rightward-deviated cardiac silhouette, a seemingly large pericardial effusion, and a hyperechoic

fluctuant mass on the pericardium (Figure 1). Due to the unusual anatomy it was difficult to determine whether the fluid was a pericardial effusion or a pleural effusion. Echo findings were discussed with the cardiology team and the decision was made to perform a thoracentesis and reassess clinical status. A left-sided thoracentesis drained murky brown pleural fluid. During the procedure, the patient began vomiting and became acutely hypoxic requiring intubation for airway protection. Based on the appearance of the pleural fluid, persistent pericardial



effusion on repeat ultrasound, and development of chest wall crepitus the patient was sent for CT imaging for suspected esophageal injury. CT of the chest showed air within the subcutaneous tissue and mediastinum suspicious for acute esophageal rupture (Figure 2).

### Diagnosis

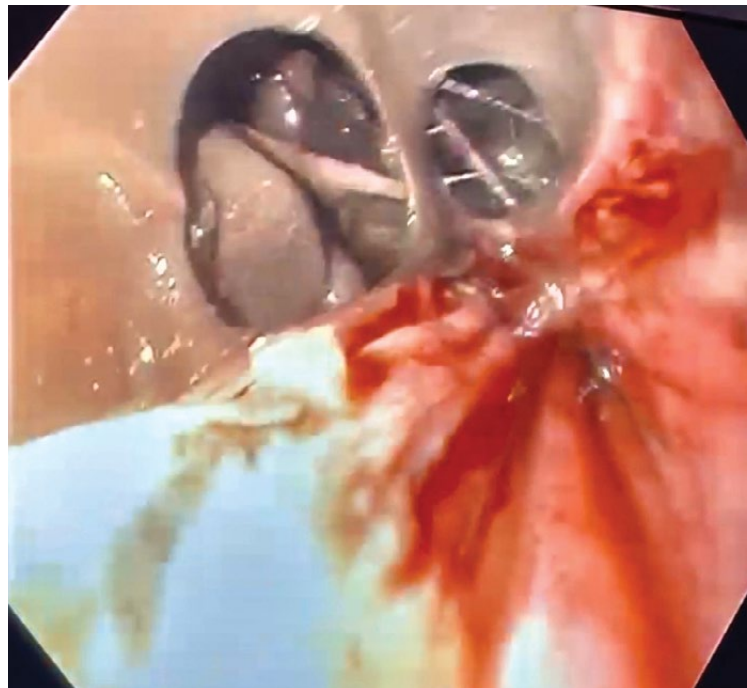
Cardiothoracic surgery was consulted and the patient was immediately taken to the OR for Esophagogastroduodenoscopy and esophageal repair. A 10 cm esophageal tear was identified intraoperatively (Figure 3).

### Discussion

Although the most common cause of esophageal rupture is iatrogenic, accounting for nearly 85% of reported cases, spontaneous rupture is the second most common cause.<sup>1</sup>

Boerhaave syndrome, first described by Herman Boerhaave in 1724, is a spontaneous transmural perforation of the esophagus usually due to forceful emesis.<sup>1</sup> Other less common culprits are trauma, defecation, seizures, childbirth and weightlifting.<sup>1</sup>

A common algorithm used to diagnose Boerhaave syndrome is Mackler's triad: vomiting, chest pain, and subcutaneous



**FIGURE 3.** Endoscopy image of large esophageal perforation.

emphysema. Unfortunately, only 14% of patients with Boerhaave syndrome present with this classic triad.<sup>2</sup> Delayed and sometimes missed diagnosis can occur, often due to non-specific and vague clinical presentation as seen in this case.

### Case Conclusion

Our case presents an unusual presentation of Boerhaave syndrome. Not only did our patient's symptoms

begin after eating soft food, but emesis and development of crepitus was a very late finding. Another layer of complexity came from the inability of the patient to provide a history herself secondary to her cognitive disability due to congenital CMV. However, this case demonstrates the importance of a high index of suspicion in patients with symptoms after choking. Although aspiration pneumonia is more common, esophageal injury should remain high on the differential especially in the setting of worsening respiratory status and overall patient decline. Another important aspect of this case is the importance of thorough chart review, which in our patient unveiled her history of esophageal stricture.

Lastly, CT imaging and esophagography have proven utility when diagnosing esophageal perforation, however emergency ultrasound is a fast, portable and non-invasive imaging modality that can aid diagnosis.

Ultrasound is routinely used by emergency physicians, primarily in those presenting with undifferentiated shock. Although ultrasound findings are not highly specific, when coupled with a high index of clinical suspicion it can be used to support diagnosis and expedite surgical intervention in patients with acute esophageal rupture. ★



**FIGURE 2.** Axial CT of the chest revealed pneumomediastinum.

Initially marketed as an atypical antidepressant for adults, bupropion gained popularity for its use in tobacco cessation and for its lack of sexual side effects commonly seen with other typical antidepressants.



# Bupropion Toxicity

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### Case Report

*An 18-year-old female with a past medical history of ADHD, depression complicated by past suicidal ideation, anxiety, and polysubstance use presented to the emergency department after an intentional polypharmacy ingestion in a suicide attempt. It was reported that the patient took a mixture of 20 pills including dicyclomine, meclizine, and bupropion 150mg XL.*

*On arrival the patient was somnolent, hallucinating, and unable to follow commands. She demonstrated inducible 3-5 beat ankle clonus with hyperreflexia. Her eye exam showed dilated pupils, nystagmus, and roving eye movements. Her workup on admission was notable for a normal metabolic panel and negative acetaminophen, ethanol, and salicylate levels. Her urine drug screen was positive for amphetamines, benzodiazepines, and cannabinoids. The patient's EKG showed sinus tachycardia with a rate of 146, a normal QRS, and a prolonged QTc of 585 ms.*

*Soon after admission, the patient had generalized tonic-clonic activity lasting about 2 minutes which terminated with 2mg of lorazepam.*

### Overview

Although this patient's suicide attempt was complicated by the mixture of anticholinergic substances (meclizine and dicyclomine), her ingestion of bupropion (Wellbutrin) posed the most significant risk for morbidity and mortality. Initially marketed as an atypical antidepressant for adults, bupropion gained popularity for its use in tobacco cessation and for its lack of sexual side effects commonly seen with other typical antidepressants.<sup>1</sup> It exerts its pharmacologic action through reuptake inhibition of norepinephrine and dopamine, however its chemical structure bears a striking resemblance to other commonly encountered drugs in the emergency



department: methamphetamine and bath salts.<sup>1,2</sup>

Bupropion is merely a side chain or two away from being structurally identical to the dreaded “bath salts” or synthetic cathinones, and a not-so-distant cousin to the laboratory-created methamphetamines.<sup>2</sup> It should not come as a surprise that when screening urine for illicit drugs, bupropion frequently produces a false positive for amphetamines, given this structural similarity. As the popularity of bupropion increases due to its efficacy and favorable side effect profile, it is paramount that the emergency physician knows the behavior of the drug in toxicity and the appropriate disposition for these patients.

### **Altered Mental Status and Vital Sign Derangements**

As expected with a medication eerily similar to potent stimulants, alterations in mental status and vital signs are common in the early post ingestion phase.<sup>3,4</sup> Classically, patients will present with tachycardia, tachypnea, hyperreflexia, and clonus. Mentation can range on a spectrum from acutely agitated to sedation requiring emergent airway intervention. Auditory and visual hallucinations may occur.<sup>2</sup> These changes in mentation and vital signs are not specific to bupropion and can be seen in many other stimulants and primary psychiatric disorders as well. Thus, it can initially be difficult to determine if the patient is exhibiting an acute intoxication from a common stimulant (eg, methamphetamines or ecstasy) versus a potentially life-threatening overdose. As with most overdoses, it is recommended that the patient undergo continuous cardiac monitoring and EKG evaluation. With potential agitation, be certain to also ensure the safety of the staff.

### **Seizures**

Classically, seizures in bupropion toxicity are tonic-clonic in nature.<sup>4</sup> Although the specific mechanism of seizure induction is unknown, research suggests that the metabolite 6-hydroxybupropion is the likely causative agent. The presence of seizures may suggest that the patient is suffering

from bupropion toxicity, as opposed to other sympathomimetics.<sup>2</sup> These seizures are dose-dependent and thus a higher ingested dose corresponds to an increased risk of neurologic effects. Benzodiazepines (eg, lorazepam 2-4mg per dose) are the mainstay of treatment for seizures in this setting. The authors recommend utilizing a fixed dose regimen rather than a symptoms or vital sign-triggered regimen like the Clinical Institute Withdrawal Assessment (CIWA). These protocols may lead to over sedation and airway compromise because the patient may not be able to accurately convey their symptoms and vital sign derangements can be present until the toxicity has resolved. Barbiturates can also be used for more long-acting seizure treatment or prophylaxis. However, they also carry an increased risk of over-sedation requiring intubation.<sup>2,4</sup>

### **Cardiac Toxicity**

The final manifestation of bupropion toxicity is arguably the most anxiety provoking and the most fatal if experienced. Although relatively rare, QTc and QRS prolongation leading to unstable ventricular dysrhythmias can be seen in overdose. Unlike tricyclics and other medications that cause QRS prolongation via sodium channel blockage, bupropion actually disrupts the myocardial gap junctions causing progressive ventricular dysfunction, hypotension, and irreversible cardiogenic shock.<sup>2,4</sup> Due to this lack of sodium channel blockade, administration of sodium bicarbonate is almost always ineffective in correcting QRS widening due to bupropion overdose.<sup>3</sup> Bupropion can also create a blockade of outward rectifying myocardial potassium channels, prolonging the QTc. Careful optimization of the patient’s electrolytes, namely calcium, magnesium, and potassium, should be performed to minimize any predisposition to dysrhythmia. Vasopressor support can be used for any cardiogenic shock, with the expectation that there may not be much, if any, clinical improvement. Medications like norepinephrine or inotropic agents will not solve the underlying issue of poor electrical

conduction of the heart. If recalcitrant cardiac dysfunction is present, the patient may benefit from intralipid administration or venoarterial extracorporeal membrane oxygenation (VA ECMO); however, prognosis is grim.<sup>2,3</sup>

### **Disposition**

Although the therapeutic ceiling of bupropion is 450 mg, the decision to admit or observe a patient has little to do with the dose ingested and more with the formulation. Any extended release (eg, Wellbutrin XL) overdose of greater than 600 mg, regardless of intent, requires an admission for twenty-four hours as seizures have been reported up to one day after ingestion.<sup>1,3</sup> An overdose of immediate release formulations can usually be safely discharged after four to six hours of observation in the emergency department, as they have a lower risk of delayed toxicity.<sup>3</sup> ★

### **Acknowledgements**

*The authors would like to thank Dr. Nathaniel Mann for his guidance and assistance in editing.*

## **TAKE-HOME POINTS**

- Wellbutrin, a common norepinephrine and dopamine reuptake inhibitor used for depression and smoking cessation, poses a significant risk to patients in overdose.
- Wellbutrin is structurally similar to stimulant drugs of abuse, and most closely resembles synthetic cathinones.
- Common toxic effects of bupropion include altered mental status, tachycardia, seizures, and less commonly, dysrhythmias and cardiogenic shock.
- Treatment revolves around frequent vital sign monitoring, EKG evaluation, seizure prophylaxis or treatment with benzodiazepines or barbiturates, and intralipid or VA ECMO if refractory cardiogenic shock ensues.
- As always, consult your local toxicologist or poison control center with questions regarding any overdose.



