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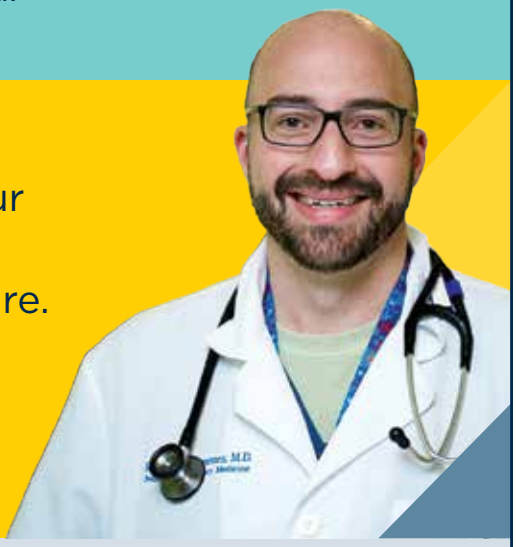

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The Evolution of the Physician's Mask

"You don't have to wear a mask around me. The pandemic is over. It's not like I have the plague."

One of my patients uttered these words to me this week in the department. I was surprised at how much it took me aback.

*"It **was** a plague,"* I thought to myself, walking out of the room. I recognize looking back that my own experiences during the pandemic gave me a raw, visceral response to those words as I heard them.

Overall, the fatality rate of the Black Death was much more severe than that of COVID-19. Despite this, both required a profound public health response. While COVID-19 may not exactly have been a plague by definition, its significant impacts have and continue to permeate the health care profession, and especially emergency medicine as a specialty. There have been several pandemics during human history, but the history of quarantining dates back to the bubonic plague itself. This stems from an inability of the medical sciences at the time to understand the disease transmission—thus an inability to educate, prevent, and treat the disease. When a disease is as highly virulent as COVID-19, it takes significant public health measures (such as quarantine) as a response.

Some of the strategies utilized to combat COVID-19 have been learned from and improved upon from these prior epidemics, including the bubonic plague. Personal protective equipment (PPE) is another important portion of control when looking into treatment of a disease process, and we are always taught in our training that if you do not protect yourself as a health care professional, you will be incredibly limited in those you can help, because you will inevitably get hurt or become ill. There is maybe no more iconic example of PPE than the beak-like masks plague physicians wore to protect themselves.

Plague physicians worked for the government, which meant they were contracted to see patients who couldn't pay and to go into the sickest of the plague-stricken neighborhoods. These physicians were often insulted and ostracized in the streets as they tried to warn and educate the public about the disease. With their cloaks, masks, and wide-brimmed hats, they waded into illness in a similar way to what we do in emergency medicine. We see everyone and anyone, all the time. We adapt to changes in the culture and disease processes. We're used to change.

The patients aren't.

In our county emergency department, we take care of a variety of patients—from those with a tuberculosis exposure or confirmed test to immunosuppressed elderly patients with pneumonia. It is pretty common for staff to put on a mask when taking care of a patient. Though there may have been significant medical advancements since prior outbreaks such as the bubonic plague, smallpox, and the Spanish flu that have given an advantage in worldwide response, a few things remain similar: public miscommunication, misinformation, and disbelief or lack of trust.

It's not as commonplace these days to see people wearing masks in public. Recent studies show that a majority of Americans believe the pandemic is over. However, ways of life have not returned completely to normal. People are adjusting to the "new normal" as we, as a society, begin to restore to pre-pandemic ways of life. As we adjust to this "new normal," we are only beginning to understand the long-lasting impacts of the COVID-19 pandemic. Though many believe it is currently over, there is still fear of re-emersion of the pandemic or of a different pandemic occurring.

There is solace in all that has been learned from the experiences during the COVID-19 pandemic, such as the research that went into rapid vaccination development and distribution. However, there is still much to be done as far as preparation for future disease. There is significant public mistrust of physicians, which is something we emergency physicians must be not only aware of, but proactively combat. We should heed the warnings of what we have already seen and look to develop trust among our patient relationships and the public, so that we can have an impact prior to any public health crisis. There is opportunity in our re-orientation as a society, and we should utilize it.

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The Value of Showing Up

David Wilson, MD
University of Cincinnati
EMRA President, 2024-2025

SO, HERE'S YOUR CALL.

Whatever your stage of training, whatever your bandwidth: show up.

Show up for your patients, your program, your peers. Show up for yourself. And if you're ready, show up for EMRA. Come to a regional event. Apply for a committee. Run for office. Write something. **Speak up. Even if your voice shakes.**

Not because it'll earn you a line on your CV—but because showing up is how we build the future of emergency medicine. It's how we support each other, especially when the system doesn't. And it's how we remind ourselves that *we* are the ones shaping what comes next.

Because emergency medicine isn't just a job. **It's a community.** And communities are built—one moment, one shift, one person at a time—by the people who keep showing up.

Emergency medicine is built on presence. We are masters of resuscitation and stabilization of the critically ill, but for many of our patients, our greatest ability is availability. Emergency medicine is rooted in **being there**: for the patient in crisis, for the team under pressure, for our colleagues in times of struggle.