# Crawling In My Skin

## An Ultrasound Teaching Case

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### Case

A 20-year-old female patient with no significant past medical history presented to our emergency department (ED) with itchy scalp lesions after returning from a honeymoon to Belize. Over the past 3 weeks, she had presented to the clinic several times, each time being treated for “bug bites” with topical steroid cream. At her most recent clinic visit, the patient described feeling strange sensations like wriggling under her skin. She was provided a psychiatry referral out of concern for delusional parasitosis.

The morning prior to presentation, the patient’s partner cleaned her scalp and noted something moving under her skin (Figure 1-B). Bedside ultrasound performed in the ED demonstrated two large foreign bodies within the scalp, with internal movement (Figure 1-A and video). We discussed treatment options with the patient, including watchful waiting, occlusion of the breathing pore, or procedural excision. After discussing the risks and benefits, the patient elected for bedside excision, which was completed using ultrasound guidance (see below) (Figure 1-C). The patient tolerated the excision well. Specimens were sent to the pathology lab for analysis and were consistent with *Dermatobia hominis* (human bot fly).

### About the Bot Fly

The human bot fly (*D. hominis*) is endemic to Central and South America, transmitted primarily via mosquito bite,<sup>1</sup> leading to cutaneous myiasis. There is a distinct species of bot fly (*Cordylobia*)

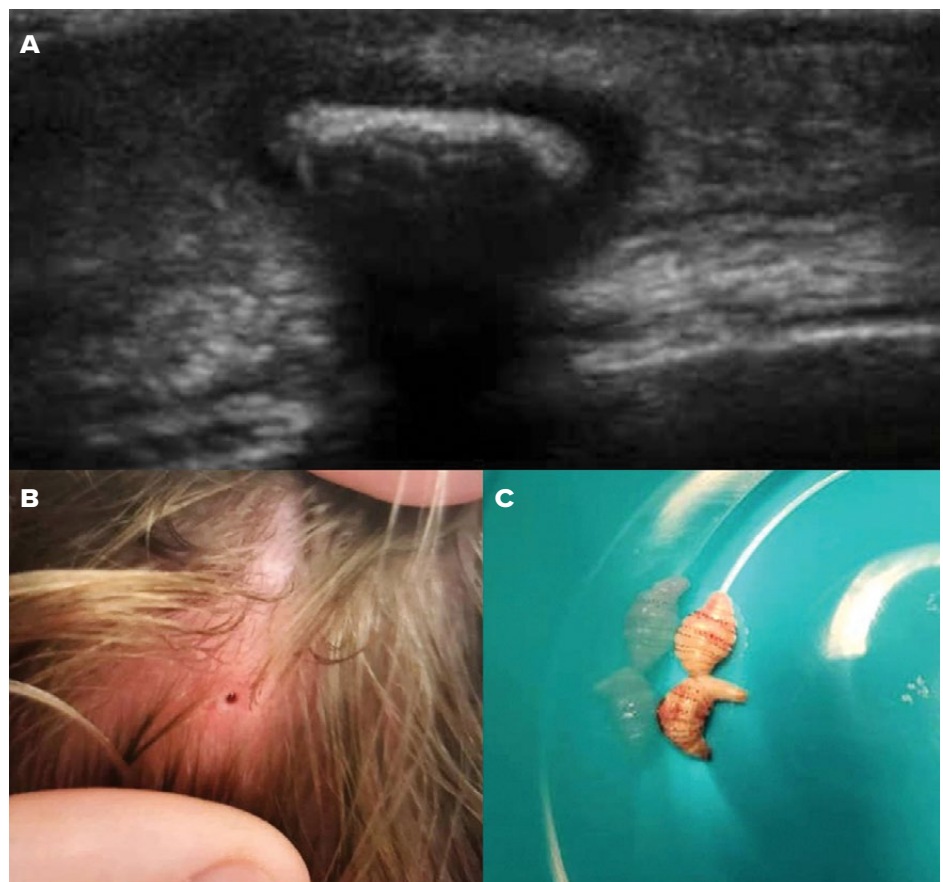
that is endemic to the African tropics. Therefore, patients returning from an endemic area may have appropriate concern when presenting to your ED with feelings of something moving under their skin, and you should have a higher index of suspicion for a parasitic etiology in such patients.

The bot fly life cycle begins with the female laying eggs in a mosquito or other vector insect. The eggs are harbored in the insect’s abdomen and released into a host during feeding. After seeding of the host, self-expulsion of the larva typically occurs after 5-10 weeks. Disclosing this time frame to patients is important when discussing expectant management.

There are many different species of bot fly that primarily affect other mammals, and rarely, if ever, transmit to humans.<sup>2</sup>

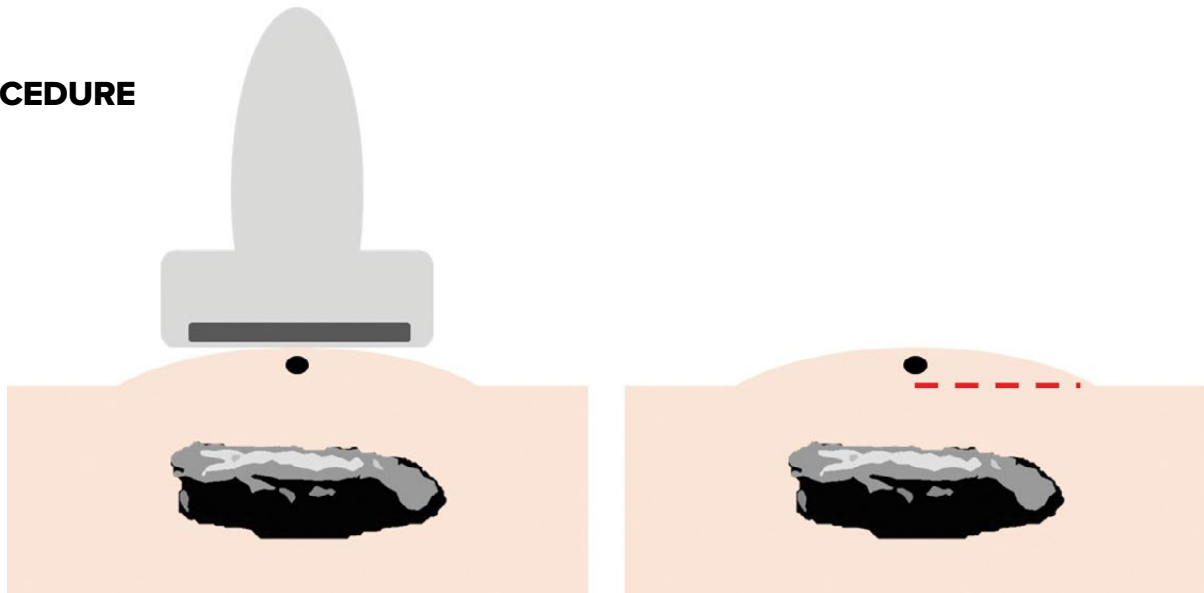
### About the Procedure

Prior to incision, the site of each larva was treated with lidocaine-tetracaine-epinephrine (LET) gel for topical anesthesia, with a secondary benefit of stunning the larva (larvae that are not adequately weakened either pharmacologically or via asphyxiation are likely to resist removal via use of their spicules, see discussion below). A linear probe was used to determine the long axis of the larva and optimize the small (less than one cm) incision) made to deliver



**FIGURE 1. Cutaneous myiasis of the scalp. A.** Still of the ultrasound image that was used to identify one of the larvae (arrow) at the time of initial evaluation in the emergency department. **B.** Appearance of the patient’s scalp with prominent breathing pore (arrow). **C.** Two live, intact bot fly larvae (arrow) post excision.

## PROCEDURE



**FIGURE 2. Approach to bedside excision.** Figure above demonstrates the larva embedded in the skin with an overlying pore (dark circle). Linear ultrasound probe was used to identify the long axis of the larva and a small skin incision (dashed red line) was made.

**Video: Cutaneous myiasis of the scalp, ultrasound video clip.** This video demonstrates the bot fly larva (arrow) embedded in the patient's scalp. The wriggling movement in the video narration represents the larva's spicules, which it uses to grip the host skin in order to resist expulsion. See online edition for US video.

each larva. Once the incision was made, gentle pressure was applied, and each larva was expelled quickly. The wounds were irrigated with sterile saline and left open to heal by secondary intention with plan for PO antibiotics on discharge.

### Management Options to Offer the Patient Presenting with Cutaneous Myiasis

Conventional teaching for management of cutaneous larvae has generally been to asphyxiate the larvae. Asphyxiation encourages the larvae to leave the skin voluntarily or weakens it to facilitate manual removal by squeezing the larvae out.

Attempting to remove the larvae without prior asphyxiation or stunning is not recommended, as the larvae unfortunately resist removal by digging into host skin with their spicules (see ultrasound video to observe movement of the larva spicules in our case patient).<sup>3</sup>

Options for asphyxiation include nail polish or petroleum jelly, both of which are intended to weaken the larvae for extraction. An additional option characterized in the literature is applying

bacon,<sup>4</sup> which reportedly encourages the larvae to leave the skin either through asphyxiation and/or attraction to the bacon or bacon grease itself; some patients may attempt this as a home remedy.

There is also a case report describing the use of a commercial venom extractor.<sup>5</sup> There have been prior case reports<sup>6,7</sup> utilizing ultrasound for diagnosis and management, including those describing bedside excision in the ED.<sup>8,9</sup>

Possible complications include cellulitis, especially if there is any concern that the larva may have been transected or otherwise incompletely removed and dies within the skin, therefore In this case, treatment with a course of antibiotics targeting skin and soft tissue infections is very reasonable.

### Conclusion

Cutaneous larvae is an uncommon ED finding in the United States. Frequently patients who describe worms or insects under their skin are not found to have active parasitic infestation; their symptoms may instead be attributable to a wide variety of causes including dry

skin, eczema, infection, allergic reaction, or much less commonly, psychiatric illness.

For patients with relevant travel history or clinical exam findings, soft tissue point of care ultrasound can assist in evaluation for active parasitosis along with other etiologies such as fluid collection or foreign body.

In the ED, ultrasound can aid in diagnosis and provide procedural guidance for management of a variety of skin and soft tissue infections. Ultrasound may also have a place as a routine study to complete during the physical exam for these patients.

This case serves as a reminder to complete appropriate evaluation in any patient expressing concern for a parasite with correlating exam findings. When patients present with either nonspecific or suspect ("worms under my skin") signs or symptoms, bedside ultrasound can offer a very effective and rewarding approach to evaluating skin and soft tissue complaints, especially when clinical gestalt suggests there may be more than meets the eye. ★

# Torsades de Pointes after Olanzapine Use for Agitation

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Second-generation antipsychotics (SGA), including olanzapine, are effective and appropriate treatments for acute agitation and psychosis in the emergency department.<sup>1</sup> SGAs have increasingly replaced first-generation antipsychotics like haloperidol due to their safer side effect profile.<sup>2</sup> Among the antipsychotics, olanzapine has shown to have minimal QTc-prolonging effects.<sup>3</sup> Nevertheless, this case demonstrates that there is still a risk of QTc prolongation and torsades de pointes (TdP) with olanzapine use in the emergency setting.

## Case

A 39-year-old female with a past medical history of diabetes mellitus type II, hypertension, hyperlipidemia, chronic kidney disease, right parietal watershed stroke presented with altered mental status. Per her family, last night, the patient was vomiting and had elevated finger stick blood glucose levels at home. The patient's husband went to work this morning and was unable to contact her throughout the

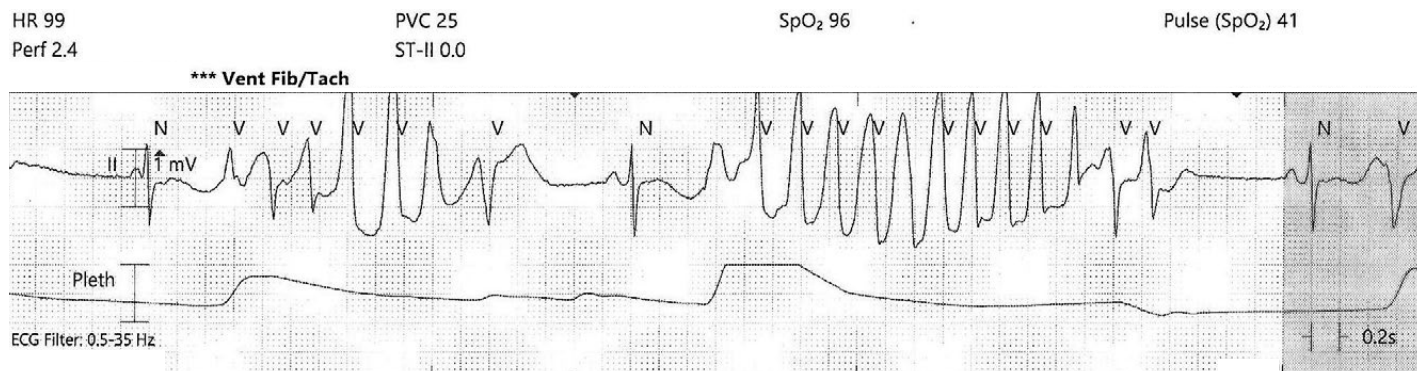
day, so he called 911. EMS found the patient lying on the floor. The patient denies pain but is confused, only saying "yeah" or "no" to most questions. Per EMS, the patient's finger stick blood glucose read "high." Her most recent documented medication use included only methocarbamol and tramadol from an emergency department visit after a motor vehicle crash.

On arrival in the ED, her vital signs were significant for hypertension to 212/107, but were otherwise normal. On exam, she was obese, awake, not following commands, not conversational, but answering some yes or no questions appropriately. Her pupils were equal and reactive, she was moving all extremities, and her neck was supple. However, she did have mild tenderness to palpation to the lower abdomen without rigidity, rebound, guarding, or costovertebral tenderness bilaterally. Labetalol 10 mg IV was also ordered.

Initial labs and imaging showed Na 134, K 3.3, Cl 95, Cr. 2.2, glucose 561, CO<sub>2</sub> 23, anion gap 19, albumin 2.8, WBC 18.66, beta-hydroxybutyrate 0.3, urine drug screen positive for oxycodone, a urinalysis with no signs of infection, chest X-ray with only hypoinflated lungs without infiltrates, and pH of 7.43 and lactic acid 2.3.

While the work-up was pending, the patient became agitated and poorly redirectable, then started to yell and get out of bed. 10 mg of IM olanzapine was ordered and administered prior to obtaining an EKG. About 5 to 10 mins later, the patient started to have polymorphic premature ventricular contractions lasting several seconds. Before the nurse could perform a stat EKG, the patient fell unconscious without a pulse and ACLS was initiated. Multiple rhythm checks showed torsades de pointes then pulseless ventricular tachycardia requiring 4 defibrillations. The patient was given 4 mg of IV magnesium sulfate, 3 doses of epinephrine, 1 ampule of bicarbonate, 150 mg IV amiodarone. She was intubated quickly and eventually achieved return of spontaneous circulation after about 10 minutes. The initial EKG after ROSC showed normal sinus rhythm with a prolonged QTc of 513 ms, but the longest QTc seen was 687 ms about 3 hours later.

Repeated labs after ROSC showed K 2.6, glucose 457, Cr. 2.3, Mg 2.8, total Ca 8.4, lactic acid 5.0. CT head without contrast redemonstrated her prior right MCA distribution infarct. CTA of the head and neck showed no significant abnormalities aside



**FIGURE 1. Polymorphic Premature Ventricular**



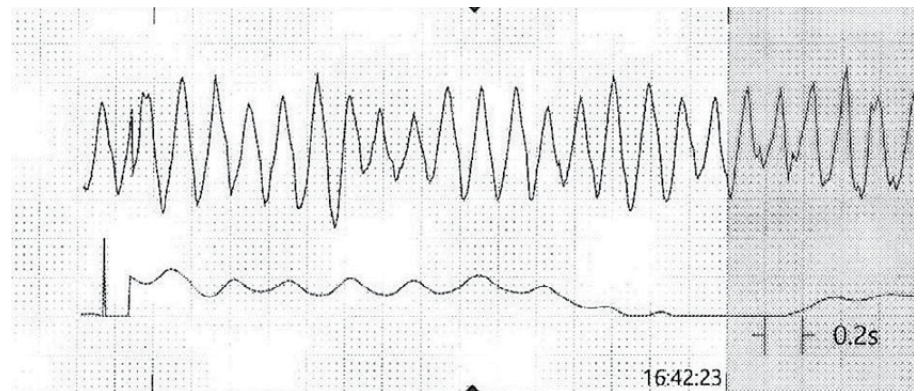
from large nodular left thyroid lobe. CT chest, abdomen, and pelvis with IV contrast showed left anterior rib fractures and no other significant findings. Thyroid studies later resulted with TSH mildly low at 0.11, free T3 normal at 1.41, and free T4 normal at 1.0.

MRI brain several days later would show global anoxic brain injury. Repeat thyroid studies showed a TSH 0.12 and free T3 elevated to 67, but T4 1.55 consistent with sick euthyroid disease. Her hospital course was complicated by MSSA and Acinetobacter pneumonia, ESBL E. coli UTI, persistent fevers, and decubitus wounds. She was discharged nearly 3 months later after tracheostomy and PEG tube, awake, but not conversational nor following commands.

## Discussion

There is only 1 published case report of torsades de pointes associated with olanzapine use in recent medical literature. Huang et al. documented a case of torsades de pointes found by her ICD in an elderly female who had started oral olanzapine for suicidality 3 months earlier.<sup>4</sup> She was also found to have a potassium level of 3.1 meq/L and QTc of 480 ms when hospitalized. She was switched to oral risperidone, her potassium level was repleted, and her QTc interval normalized. Hypokalemia could have been a second contributing factor to QTc prolongation, which finally led to TdP. However, she had no other reason to be hypokalemic, so the author postulates that olanzapine caused the low potassium level in this patient. Nevertheless, TdP is a rare phenomenon associated with olanzapine.

Multiple randomized control trials have also concluded that olanzapine has little to no effect on the QTc interval. Lindborg et al. studied the effects of intramuscular olanzapine by pooling data from four different double-blinded trials of acute agitation with schizophrenia or dementia in the emergency department and



**FIGURE 2.** Torsades de Pointes

**This case demonstrates a likely causation between the use of olanzapine and this occurrence of torsades de pointes and QTc prolongation, but there are many other factors that could have contributed to her arrhythmia.**

found that there was no significant QTc prolongation with olanzapine use.<sup>5</sup> Studies of oral olanzapine for psychiatric patients have demonstrated very mild increases in QTc compared to baseline.<sup>6-7</sup> Czekella et al. found only a clinically insignificant QTc lengthening with oral olanzapine in psychotic patients.<sup>8</sup> Hasanain et al. published a systematic review of second-generation antipsychotics in 2014 that also concluded that olanzapine only had “modest” effects on the QTc interval at therapeutic doses.<sup>3</sup>

This case demonstrates a likely causation between the use of olanzapine and this occurrence of torsades de pointes and QTc prolongation, but there are many other factors that could have contributed to her arrhythmia. Though the initial labs showed a slightly low level of potassium, it would not cause a prolonged QT. Unfortunately, a magnesium level was not ordered early on. After the cardiac arrest and administration of epinephrine and sodium bicarbonate, potassium dropped to 2.6 mmol/L. Though the appearance of polymorphic ventricular

contractions occurred quickly, about 5 to 10 minutes after the administration of IM olanzapine, intramuscular absorption of olanzapine occurs rapidly, and 5-10 minutes is likely enough time to see adverse effects.<sup>9</sup>

Though olanzapine and other second-generation antipsychotics are known for their favorable side effect profiles, QTc prolongation and torsades de pointes are still potential complications. Therefore, be wary of administering the medication concomitantly with other QTc-prolonging medications.