As medical students and then as residents, we do a lot of showing up—at conference, on shift, showing up to take a test. It is a huge investment of time, but every day, I'm inspired by how often we show up for even more. We don't just clock in for shifts. We advocate for patients who have no one else. We teach, mentor, listen, hustle, troubleshoot, and care. We lead simulation sessions, organize wellness events, contribute to research, and write policies—all while navigating the whirlwind of training.

What I've come to realize is that s**howing up is more than a responsibility—it's a superpower, and we should strive to show up more.** It's how we take better care of patients, shape the culture of our programs, and strengthen our specialty.

SHOWING UP HELPS YOUR PATIENTS.

During training, we take care of thousands of patients. While we might like to say "treat 'em and street 'em," we do so much more for our patients. We refill critical medications, we get patients connected to primary care, we reassure patients and families when they are okay, and we comfort them when they may be dying. While much of our true responsibility is to identify and treat life- and limb-threatening illnesses and injuries, by showing up for our patients, we do so much more.

SHOWING UP CHANGES YOUR RESIDENCY PROGRAM.

Your presence carries weight. When you check on a classmate after a tough shift, offer a word of encouragement to an intern, or sit with a medical student to review an EKG, you're doing more than being kind—you're building a supportive culture that makes training better for everyone. The way we treat each other becomes part of the institutional memory. The habits of inclusion, kindness, and curiosity are contagious, and they start with someone deciding to show up and model them.

We often think about great residencies being defined by high volume and high acuity—but in reality, great residencies are defined by the culture of the program. Greatness is built by people who invest in each other—who show up for one another. Taking a shift for a co-resident working on personal issues, taking extra time to debrief a complicated case, supporting a junior resident who is challenging themselves to see more volume, or showing up to a co-resident's lecture post-nights: This culture makes training programs excellent.

SHOWING UP STRENGTHENS OUR SPECIALTY.

Emergency medicine is evolving. There are real challenges ahead: workforce trends, debates about training length, and questions about how we protect the well-being of our clinicians. This moment of change is also an opportunity. Showing up allows us to shape our future.

You don't have to be an expert to have a voice. Start by joining a town hall. Come to EMRA's Representative Council meetings. Ask a question. Submit an idea. Volunteer for a committee. Write a letter. **EMRA exists because people like you decided to raise their hands, not because they had all the answers, but because they cared enough to be part of the conversation.**

Time and again, I've seen students and residents show up for something small and discover a larger purpose. One resolution. One committee meeting. One shared idea. That's all it takes to start building impact that lasts far beyond residency.

SO, HERE'S YOUR INVITATION.

Whatever your level of training, wherever you are in your journey: show up. Not out of obligation, but out of curiosity, purpose, and the knowledge that your presence matters. Whether it's supporting a co-resident, joining an EMRA event, mentoring a student, or raising your hand in a meeting, your presence will make us all better. Emergency medicine thrives because of people like you who show up.

References available online.

Case Report: Severe Finger Entrapment in Gas Filler Cap Valve

A 48-year-old female with no contributing past medical history presented to the emergency department with her left index finger trapped in the gas filler cap valve of a vehicle.

HISTORY OF PRESENT ILLNESS

The patient was attempting to inspect the gas filler area of her vehicle when her left index finger became lodged in the gas filler cap valve. Despite multiple attempts to free the finger, including the use of lubrication, Detroit emergency medical services were unable to extricate the finger and sought assistance.

The Detroit Fire Department was called to the scene and initially attempted to cut open the gas access door and pipe to facilitate removal. Despite these efforts, the finger remained trapped, leading to her transfer to the Henry Ford Hospital emergency department for further intervention.

Figure 1. The patient arrived at the ED with her left index finger stuck in the gas filler cap valve. Sterile gauze tied to an IV pole was used to stabilize the patient and machinery to facilitate inspection.

PHYSICAL EXAMINATION

The left index finger was swollen, tender to palpation, and visibly trapped within the gas filler valve. There was worsening pain with any attempt to free the finger by attempting to open the filler valve. The left radial pulse was able to be easily palpated, and all soft tissue compartments of the finger, hand, and proximal left upper extremity were soft. The gas filler valve and finger were carefully inspected with flashlight and, ultimately, fiber-optic nasopharyngeal scope. Endoscopic evaluation showed the gas cap valve itself was tightly secured around the finger on the hinge side.

Figure 2. The view of the patient's hand entering the gas filler cap.

Figure 3. An external view of the left index finger exiting the gas filler cap to be stuck within the valve mechanism.

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CONSIDERED INTERVENTIONS

Digital nerve block was deferred in order to preserve frequent re-assessment of the finger's neurovascular status, particularly in the setting of anticipated heat-generating removal procedure. There was also concern that performing a digital nerve block could worsen swelling and possibly make the finger more difficult to extricate or possibly precipitate compartment syndrome over time with further swelling from the injury. The patient responded well to serial doses of morphine and midazolam while finger was entrapped.

References available online.

FAILED INTERVENTIONS

- **Lubrication:** Initial attempts to free the finger in the field using lubrication, such as with petroleum jelly, were unsuccessful. As the patient's finger was stuck in the hinge mechanism of the gas filler cap valve, which was not known until later endoscopic visualization in the emergency department, lubrication and attempts at opening the gas filler cap valve were unsuccessful.
- **Bougie and Suture Removal:** In the emergency department, prior to endoscopic visualization, running a bougie and suture through the gas filler cap valve were attempted to better open the valve to facilitate extrication. As above, this method failed due to the nature of the entrapment.
- **Operating Room Equipment:** The patient's case was discussed with orthopedic surgery regarding any OR equipment that could help facilitate freeing the finger. After discussion, it was determined that their bolt cutters and bone saws would not be effective to extricate the finger.