If time and resources are permissible, check an EKG for any suspicion of a pre-existing prolonged QTc. If there are any indications of possible hypokalemia or hypomagnesemia, check serum electrolyte levels. For alternative chemical restraint medications, benzodiazepines and ketamine may be considered, though they carry their own risks and contraindications. Olanzapine is an extremely practical medication in the emergency physician’s toolkit, but must be used in the right setting and with vigilance for possible side effects. ★

# Spinal Cord Ischemia in an Undifferentiated Patient

## A Case Report

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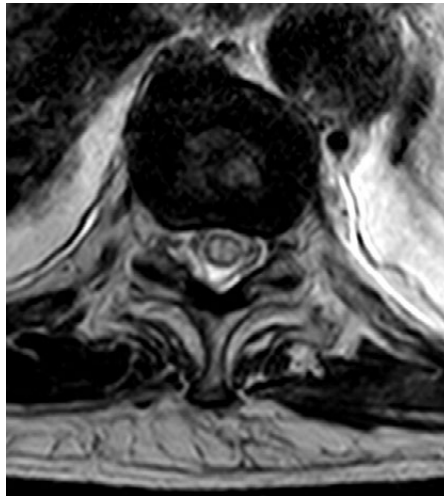
### Nicholas Jouriles, MD, FACEP

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Spinal cord ischemia is a rare cause of paraplegia, with spinal infarction accounting for only 0.3 – 1% of all strokes.<sup>1</sup> Though many of the documented cases of spinal strokes have occurred in the setting of acute aortic pathology or aortic surgery, the majority of spinal strokes (86%) are spontaneous.<sup>2,5</sup> Risk factors include atherosclerosis, degenerative disease, cardiac emboli, hypotension, and intercostal nerve blocks.<sup>7</sup> Most cases have been seen in young women, though there is not a strong correlation.<sup>1</sup>

Clinical presentation can be explained by the anatomy of the vessels supplying the spinal cord. The vasculature of the spine has great variation, but always involves the anterior spinal artery (ASA) as the dominant blood supply followed by two posterior spinal arteries (PSA). Additional branches of the aorta perfuse the spine depending on the vertebral level, which can be divided into 4 distinct territories. C1 to T3 receives supply from the vertebral arteries, T3 to T7 is often supplied by branches of the intercostal arteries, T8 to T12 (and sometimes the conus medullaris) receives blood by the major radicular artery of Adamkiewicz (AA), and the conus sometimes receives supply from the iliac branches.<sup>2,5</sup>

Spinal cord infarction is infrequent and if missed can result in significant mortality, disability (including irreversible paralysis), and reduced quality of life.<sup>2</sup>



**FIGURE 1. Axial — Owl Eyes**



**FIGURE 2. Sagittal T2**

*Reproduced from a case courtesy of Dr. Vincent Tatco Radiopaedia.org, rID: 45707, these MRI images demonstrate changes seen in a different patient with spinal cord ischemia due to aortic dissection. The sagittal views of T2-weighted and STIR sequences reveal a pencil-shaped area of increased signal in the intramedullary spinal cord from T8-T9 to the conus medullaris. The axial views of T2-weighted MRIs may exhibit an owl's eye appearance where the patient has high signals in the anterior horn cells bilaterally.*



**FIGURE 3. Sagittal STIR**

Here we discuss a case of spinal stroke in an undifferentiated patient.

### Case

A 48-year-old female was transferred from an outside hospital with concern for numbness, paresthesias, and weakness of the bilateral lower extremities. The patient had a past medical history of chronic abdominal pain and opiate use disorder. She reported IV drug use within the prior 2 weeks. She said had snorted fentanyl, taken a nap, and woke with inability to

move her lower extremities before going to the hospital. She reported a progressive history of weakness in her legs for several weeks leading up to this event. She denied fever, chills, saddle anesthesia, and urinary or fecal incontinence. She denied tick exposures. Of note, the patient had a recent admission (10 days prior) for generalized fatigue and weight loss. At that time, imaging demonstrated possible bilateral renal infarcts. She left the hospital against medical advice before completion of her workup.

On exam, she was alert with vital signs of: T 98.5°, P 112, R 16, and BP 154/104. There was no cardiac murmur. Neurologic exam showed lower extremity strength of 1/5 hip flexion/extension, 0/5 knee flexion/extension, 0/5 dorsi-/plantarflexion bilaterally. Patellar and Achilles reflexes remained intact and she had a negative Babinski. Rectal tone was absent. She denied midline spinal tenderness. Sensation was intact throughout her lower extremities. The rest of the physical exam was non-contributory.

Laboratory studies revealed a leukocytosis of 18.2, hyponatremia of 122, and hypokalemia of 3.0. Inflammatory markers were also elevated with a CRP of 174.7 and ESR of 95. Bedside ECHO was concerning for vegetation. The patient met sepsis criteria with probable cardiac or spinal sources, including endocarditis with emboli and spinal abscess, and empiric antibiotics and fluids were initiated.

MRI did not show evidence of spinal epidural abscess or discitis/osteomyelitis, but it was suggestive of infarction secondary to occlusion of the anterior spinal artery.

She was admitted for further workup and treatment.

## Discussion

This patient presented with symptoms consistent with cauda equina syndrome. Though it is classically taught that cauda equina syndrome is caused by compressive lesions, such as disc herniation, epidural abscess, tumor, hematoma, and canal stenosis, there are a number of non-compressive causes, including discitis, trauma, and aortic obstruction.<sup>4</sup> Typical symptoms of cauda equina syndrome include lower back pain, fever, saddle anesthesia, fecal or urinary incontinence or retention, and lower extremity weakness or paralysis. Often, subtleties on history or exam can point toward the correct etiology.

## Diagnosis

In this case, the patient's history of IV drug use and that she met SIRS criteria (tachycardia, leukocytosis) initially prompted concern for spinal epidural abscess. However, the clue that suggested spinal stroke of the anterior spinal artery instead of epidural abscess was the preservation of sensation on physical exam. This is because occlusion of the anterior spinal artery, which supplies the anterior portion of the spinal cord, would cause loss of function of the corticospinal (motor) tract but would spare the sensory tracts of light touch and proprioception.

It is important to note that the clinical presentation of spinal infarction varies based on the involved vessel and vertebral level. Patients can present with acute painless paraplegia, bowel and bladder dysfunction, loss of pain and temperature sense, but typically have preserved proprioception, vibration, and sensation to light touch.<sup>3,6</sup> In contrast, spinal epidural abscesses typically present with hypoesthesia secondary to local ischemia from direct compression of the spinal vasculature or via septic thrombophlebitis.<sup>3</sup>

Ultimately, MRI with contrast is the gold standard diagnostic test for both spinal infarctions and spinal epidural abscesses.<sup>1,3</sup> It should be obtained emergently if either condition is on the differential. ★

## TAKE-HOME POINT

It is important to consider spinal infarction in the differential for back pain with neurologic deficits, as prompt recognition can improve patient outcomes, prevent long-term disability, and maintain quality of life.

# WHAT KEEPS YOU UP AT NIGHT?

## EM Residents Face Challenges on Many Fronts



Caring for patients during a pandemic



Ensuring that care in the ED is provided by physician-led teams



Psychiatric and SUD Patients with nowhere to turn but the ED



Shortages of medicines that your patients need



Financial and career stability



Time for personal wellness

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# Not All Halos Are Glaucoma

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## Introduction

Eye-related emergencies make up close to 2% of all emergency department (ED) visits in the United States.<sup>1</sup> As Emergency Medicine physicians, the priority is to recognize and manage time-sensitive, vision-threatening diagnoses, and ensure appropriate management once the emergencies are ruled out. This is a case report of an uncommon diagnosis masquerading as a vision-threatening emergent eye complaint.

## Case Report

A 60-year-old male presents to the ED with worsening left eye redness for the past 4 days with associated photophobia and blurred vision. One day prior to arrival, he began to feel pressure in the left eye and observed halos around objects. He denied nausea, vomiting, rashes, or headache. He denied recent trauma or surgery to the eye. He used saline drops and ibuprofen with minimal relief of his symptoms. Relevant history included an infection of the left eye as an infant with subsequent visual disturbances; however, his vision was worse than baseline on presentation to the ED. Additionally, the patient had similar symptoms a few years ago, which were treated with “eye drops,” but he could not recall the diagnosis or the medication given at that time. Although the patient did not have any active cold sores at the time of

presentation, he did confirm a history of labial cold sores during subsequent outpatient follow-up.

Initial exam of the left eye was remarkable for 3+ injection of the sclera and conjunctiva, pain with extraocular movements, and a fixed, dilated pupil at 5mm. The left eye also had severely reduced visual acuity and an intraocular pressure (IOP) of 20mmHg. The right eye exam was unremarkable. Despite the IOP being within normal range, there was a high suspicion of acute angle closure glaucoma (AACG) and ophthalmology was immediately consulted. As the team was in-house and findings were inconsistent with glaucoma, initiating treatment in the ED was deferred. Per ophthalmology, visual acuity was 20/800 -1 in the left eye. The slit lamp exam of the left eye revealed decreased corneal sensation, diffuse corneal edema

including Descemet's folds, iris posterior synechiae, and anterior chamber cells. The anterior chamber of the left eye was deep. Posterior segment exam was unremarkable for both eyes with normal optic nerve and retinal findings.

Given the anterior chamber inflammatory reaction with the presence of corneal edema and absence of corneal sensation, the diagnosis of endotheliitis likely secondary to herpes simplex virus (HSV) was made by ophthalmology. He was discharged with valacyclovir 500mg three times daily, prednisolone acetate 1% topical drops four times daily, and cycloplegic cyclopentolate 1% topical drops four times daily. Outpatient follow-up with a cornea specialist was scheduled for 2 days following the ED visit.

At the follow-up visit, the patient's ocular pressures in the left eye were 19 and 21 mmHg, with scattered inferior keratic precipitates (KP) and improved corneal edema (see Image 1) further confirming the diagnosis of HSV keratouveitis. The corneal edema and anterior chamber reaction resolved with a slow taper of the steroid eye drops and oral valacyclovir over the following 6 weeks.

The patient's presentation of scleral and conjunctival erythema, severe eye pain, photophobia, decreased vision, and a dilated pupil raised concern for AACG. However, given the patient's high-normal IOP, anterior chamber inflammation, and lack of anterior chamber shallowing, inflammatory causes were considered including HSV keratouveitis.