SUCCESSFUL INTERVENTIONS

- **Endoscopic Visualization:** An endoscopic approach was utilized to better understand the nature of the entrapment. A fiber-optic nasopharyngeal scope was inserted into the gas access pipe and advanced until the finger and valve were visualized, revealing that the tip of the finger was trapped in the valve hinge. This visualization allowed for a more precise understanding of the entrapment mechanics and explained why previous attempts were unsuccessful and often led to worsening pain experienced by the patient.
- **Angle Grinder Utilization:** Due to the persistent nature of the entrapment and the failure of previous methods, an angle grinder was employed to cut through the gas access pipe connecting to the gas filler cap valve. The facilities department at the hospital was initially unreachable in the search for an angle grinder. Local hardware stores in downtown Detroit did not stock angle grinders, and nurses who lived nearby did not have one at home. The Detroit Fire Department would not agree to return to the ED to allow us to use their angle grinder. It was decided to use DoorDash to facilitate delivery of an angle grinder or a reciprocating saw. While DoorDash was en route, a facilities employee brought an angle grinder to the ED. This approach was chosen to facilitate safe and effective removal of the finger. The procedure was conducted with mild sedation to minimize discomfort. After the gas access pipe was cut, the gas filler cap valve hinge was cut with wire cutters, and the finger was freed from the gas tank.

Figure 4. Endoscopic visualization with a flexible fiberoptic nasopharyngeal scope reveals the finger to be caught in the hinge mechanism of the valve.

PROCEDURE DETAILS

The gas access door and pipe were carefully cut from the rest of the car by the Detroit Fire Department in the field to allow access to the gas filler cap valve. In the emergency department, the angle grinder was used to precisely cut and remove sections of the gas access pipe, creating sufficient space to visualize the finger in the gas filler cap valve, and the finger was subsequently freed by cutting the valve hinge with wire cutters. The procedure was performed under unsterile conditions and was performed 3 hours past the initial entrapment time, allowing for volatile gasoline vapors to be eliminated. Wet towels were used to protect the patient from sparks. Extra wet towels were available in case of fire, which was low risk. Saline was applied to the cutting surface via IV tubing to minimize heat production. The gas

Figure 5. An angle grinder was used to cut the gas access pipe. Wet towels were used to protect the patient and staff, and saline was applied via IV tubing to decrease heat production.

Figure 6. After cutting the gas access pipe, easy access was obtained to the patient's finger within the gas filler cap valve.

filler cap valve and finger were carefully examined post-removal for any residual debris or damage.

POST-PROCEDURE CARE

The patient's finger was cleaned, and an ice pack was applied to reduce swelling. Post-procedural X-ray was negative for fracture, dislocation, and radio-opaque foreign body. The finger was bandaged and closely monitored for signs of infection or further complications during the remainder of the patient's emergency department stay. Pain management and follow-up care instructions were provided to the patient, including monitoring for signs of infection and changes in sensation.

References available online.

Figure 7. A focused image of the patient's finger within the gas filler cap valve hinge after the gas access pipe was cut.

Figure 8. The hinge of the gas filler cap valve was cut with wire cutters, was removed, and the hand is subsequently visualized to be within the gas filler cap.

Figure 9. The gas filler cap and gas access pipe are shown after removal from the patient.

Figure 10. The patient's finger is visualized immediately after freeing from the gas filler cap valve.

OUTCOME

The patient experienced relief following the procedure and showed no immediate complications. The patient signed consent for publication purposes and was lost to follow up.

DISCUSSION

This case illustrates the complexity of managing severe entrapment incidents involving automotive components. The use of an angle grinder, while effective, underscores the need for a methodical approach and readiness to employ advanced techniques when initial interventions fail. Endoscopic visualization played a crucial role in identifying the exact nature of the entrapment, guiding the subsequent treatment strategy.

CONCLUSION

The patient's severe entrapment was successfully managed through a combination of emergency services and advanced medical techniques. The case highlights the importance of a comprehensive approach in handling complex entrapment scenarios and the need for both initial and advanced intervention strategies.

TAKE-HOME POINTS

- **Need for increased public and provider awareness regarding safety measures when interacting with automotive components**
- **Training for emergency responders regarding handling unusual entrapment situations with appropriate tools and techniques**
- **Use of fiber-optic nasopharyngeal scopes to better visualize and diagnose entrapment situations**
- **Creative resource utilization such as hospital facilities, employee personal tools, and delivery services**
- **Fire safety when performing heat and spark generating procedures near hospital oxygen and gasoline fumes**

References available online.

Onset of Behçet's Disease Following COVID-19 Infection:

The Role of Emergency Physicians in the Diagnosis of Rare Diseases

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This case represents a classic progression of a rarely diagnosed disease and showcases the role of emergency physicians in ensuring patients with rare pathology receive appropriate care and diagnoses during evolving symptomatology.