## Discussion

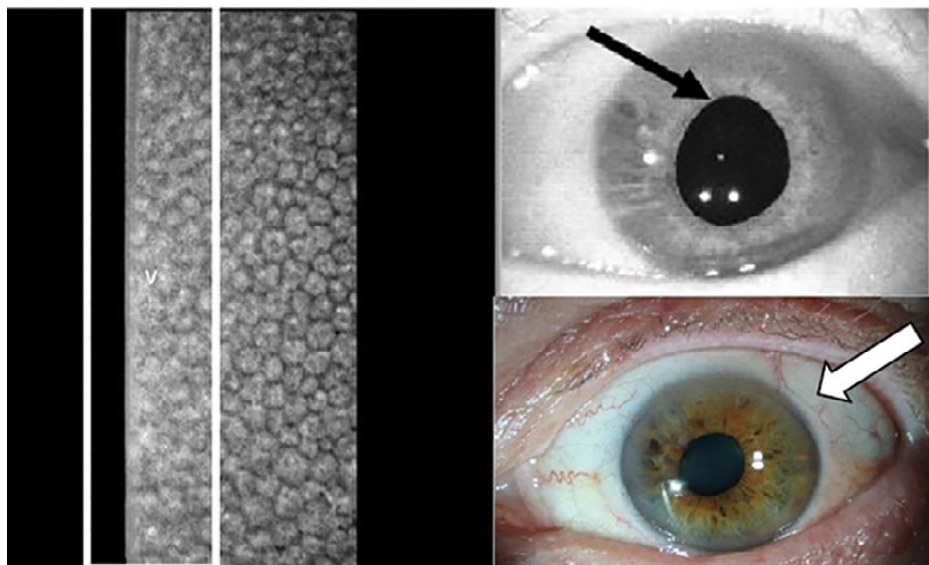
Herpes simplex virus keratouveitis is an uncommon presentation of an HSV ocular infection, and HSV contributes to about 5-10% of uveitis cases.<sup>2</sup> Uvea refers to the middle part of the eye. The iris and ciliary body form the anterior portion of the uvea.<sup>3</sup> Keratouveitis is inflammation of the cornea and anterior uvea. Symptoms include pain, blurry vision, photophobia and redness. Exam findings can include a combination of corneal epithelial and stromal edema, stromal keratitis, keratic precipitates, endotheliitis, and anterior chamber cells and flare.<sup>2</sup> Endotheliitis is inflammation of the corneal endothelium. The endothelial layer is the innermost layer of the cornea with a specialized basement membrane called Descemet membrane. Folds in the Descemet membrane are

caused secondary to severe corneal edema.<sup>4</sup> Keratic precipitates are inflammatory cellular deposits on the corneal endothelium and can vary in form and location.<sup>2</sup>

A distinguishing factor for HSV keratouveitis from other causes is an increased IOP secondary to inflammation of the trabecular meshwork, or trabeculitis, which impacts aqueous flow.<sup>5</sup> Other forms of uveitis typically have a low IOP as ciliary body inflammation leads to decreased aqueous humor production.<sup>6</sup> In this case, the IOPs were on the higher side of normal, in direct contrast with the IOPs of AACG which tend to be much higher. This patient also had a non-reactive pupil which was due to formation of scarring on the posterior iris to the anterior lens capsule due to intraocular inflammation. Additionally, HSV keratouveitis has significant corneal edema compared to other etiologies.<sup>5</sup> Treatment includes topical glucocorticoids and systemic antivirals.<sup>2</sup>

## Conclusion

As demonstrated in this case, it is not only important to have a high suspicion of emergent diagnoses, but to also consider other diagnoses. As emergency physicians, we must perform a complete physical exam to consider all diagnoses and further differentiate them. In this case, the vitals of the eye (pupils, vision, and pressure) were essential and saved the patient from unnecessary glaucoma treatment. Appropriate management of the presenting complaint and early discussion with consultants, if available, can lead to a more focused exam and expedited diagnosis and treatment. If consultants are not available, proper bedside exam and early discussion can facilitate appropriate transfer to a higher level of care or timely outpatient follow up. Given early timing of treatment for this patient, his condition resolved with no decline in vision. If the patient had not received the necessary steroids and valacyclovir prior to discharge from the ED, his condition would have continued to worsen until his outpatient follow-up. ★



**IMAGE 1.** Corneal endothelial cell electron specular microscopy and slit lamp images of the left eye obtained 2 days after initial presentation with herpes simplex virus keratouveitis. The white rectangle shows cell drop out and the white dots in the rectangle are keratic precipitates. The black arrow highlights the posterior synechiae, adhesions between the iris at the pupillary margin and anterior lens capsule that occur with inflammation, causing the pupil to be irregular and dilated. The white arrow shows the mild corneal edema.





## DUTY TO SERVE

# Federal Law Enforcement and the Foundation for Emergency Medicine

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Emergency Medicine Resident  
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### Nicholas P. Cozzi, MD, MBA

EMRA Representative to ACEP  
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Emergency medicine attracts a unique personality dedicated to excellence and teamwork, driven to work hard and push the boundaries of what is possible. Here we present the backstory of two emergency medicine physicians-in-training linked by the bond of having spent time working with federal law enforcement prior to medical school. Both trainees share the value of hard work and service.

### Aaron R. Kuzel, DO, MBA

#### ***Forensic Scientist, Federal Bureau of Investigation (2013-2015)***

Fidelity, Bravery, and Integrity are the core values of the Federal Bureau of Investigation. Each member of the FBI, whether they are a Special Agent, scientist or support staff, swears an oath to uphold these values in their duty to serve the American people and seek justice for those afflicted by criminal enterprise. This strict adherence to these principles over generations has secured the FBI's place as the most prestigious law enforcement and domestic intelligence agency in the world. As a former FBI forensic scientist serving in the FBI Laboratory, I endeavored to embody these values in

each case and service provided to the public as a representative of the FBI. Although I now serve a new mission to provide emergency care to the Louisville community, I continue to endeavor to uphold these values in my practice; to care for those most vulnerable in the community, uplift the downtrodden, and provide compassion to those experiencing unconscionable tragedy.

I am humbled to have served as a forensic scientist with the Federal Bureau of Investigation. My experience with the FBI prepared me for a career in emergency medicine, providing the fortitude to adapt to challenges of emergency medicine residency during a pandemic. During my brief appointment to the FBI, I had the opportunity to serve in the forensic evaluation of improvised explosive devices (IEDs) with the Terrorist Explosive Device Analytical Center (TEDAC) and perform research in X-Ray fluorescent technologies in the identification of human skeletal remains. Now as an emergency physician, I have adopted the core values of the FBI as my own principles in my practice of emergency medicine and how I best serve my patients.

My experience in the FBI was formative, preparing me for the rigors and challenges of emergency medicine. I am humbled and honored to have had the opportunity to serve the Federal Bureau of Investigation. The experiences and lessons of the FBI continue to

have an impact on me as a physician and in my patient care. While I am no longer working with the FBI, I continue to apply these values in my work as a physician. Faithful fidelity to my studies, my colleagues, and my patients, bravery to accept the challenges inherent to the practice of medicine, and, most importantly, the integrity to acknowledge the inevitable opportunities for reanalysis and reevaluation in order to develop into a more competent physician.

### Nicholas P. Cozzi, MD, MBA

#### ***Intern, United States Secret Service (2012)***

I vividly recall the scene as I entered a non-descript Chicago-based government building as a 22-year-old starting my first day of internship with the United States Secret Service. Fast-forwarding 7 years later, I would feel that same trepidation, sweaty palms, and sense of inadequacy as I began my internship as an emergency medicine resident. As I entered the lobby, I was struck by a steel phrase chiseled into the wall: "Worthy of Trust and Confidence." I knew at the moment the phrase would be more than a motto but a way I wanted to live my life. The fact is that we are all trying to be worthy of trust and confidence as we enter the thousands of emergency departments across the nation. I believe all emergency physicians-in-training are striving to be worthy of the trust and confidence of



our patients, our co-residents, and our support teams. We bear witness to our patients who are enduring some of the worst moments of their lives in situations thought unimaginable. We listen to guarded secrets. We hold the hands of grieving widows and new mothers as they enter the next phase of their lives.

As an intern, I performed many intern-level tasks. I made copies, learned how to recognize fake paper currency, and gained an appreciation for the work ethic of our nation's unheralded professionals and their dedication to the mission and our nation. They sacrifice at tremendous personal cost to their families much to ensure the continuity of our government and our nation's blood supply, our financial system. My experience culminated with learning and participating in the planning and execution of Election Night 2012. The breadcrumbs of event medicine were dropped that evening in McCormick Place in Chicago, and it ultimately led me to my passion for EMS.

As I transitioned to medical school after my MBA, I realized the key to success was more about being a problem

## TOP TEN PEARLS

1. Velocity is more important than speed. Ensure your professional ambition has direction and surround yourself with people who can kindle the flame.
2. The ability to quickly establish a genuine rapport with a wide variety of people is a superpower.
3. Second-order thinking separates pros from amateurs.
4. Celebrate your teammates' wins. Other people's success does not mean you are failing.
5. Exceeding expectations, accurate documentation, and being deadline-cognizant are ingredients to the secret sauce of success.
6. Avoid complacency in your studies as an emergency physician. Our patients expect continued lifelong learning and growth as evidence-based medicine progresses.
7. Always remember the mission. Remember the common mission to serve our specialty, our communities, and our patients.
8. It is courageous to care for yourself, know your own limitations, and call for help.
9. Failure is a painful but incredible teacher. Failures in medicine teach us wisdom and prudence. They prevent us from falling into greater disaster. Persevere through the difficult challenges; you will arise stronger and wiser.
10. Your word is your bond. Always preserve your integrity.

solver and making the people around you better. I am grateful to have had the opportunity to complete an internship with the Secret Service at such a formative time in my life. ★

**Disclaimer:** The views expressed are those of the authors and do not reflect the official policy or position of the Federal Bureau of Investigation, United States Secret Service, or the United States government.

## Annals of Emergency Medicine Resident Editorial Board Fellowship Appointment

The Resident Fellow appointment to the Editorial Board of *Annals of Emergency Medicine* is designed to introduce the Fellow to the peer review, editing, and publishing of medical research manuscripts. Its purpose is not only to give the Fellow experience that will enhance his/her career in academic emergency medicine and in scientific publication, but to develop skills that could lead to later participation as a peer reviewer or editor at a scientific journal. It also provides a strong resident voice at *Annals* to reflect the concerns of the next generation of emergency physicians.

### Selection Process

The Resident Fellow is chosen in a competitive selection process in July. Members of the selection committee, including the Editor in Chief, the deputy editors, *Annals'* Editorial Director, and the current Resident Fellow, review all curriculum vitae and select the top three candidates. These candidates are interviewed by the Editor in Chief, who then makes the final selection. Preference will be given to senior-level residents; applicants currently in training as EM fellows are not eligible.

### Application Process Requirements

#### Required materials:

- Completed application form
- Current curriculum vitae
- Cover letter
- 2 letters of recommendation
- 1-page personal statement
- 2-3 proposed topics for future "Residents' Perspective" columns

**Deadline: July 25, 2022 • Download the application packet at [www.annemergmed.com](http://www.annemergmed.com)**

### Questions?

Please contact Martha Villagomez, Editorial Associate, *Annals of Emergency Medicine*  
mvillagomez@acep.org or 800-803-1403, ext. 3223



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# A Fellowship for the Future

**Jonathan Oskvarek, MD, MBA**

Innovation Fellow, US Acute Care Solutions

**Angela Cai, MD, MBA**

Innovation Fellow, US Acute Care Solutions

@angelagcai

The need for innovation in EM has never been greater. The pandemic has strained physical and emotional resources and exposed the fragility of volume-based, fee-for-service reimbursement. Payers and legislators are pushing back on rising health care costs with decreasing reimbursement and new payment models. Patients and society are demanding value, outcomes, and convenience, necessitating our profession to evolve into new clinical and administrative roles. That's why US Acute Care Solutions (USACS) formed an innovation team, including two Innovation Fellows, who are the authors of this article. USACS is a physician-owned clinical practice group that provides patient care at more than 300 facilities in EM, hospital medicine, critical care, obstetrics, observation medicine, and post-acute care.

Innovation is difficult, especially for large, established companies that might have a lot to lose on new endeavors. *The Innovator's Dilemma* by Clay Christensen is a classic business book that describes how successful companies can do what seems right, but still fail in the long run as new, radically innovative competitors rise. According to Christensen, there are two types of innovation: sustaining and disruptive. Think of sustaining innovations as making incremental improvements to existing products or services (eg, a flip phone with a better battery, faster CT scanners, or video laryngoscopy intubation). In comparison, disruptive innovations fundamentally change the product or business model (eg, iPhones replacing flip phones, or telehealth challenging the notion of EM existing within the four walls of a

hospital-based ED). Christensen points out that disruptive innovations rarely come from established companies because they struggle to make high-risk investments into uncertain innovations. Companies feel comfortable sticking to what they do well and hoping that it takes a long time before disruptive competitors take over.

At USACS, our mission is to care for patients by being national leaders in EM and acute care. In order to achieve that, we must pursue sustaining and disruptive innovation. Our innovation team includes ED physicians with extensive research experience, data scientists, and now two Innovation Fellows. We have access to data from over 7 million patient encounters per year, our senior leaders at USACS, as well as over 3,500 practicing clinicians.

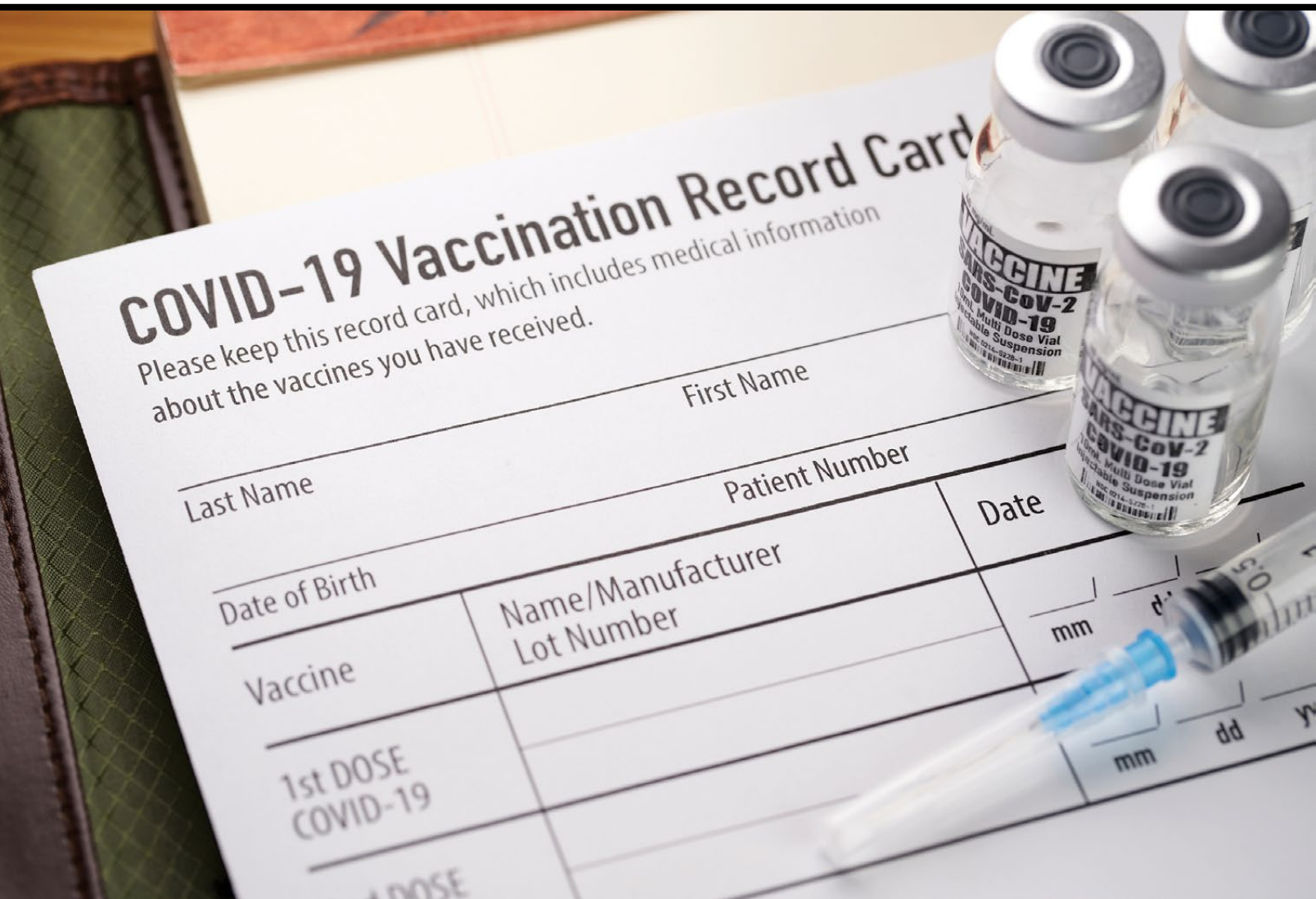
As the fellows on the innovation team, we are leading projects to improve the quality and value of care. For example, USACS discovered that opioid prescription rates varied greatly between clinicians and sites. Working with our data analytics team, we developed a dashboard to show clinicians their opioid prescription rates compared to their peers. Outliers in prescribing were contacted by USACS' Chief Medical Officer team. This program led to a 19% relative reduction in opioid prescribing which has been sustained since program implementation two years ago. The next step in this project is to increase the use of medication-assisted treatment (MAT) for opioid use disorder by increasing buprenorphine prescriptions and promoting local MAT champions. These programs will have a positive impact on minimizing the risk of our patients

developing opioid use disorder.

The Innovation Team at USACS also leads metric and intervention development to respond to increased pressure from payers on ED clinicians to justify the costs of care. As Porter and Lee point out, health care organizations will need to focus on creating value for patients to survive as payers focus on costs.<sup>1</sup> Our leadership at USACS understands that there is often wide variability in care. Two patients could have completely different emergency care experiences, even with the same complaints and risk factors. Coupled with USACS' evidence-based clinical management tools, we are developing an admission intensity measure that will help clinicians assess and safely disposition patients in a cost-effective manner. We are also working with the state of Maryland to develop bundled payments, which sets a price for a specific clinical presentation over a set number of days along with quality targets. These types of reimbursement models are growing in health care, so we want to be part of their development and learn how to deliver good quality, cost-effective care in new reimbursement models.

EM continues to change. Patients deserve higher value care that optimizes the ratio of quality to cost and solves the reason they came to us. We want to be part of the solution by defining how our specialty will respond to those needs. For new ideas to succeed, we at USACS, and EM as a whole, need to create space to try new ideas. This innovation fellowship allows us to play in that space on a national scale to ensure that physicians like us take charge of the future of healthcare. ★





# COVID-19 Vaccine Equity and Distribution in EDs

**Rachel Kearney, MPH**

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[@sk\\_leung](#)

Social EMpathy is a working group of medical students, residents, and faculty interested in social emergency medicine working together to organize initiatives in social EM topics.

As part of Social EMpathy's first roundtable, we aimed to explore the topic of vaccine equity and distribution in the ED. Vaccine equity has been a major area of concern particularly when it comes to COVID-19. The most recent CDC data shows that among the vaccinated population, 60% were white, 9% were Black, 15% were Hispanic, 6% were Asian, 1% were American Indian or Alaska Native, and <1% were Native Hawaiian

or other Pacific Islander.<sup>1</sup> Ultimately these data show that proportionally, marginalized groups continue to be underrepresented in COVID-19 vaccinations. Even the data being collected to ask questions about racial equity in distribution is lacking.<sup>2</sup>

Given the pressing nature of these disparities, both in COVID-19 vaccines and others, we gathered three leaders in vaccine equity from around the country who shared their experiences with COVID vaccine administration in their respective communities. Each speaker shared practical advice based on their experience working toward greater vaccine equity.

### Meet the Experts

**Evan Rusoja**, MD, PhD, is the current Quality and Safety Innovations Fellow as well as an attending emergency physician at Alameda Health System in Oakland, CA. Dr. Rusoja has applied his expertise in global health to vaccine equity by leading efforts to bring the vaccine to patients directly in their own communities as well as administering the vaccine to patients in the ED.

**Morgan Hutchinson**, MD, is the Medical Director for Vulnerable Communities and Assistant Medical Director of the emergency department at Thomas Jefferson University Hospital in Philadelphia. Dr. Hutchinson's current work around vaccine equity focuses on partnering with community clinics to vaccinate Philadelphia's immigrant and refugee populations along with administering the Johnson and Johnson vaccine in the emergency department.