CASE PRESENTATION

A 51-year-old male with past medical history of tobacco use, hyperlipidemia, and bipolar disorder presented to the emergency department for eye pain, eye drainage, and sore throat 11 days after being diagnosed with COVID-19. Initial symptoms of COVID-19 were mild and included fatigue, headache, and non-productive cough. However, 6 days after positive PCR testing, he noticed worsening bilateral conjunctival injection, yellow eye drainage, sore throat, dysuria, and decreased appetite. He did not report fevers, chest pain, shortness of breath, myalgias, arthralgias, or GI complaints. The patient was not vaccinated against COVID-19.

Initial workup showed slight elevations of AST and ALT, significantly elevated CRP of 15.11mg/dL, proteinuria, and urobilinogen. Chest X-ray was unremarkable. Exam was notable for severely injected conjunctiva bilaterally with thick yellow discharge. Skin exam showed multiple circular erythematous macules to the left anterior leg. Oral exam showed few aphthous ulcers and was negative for erythema or tonsillar exudates. He was well-appearing with tachycardia that improved with 1L of crystalloid fluids. Symptoms were felt to be due to viral conjunctivitis, but given eye discharge he was prescribed polymyxin B/trimethoprim eye drops for superimposed bacterial infection. He was discharged and instructed to follow-up with his PCP and ophthalmology.

The patient returned to the ED the following day due to worsening symptoms. Sore throat had progressed significantly, and he now reported hoarseness, increased oral lesions, and pain that limited his ability to eat, swallow, and speak. Bilateral eye pain, swelling, and discharge continued to worsen despite treatment with new bilateral subconjunctival hemorrhages. He reported spreading of lower extremity rash to involve bilateral legs and upper extremities. On the day of the second ED visit, he noticed dysuria and a small amount of blood from his urethra after urination. The patient denied other symptoms including myalgias or arthralgias. He had been treated with lamotrigine for bipolar disorder for several years and denied any new medications. He denied a history of STIs, UTIs, or being currently sexually active.

On evaluation, the patient was in obvious discomfort with speaking and swallowing secretions. Oral exam showed diffuse aphthous ulcerations of the mucosa, swelling, and tenderness to palpation **(Fig. 1-3)**. Eye exam was notable for bilateral subconjunctival hemorrhages, yellow discharge, photophobia, and periorbital erythema and swelling **(Fig. 4)**. Genital exam showed thin yellow discharge at the urethral meatus with tenderness to palpation of distal penis. Circular, nonpruritic macules were present over the bilateral anterior legs **(Fig. 5)**. Work-up was notable for AST of 90U/L, ALT of

References available online.

Image 1: Oral Lesions

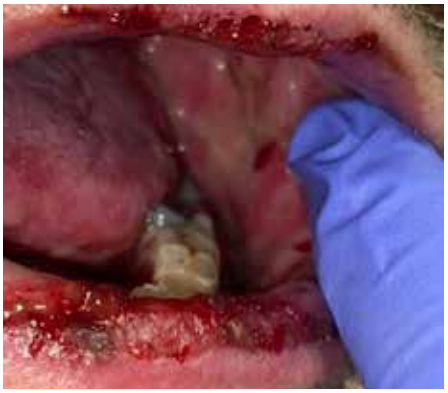

Image 2: Oral Lesions

Image 3: Oral Lesions

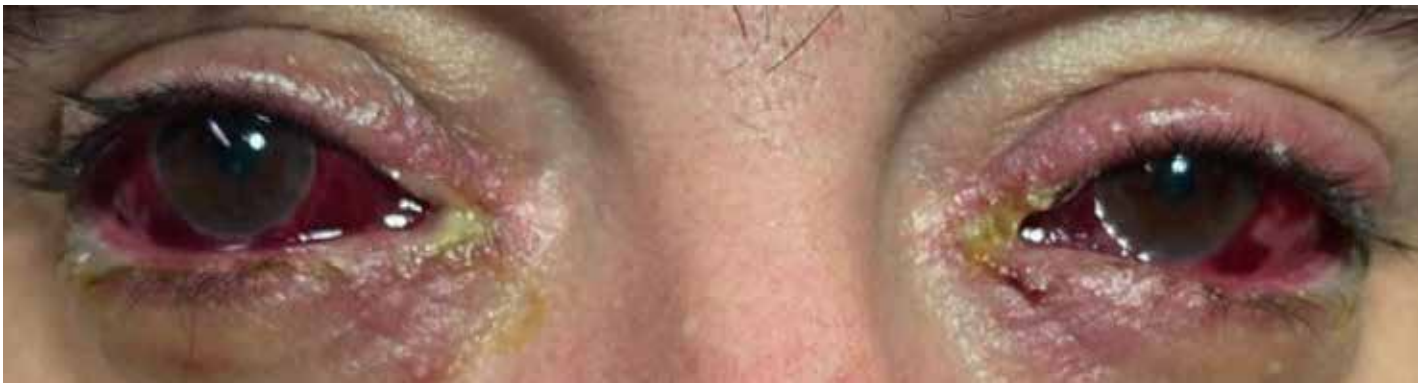

Image 4: Ocular Findings

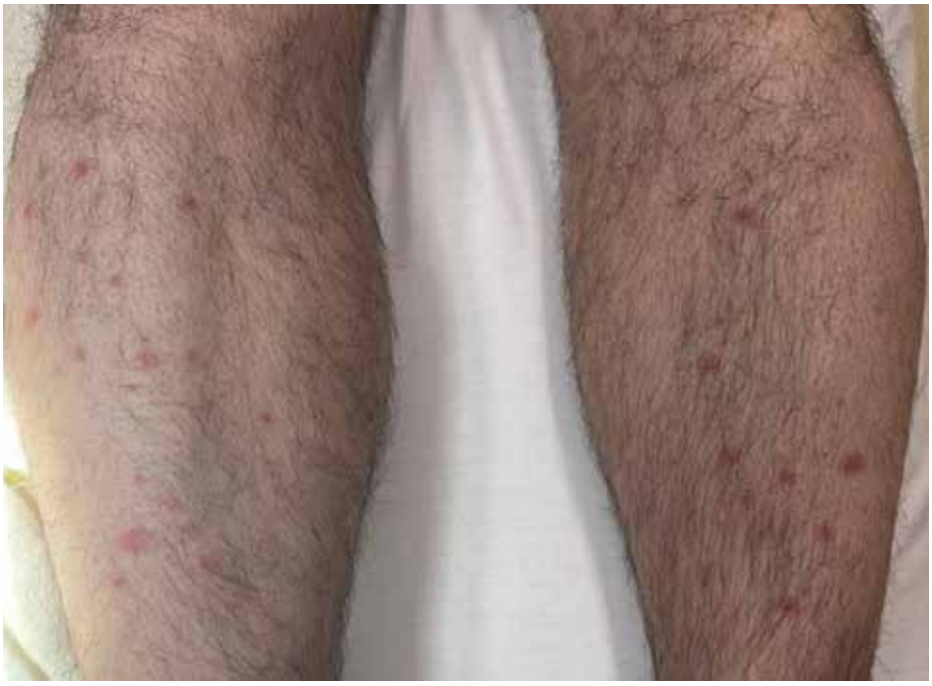

Image 5: Rash

72U/L, ESR of 38mm/h, CRP 18.44mg/dL, fibrinogen of 745mg/dL, UA showing 2+ protein, and 11-20 WBCs and RBCs. Gonorrhea and chlamydia testing was negative. The patient was given ketorolac, morphine, and oral viscous lidocaine for pain.

BACKGROUND

Behçet's disease (BD) is a rare, multisystem inflammatory disease characterized by vasculitis of all vessel sizes. The exact cause of and underlying pathophysiology of BD is unclear, although there are established risk factors including genetic predisposition, male sex, immunological triggers including viral infections or vaccinations, age, and ethnicity. While it is thought to be autoinflammatory in nature, there is no evidence that specific autoantibodies are involved in the development of BD.[1-3] Incidence and prevalence vary widely across geographical areas, with an estimated incidence of 0.58 per 100,000 in Middle Eastern and Japanese populations, and incidence of 0.24 per 100,000 in European populations. Studies in Western populations are limited but show an approximate incidence of 0.38 per 100,000 in the Midwest United States.[6] Diagnosis of BD is clinical, based on the presence of classic symptoms and supported by a relapsing and remitting course of disease. Multiple

References available online.

diagnostic criteria have been developed, with the most established being the International Criteria for Behçet's Disease which uses a scoring system based on symptoms of oral aphthosis, vascular lesions (DVT, LVT, arterial thrombus, superficial phlebitis, and aneurysm), genital lesions, inflammatory eye disease (anterior uveitis, posterior uveitis, and retinal vasculitis), rash, and a positive pathergy test.[4]

More serious complications include bowel perforation from GI ulcerations, ruptured coronary, pulmonary, or peripheral aneurysms, pulmonary embolism, stroke, dural venous thrombosis, meningoencephalitis, and blindness. Due to the rarity of BD, multisystem involvement, and relapsing and remitting symptoms, diagnosis is clinically challenging. Patients on average have symptoms for 5.3 years and meet diagnostic criteria for 1.1 years before an official diagnosis is made.

Timely diagnosis and treatment with appropriate, symptom-based therapy is essential in reducing patient morbidity and mortality along the disease course.[2] First-line treatment is colchicine +/- apremilast and may include oral, topical, and ophthalmologic steroids. Severe or refractory symptoms require treatment with azathioprine, interferon-alfa, cyclosporine, methotrexate, and TNF-alpha inhibitors.[3]

DIFFERENTIAL DIAGNOSIS

The differential diagnoses for a patient who presents with oral ulcers, rash, ocular, and urinary symptoms are broad and present a diagnostic challenge. Differential diagnoses including HSV, Steven-Johnson syndrome (SJS), reactive arthritis, lupus, measles, and (in the setting of COVID-19 infection) multisystem inflammatory syndrome must be considered. While rare, it is important to consider the various forms of vasculitis including BD, polyarteritis nodosa, and microscopic polyangiitis depending on the patient's age and demographics. In our patient, history of treatment with lamotrigine increased suspicion of SJS; however, the clinical presentation and symptoms were not typical. Multisystem involvement and recent viral infection helped to narrow our differential diagnosis, and the primary concerns were for reactive arthritis and MSIS.