**Amita Sudhir**, MD, is an attending emergency physician and Program Director of the EM residency at the University of Virginia. In addition to authoring multiple articles on vaccine equity, Dr. Sudhir has spearheaded the recent effort to vaccinate patients in the emergency department at the University of Virginia, many of whom come from rural areas or are undocumented workers with poor access to healthcare.

### Logistics of Distributing Vaccines

Because several clinic locations already had COVID testing labs, a few of them were able to be converted to vaccine distribution sites as well. Incorporating a

mobile vaccine element and distributing vaccines at school and church parking lots also proved to be beneficial. Having this mobile distribution arm helped reach vulnerable communities and eliminated any traveling or access barriers.

Stakeholder buy-in is also key. At one speaker's institution, a vaccine task force was created that had representatives from stakeholders, department heads, and others. This team was willing and ready to make timely and well-informed decisions to help with rapid vaccine distribution which accelerated various efforts during a tumultuous time in distributing vaccines.

Regarding vaccine logistics, the current vaccine vials have five doses in each vial, so the timing of vaccine distribution is critical in order to make sure that no vaccine is wasted. Funding can be a challenge to obtain, and departments often purchase the vaccines while clinic staff members volunteer to support operations. City grants may also be available to help fund operations in particularly vulnerable communities. Medical students have been helpful volunteers for staffing the clinic and helping with education efforts in the community. Also, trusted messengers from a variety of cultural and linguistic backgrounds were educated about the vaccine and helped establish trust with communities that might be hesitant about receiving the vaccine.

### Challenges in Vaccine Distribution

Obtaining buy-in from stakeholders was initially a labor-intensive and time-consuming challenge. Lack of clarity about priority groups and perceptions on distribution were some of the many hurdles. Engaging stakeholders in the conversation early, performing background research, and maintaining persistence are key to success. Cost may be mitigated by use of volunteer clinicians and medical students. City grant funding may also be available to support ED vaccination efforts.

Other challenges include barriers to patients obtaining the vaccine. Addressing patient fears and concerns in a clear and non-judgmental way is incredibly important when discussing

vaccination. Consideration of factors beyond general vaccine hesitancy is equally important, however. Often patients who are labeled as noncompliant or hesitant in fact have concrete barriers to care, such as incarceration, lack of transportation, financial constraints, or fear of the repercussions of documentation. Recognizing and addressing the unique challenges of patients instead of making assumptions is an essential part of the vaccination effort.

### Success Metrics

In order to create more effective vaccine distribution programs, targets need to be defined and outcomes need to be measured. Defining success can be challenging, however, when the goal is not simply to increase the number of vaccines delivered, but to increase access and equity as well. Focusing solely on the number of vaccines administered can bias providers from trying to provide access to those who may be harder to reach (e.g. elderly, patients with language barriers, limited access to tech, uninsured/don't want to share SSN, patients with disabilities). As a result, it is important to measure demographic data and identify barriers faced by patients who receive the vaccine. Demographic data on vaccine recipients can be compared to peer institutions and departments and serve as a proxy measure for increasing access and equity.

In addition, while it is important to collect data on those who receive the vaccine, it is equally important to collect data on those who do not. Providers should document when they offer the vaccine and when patients decline for example, through the use of EPIC smart-phrases. Data on demographics of individuals who decline the vaccine and reasons for declining can be used to target future educational campaigns and outreach initiatives. While outstanding progress has been made in vaccine distribution, continued work is needed to define and measure success with a focus on access and equity.

### More on SocialEMpathy

Learn more and join our group at [SocialEMpathy.carrd.co](https://socialempathy.carrd.co). ★

S A N F R A N C I S C O

# EMRA @ ACEP22

Sept. 26 – Oct. 4

## Monday, Sept. 26

6 pm CT EMRA Resolution Review (virtual)

## Friday, Sept. 30

4 – 6:30 pm EMRA/ACEP Leadership Academy  
*(invitation only)*

7 – 9 pm EMRA Leaders Meet Up  
*(invitation only)*

## Saturday, Oct. 1

TBD Committee Meetings

TBD Case-Con Competition

5 pm – 7 pm EMRA Job & Fellowship Fair

## Sunday, Oct. 2

7 am – 1 pm EMRA Representative Council  
& Town Hall Meeting  
*EMRA Board of Directors Elections!*

TBD Committee Meetings

9 am – 3 pm EMRA Resident SimWars

3 – 6:30 pm EMRA Awards & VIP Reception

10 pm – 2 am EMRA Party



*See you in  
San Francisco!*

## Monday, Oct. 3

TBD Committee Meetings

1 – 3 pm 20 in 6 Resident Lecture Competition

## Tuesday, Oct. 4

7 am – 5 pm EMRA MedWAR

*All times listed are Pacific time unless otherwise noted.*

# Sign up this summer!

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



# EMRA Honors 2022 Winter Award Recipients

Please join EMRA in congratulating  
our 2022 Winter Awards recipients.

## 2022 EMRA WINTER AWARD WINNERS

### Academic Excellence Award

Nicole Duggan, MD | Harvard Affiliated Emergency Medicine Residency  
Ultrasound Fellowship

### Dr. Alexandra Greene Medical Student of the Year

David Wilson | Sidney Kimmel Medical College

### Chief Residents of the Year

University of Louisville – Kentucky

Aaron R. Kuzel, DO

Joseph McKinney, MD

Ross Sizemore, MD

**Fellow of the Year:** Mina Ghobrial, MD |

**Resident of the Year:** Joshua Lesko, MD | Naval Medical Center Portsmouth

**Jean Hollister Contribution to Prehospital Care Award:** Ryan F. DeVivo, DO | LAC Harbor UCLA Medical Center

**ACEP CORD Teaching Fellowship Scholarship:** Christopher Reilly, MD | Brandon Regional Hospital

### Faculty Awards

**Department Chair of the Year:** Brendan Carr, MD, FACEP | Icahn School of Medicine at Mount Sinai

**Residency Director of the Year:** Arlene Chung, MD, FACEP | Maimonides Medical Center

**Associate Residency Director of the Year:** Cynthia Price, MD | University of Connecticut Hartford Hospital

**Residency Coordinator of the Year:** Julie Mendez | Rush University Medical Center

### Travel Scholarships

**ACEP Scientific Assembly:** Anthony Thai

**SAEM Annual Meeting:** Brittany Ladson | Michigan State University College of Osteopathic Medicine

### EDDA Scholarship

Hamza Ijaz, MD | University of Cincinnati College of Medicine

Rahul Tilani, DO | Hackensack University Medical Center



# EM Leaders of Tomorrow

One of the things EMRA loves most — and does best — is help to create confident, inspiring leaders to guide the specialty into the future.

Through the EMRA & ACEP Leadership Academy, our roster of 20 EMRA committees, the EMRA Medical Student Council, the Health Policy Academy, and EMRA representatives to ACEP sections and committees, EMRA strives to offer opportunities to connect, grow, and influence the issues that will define EM moving forward.

Please help us recognize the lasting contributions and positive impact of these rising leaders.

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AMA-MSS Representative: Spencer  
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Southeast 2: Kevin Bennett  
International: Matthew Carvey

***“I have gained multiple mentors across the country who have provided me with career advice, field experience, and academic opportunities. I would highly recommend getting involved as a rep to anyone interested.”***



***“Working as an EMRA representative to the ACEP Social Emergency Medicine Section has been a unique and rewarding opportunity to work alongside leaders in the field. It has allowed me to network with faculty and fellow residents across the country and collaborate on projects to advance the care of patients facing socioeconomic disadvantages.”***

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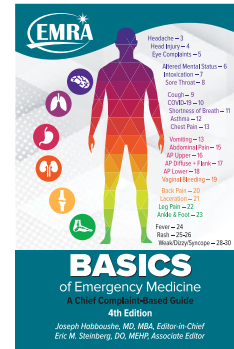
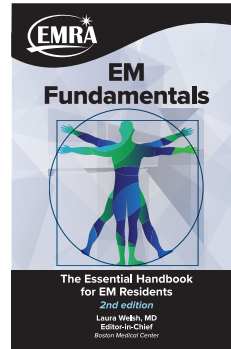
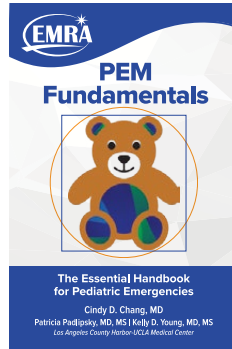
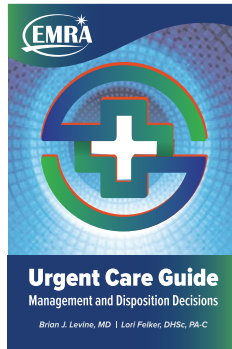
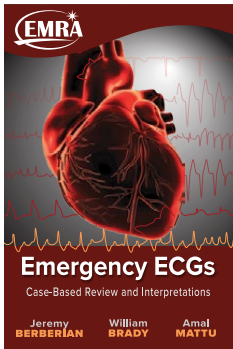
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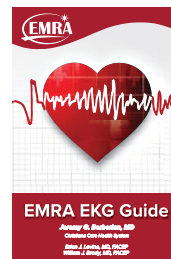
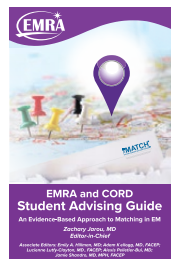
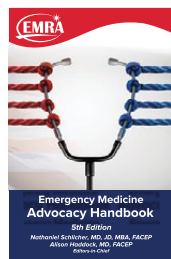
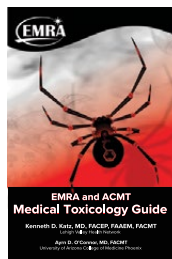
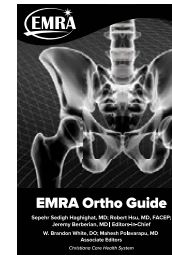
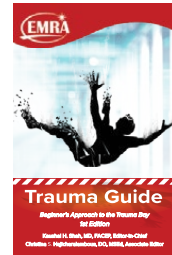
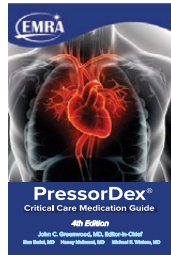
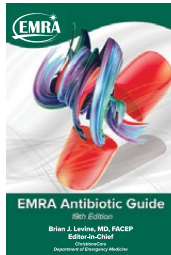
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# ECG Challenge