DIAGNOSIS AND TREATMENT

Infectious disease was consulted in the emergency department for known COVID-19 and suspected MSIS, as at the time of patient's presentation, treatment options were rapidly evolving. In the setting of oral aphthous ulcerations, GU symptoms, anterior uveitis/conjunctivitis, and rash following viral infection in a middle-aged male, BD was considered the most likely diagnosis. Using the International criteria for Behçet's disease, the patient met the criteria score of ≥4 for the following symptoms: oral aphthous ulcers (2pts), ocular manifestation (2pts), rash.[1] Although the patient was complaining of penile pain, no genital ulcerations were noted at the time of evaluation.

Given the severity of symptoms and inability to tolerate oral hydration, the patient was admitted to the hospital for initiation of treatment and supportive therapy. He was given a 3 g loading dose of colchicine, followed by 0.6 mg TID and started on 5 mg prednisone daily. During hospital stay, he had improvement in symptoms and was discharged to home on colchicine and prednisone with ophthalmology and rheumatology follow-up. Steroids were tapered after one month and after several months without symptoms, colchicine was tapered as well.

DISCUSSION

Behçet's disease is a rare vasculitis that affects large, medium, and small vessels leading to multisystem involvement and a wide variety of symptoms. It is a diagnosis that often requires multiple health care visits for identification and requires appropriate treatment to prevent significant long-term morbidity and increased risk of mortality.

This case demonstrates a classic presentation of BD diagnosed in the ED after 2 visits for worsening oral ulcerations, anterior uveitis, rash, and genital pain in the setting of a recent infection. While long-term management is outside of the scope of the emergency physician, this case demonstrates the importance of identifying abnormal pathology or clinical courses requiring subspecialist consultation and hospitalization. Following appropriate diagnosis and treatment, our patient recovered without long-term effects.

LEARNING PEARLS

- **Behçet's disease is a rare condition that may go undiagnosed for years and carries a high risk of morbidity and mortality.**
- **Patients with Behçet's disease most often present with oral aphthous ulcers and ocular disease early in disease course and may have additional organ involvement including rash, genital ulcers, arthritis, and vascular pathology.**
- **Behçet's is most often diagnosed in the 2nd-4th decades of life but may occur at any time and often has a preceding immunological trigger including viral infection or recent vaccination.**
- **Behçet's disease can lead to significant complications including blindness, aneurysms, bowel perforation, or meningoencephalitis, and patients may require hospitalization for initiation of treatment or stabilization. Prompt diagnosis and treatment is essential in preventing long-term sequelae.**

References available online.

Silent Strangulation: A Unique and Unpredictable Encounter of a Spontaneous Thyroid Hematoma in the Emergency Department

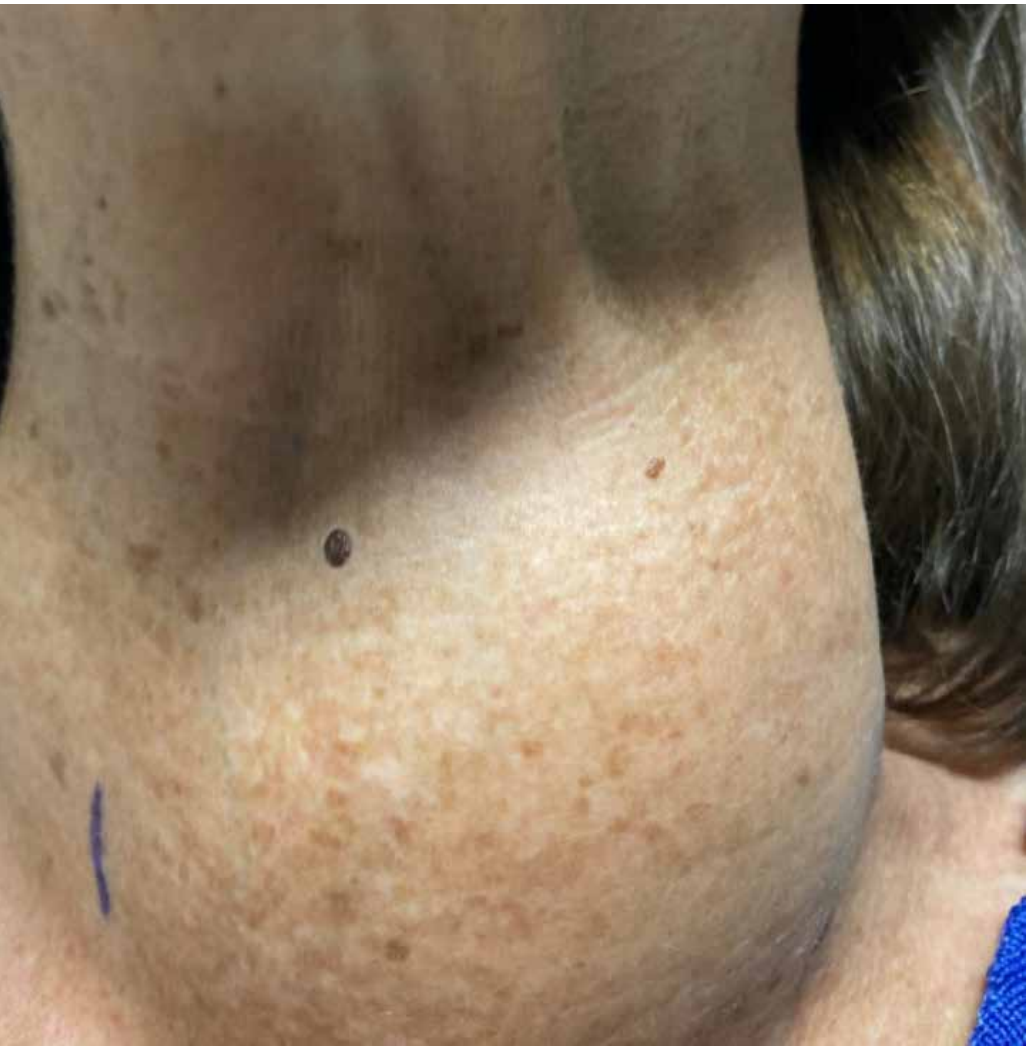

Figure 1. Expanding neck mass upon exam

Airway management of expanding neck hematomas can challenge even the most expert emergency clinicians. A myriad of decisions contributes to the complexity of these cases, such as timing of intervention, medications for induction, ancillary resources, equipment, and clinical picture.

Literature is robust in the discussion of expanding neck hematomas stemming from trauma or post-surgical complications. In this case, we highlight a unique presentation of a 67-year-old female presenting with a spontaneous thyroid hematoma originating from a thyroid nodule, and the distinctive challenges this presented that subsequently weighed heavily into management decisions. This case report highlights the importance of preparedness and management considerations in an atypical presentation of an expanding neck mass.

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INTRODUCTION

The thyroid gland is a highly vascularized organ that encircles the anterior portion of the trachea, with the thyroid isthmus crossing just below the cricoid cartilage. Due to its location, acute expansion of a thyroid hematoma has the potential for airway compromise, and potentially fatal outcomes.

Previous literature points to factors such as anticoagulation,[1] intubation,[3] and trauma[4,5] as risks of developing an acute thyroid hematoma. The goal of this case is to highlight a more unique presentation of spontaneous hemorrhage without clear risk factors. Additionally, we aim to discuss the management considerations of an expanding neck or thyroid hematoma, and specifically highlight the unique characteristics of this presentation that guided patient care.

References available online.

CASE PRESENTATION

A 67-year-old female with a past medical history of hypothyroidism initially presented to a small community emergency department with dysphagia and expanding pulsatile neck mass. The patient had awoken 30 minutes before arrival with marked swelling along the left anterior neck without any historical reports of trauma or recent surgery. Upon initial presentation to the outside hospital, the patient was noted to be without any evidence of respiratory distress, wheeze, stridor, or vocal changes. Examination of her neck revealed a firm pulsating mass **(Figure 1)**.

The patient was immediately taken to CT, where a CT soft tissue neck without contrast was performed. Findings of CT neck revealed a dense mass within the left lower neck arising from the left thyroid lobe compatible with a hematoma measuring 97 X 82 X 71 mm with associated rightward displacement and effacement of trachea. The greatest narrowing was below the level of the chords **(Figure 2)**. The patient was subsequently transported to the tertiary referral center for emergent ENT evaluation due to concern for airway compromise.

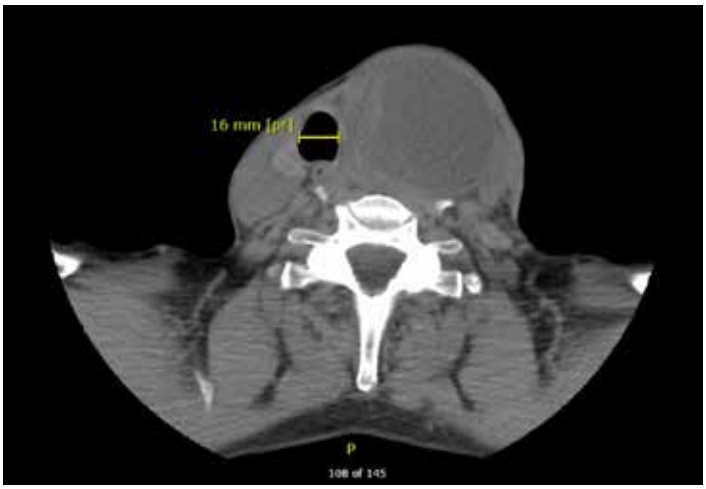


Figure 2. CT imaging

On arrival at the tertiary care center, the patient had no further expansion of the hematoma, and remained without any subjective evidence of respiratory distress. Initial evaluation included a point of care ultrasound (POCUS) to evaluate for active bleed as the initial CT scan was performed without contrast. POCUS findings revealed a well circumscribed mass with mixed echogenicity without evidence of active bleeding on color flow **(Figure 3)**. Anesthesia and ENT were immediately consulted for co-management.

After interdisciplinary deliberation, a decision was made to forgo airway management in the ED, and the patient was taken to the OR with anesthesia and ENT for intubation and clot evacuation. Anesthesia performed sedative-only induction with the use of a video-assisted laryngoscope. A 5.5 reinforced endotracheal tube was passed without resistance, and a flexible bronchoscope was then utilized to ensure no sign of any significant tracheal compression. Decompression of the mass was achieved via aspiration with an 18-gauge needle, where 30 mL of pink serous fluid was evacuated with significant decompression of the swelling. No obvious venous or arterial blood was observed, and the patient was taken to the ICU intubated with plans to be extubated in the morning. Surgical cytology results of the fluid collected showed abundant bloody fluid with no follicular cells present. The patient was then taken to OR 5 days later for a thyroid lobectomy due to some smaller re-accumulation of fluid.