**Graham Clifford, DO**

Emergency Medicine Chief Resident  
University of Florida at Shands Hospital

**Benjamin Arnold, MD**

Emergency Medicine Resident  
University of Florida at Shands Hospital

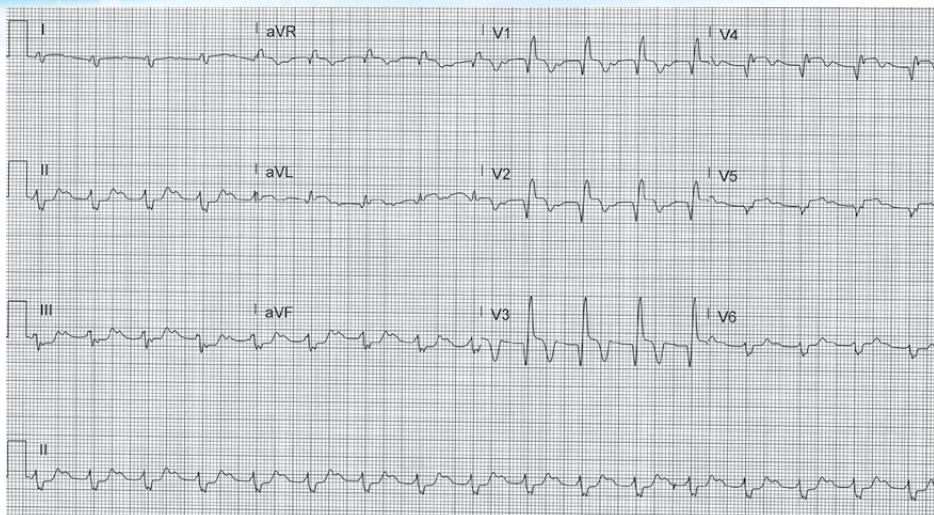
**Jeremy Berberian, MD**

Associate Director of Emergency Medicine Resident Education  
Dept. of Emergency Medicine, Christiana Care Health System  
@jgberberian

**CASE.**

A 67-year-old male with a PMH of HTN, HLD, DM, and COPD presents to the emergency department due sudden chest discomfort.

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



See the ANSWER on page 52



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"The most significant impact of the grant was that it solidified my desire to pursue a career in EM cardiovascular research. It helped align me with an excellent team of mentors."

NICK ASHBURN, MD

2019-2020 EMF/EMRA Resident Research Grant Recipient



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# ECG Challenge

## RBBB STEMI

This ECG shows a normal sinus rhythm at 99 bpm, northwest/extreme axis deviation, 1st degree AV block, prolonged QRS complex duration with a RBBB, STE in leads II and V2-V5, and STD in leads II, III, and aVF. These findings are consistent with an anterolateral STEMI.

The characteristic findings in a normal RBBB (see Figure 1) include:

- QRS complex duration  $\geq 120$  msec
- $rsr'$ ,  $rsR'$ , or  $rSR'$  pattern in lead V1 +/- V2
  - Variation in lead V1 include qR pattern or broad R-wave that is often notched
  - In lead V1, the initial upward deflection should always be smaller than the 2nd upward deflection
- S-wave duration  $>$  R-wave or  $>$  40 msec in leads I and V6
- Normal R-wave peak time in leads V5 and V6 but  $>$  50 msec in lead V1 (only required if broad R-wave +/- notch is present in lead V1)
- Expected repolarization abnormalities include STD and TWI in lead V1 +/- lead(s) V2-V3 if they have an  $rsR'$  pattern, so STE and/or upright T-waves in those leads are concerning for ischemia

In normal cardiac conduction, impulses travel equally down the left and right bundles in a near simultaneous manner. When there is a RBBB, the left ventricle is activated normally, but there is a delay in depolarization across the right ventricle which produces a secondary R-wave ( $R'$ ) in the right precordial leads and a wide or slurred S-wave in lateral leads. In an isolated RBBB, left ventricular depolarization is unchanged so the axis is typically normal. A RBBB with an axis deviation should prompt evaluation for a concurrent left anterior or posterior fascicular block.

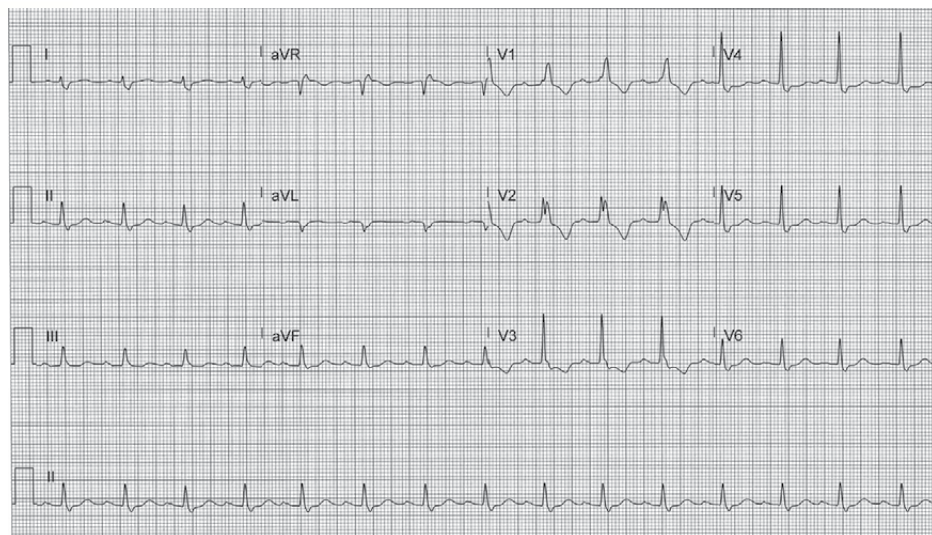


FIGURE 1. ECG showing a normal RBBB with expected repolarization abnormalities

A RBBB will typically have STD and TWI in lead V1, and if leads V2-V3 have an  $rsR'$  pattern present, they will also show this pattern. Consequently, upright T-waves or STE in those leads with an  $rsR'$  pattern is concerning for ischemia, and even isoelectric or minimally elevated ST-segments can be a subtle indicator of early AMI. In this case, the presence of reciprocal STD in the inferior leads supports, but is not required for, the diagnosis of an MI.

In general, the presence of a RBBB does not confound the ECG evaluation of ACS as does a LBBB which requires the use of Sgarbossa criteria. Notably, patients presenting with a new RBBB in the setting of an anterior STEMI have a higher rate of acute heart failure, complete heart block, need for a permanent pacemaker, and a higher 30-day in-hospital mortality.<sup>1</sup>

### Case Conclusion

This patient was taken to the cardiac catheterization laboratory where a 99% occlusion of the LAD was successfully treated with a stent. The patient was discharged home 3 days later in good health. ★

### RBBB Learning Points

- Delayed conduction through right ventricle with normal left ventricular conduction
- In lead V1, the initial upward deflection should always be smaller than the 2nd upward deflection
- Repolarization abnormalities include STD and TWI in lead V1 +/- lead(s) V2-V3 if they have an  $rsR'$  pattern, so STE and/or upright T-waves in those leads are concerning for ischemia
  - Otherwise, the presence of a RBBB does not confound the ECG evaluation of ACS as does a LBBB
- RBBB with axis deviation should prompt evaluation for a concurrent LAFB or LPFB

# Board Review Questions

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- 1. Which factor has been associated with sudden unexpected infant death?**
  - A. Firm bedding during sleep
  - B. Heavy bedding during sleep
  - C. Pet ownership or exposure
  - D. Supine position during sleep
- 2. A 35-year-old man presents with cramping abdominal pain, abdominal distention, and flatulence with frequent episodes of foul-smelling, explosive, nonbloody diarrhea. He has just returned from a camping trip and has been drinking water from local streams. What is the most appropriate treatment for this condition?**
  - A. Azithromycin 500 mg PO once daily for 5 to 7 days
  - B. Ciprofloxacin 500 mg PO twice daily for 7 days
  - C. Iodoquinol 650 mg PO three times daily for 20 days
  - D. Metronidazole 500 mg PO three times daily for 10 days
- 3. A mother brings in her 17-month-old son because he has a fever and a rash. She says he had a fever of 39.4°C (102.9°F) and a runny nose 4 days ago, but the fever resolved on the day of presentation. He has a maculopapular rash on his stomach, back, and neck that the mother says "cropped up" a couple of hours before they came in. The rash is pink and blanchable without involvement of the mucous membranes. He is otherwise healthy and well-appearing on examination. What is the most likely diagnosis?**
  - A. Erythema infectiosum
  - B. Measles
  - C. Roseola infantum
  - D. Rubella
- 4. A 23-year-old woman presents after a witnessed syncopal episode. She is 1 week postpartum and has had a mild generalized headache and vision disturbances since delivery. Her vital signs include BP 126/82 and P 90. Laboratory test results include Na 119, K 4.2, and glucose 68. What is the most likely diagnosis?**
  - A. Postpartum eclampsia
  - B. Postpartum pituitary gland necrosis
  - C. Subarachnoid hemorrhage
  - D. Vascular dissection
- 5. In which condition can noninvasive ventilation be used safely?**
  - A. Altered level of consciousness
  - B. Maxillofacial trauma
  - C. Severe hypertension
  - D. Vomiting \*



*Thank you!*

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RoshReview

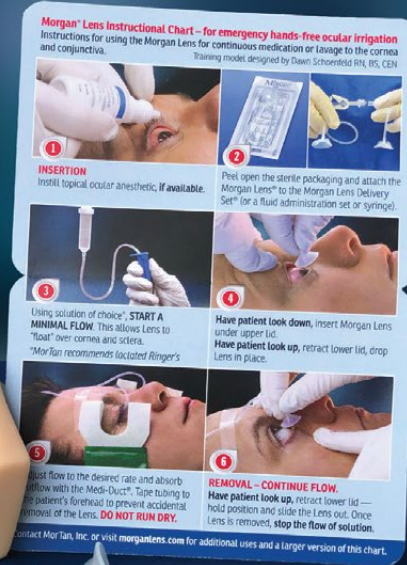




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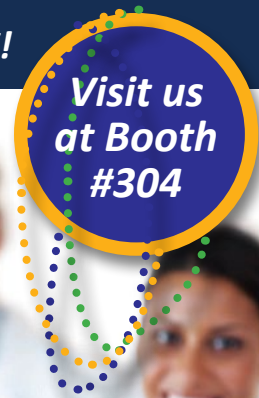
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