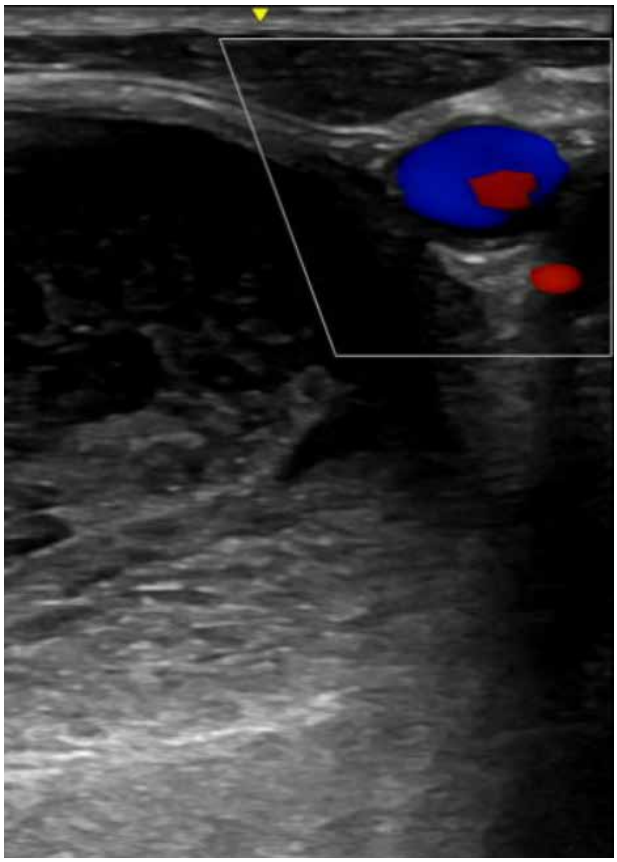


Figure 3. Ultrasound findings

DISCUSSION

Several case reports have been found on spontaneous thyroid hemorrhages. However, most cases involved the use of anticoagulation or blunt trauma to the neck. Interestingly, neither of the above are factors in this case. The literature has described some mechanisms for hemorrhage into thyroid nodules. Abnormal vascular anatomy may result in weakening of the veins or arteriovenous shunting into the nodule.[6,7,8] However, even in the circumstances of abnormal vascularity or anticoagulation, hemorrhage is often linked to an inciting event such as trauma, or extreme exertion.[1] Slow growth of thyroid nodules appears to be a risk factor for an acute airway obstruction if hemorrhage occurs into these nodules and is a factor in this case, as the patient noted undulating growth over 18 years.[2]

Many traumatic causes are due to fine needle aspiration biopsies, but usually spontaneous cases involve anticoagulation, where they do not require emergent surgery or intubation, and instead can be treated with drainage and elective thyroidectomy.[3,9] Thyroidectomy also presents such risks of anterior neck hematomas and some of the risk factors include vomiting, hypertension, constipation, coagulopathies.[11]

References available online.

The case outlined in this report contains many unique characteristics of an expanding neck hematoma:

- **Spontaneous nature without clear risk factors;**
- **Initial presentation to a community hospital with limited ancillary resources;**
- **Anatomic location of the airway compromise.**

Given the spontaneous nature, the source of the expanding neck mass was not immediately clear. The initial diagnostic evaluation was a non-contrast CT of the neck, and in retrospect, an argument can be made for consideration of either a CT angiogram or a CT soft tissue neck with contrast to evaluate for potential extravasation. There was a critical role for POCUS in this situation, which allowed for prompt visualization of the major blood vessels of the neck as seen in Figure 3. Further consideration should occur regarding the benefit of POCUS in the diagnostic evaluation of expanding neck hematomas, especially in time- and resource-limited circumstances.

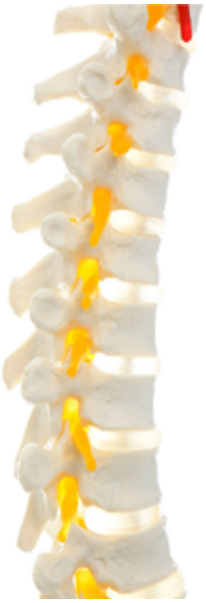
The location of the compression was mid-trachea and well below the level of the vocal cords, causing significant displacement of the airway with a narrowing to 7 mm due to mass effect (Figure 2). This factor cannot be underestimated, as a traditional endotracheal intubation may be insufficient to secure the airway due to 1) distal nature of the compression and 2) the ultimate necessity of a rigid endotracheal tube utilized by anesthesia.

These factors, when combined, ultimately weighed heavily on the decision to defer airway management in the emergency department both from the sending facility and at the tertiary referral center. Upon arrival at the tertiary referral center, anesthesia and ENT were immediately consulted for co-management. The consensus was that for further airway compromise, the immediate plan would be to proceed to secure the airway along with immediate decompression with an 18-gauge needle or incision with a scalpel.

CASE CONCLUSION

Delayed sequence intubation was discussed with anesthesia via video assisted laryngoscope or fiberoptic scope, but ultimately this decision was delayed until the operating room. Additional tools were made available such as a cricothyrotomy kit; however, this was complicated by the anatomic variation given the tracheal deviation. POCUS was utilized to identify the cricothyroid membrane and marked with a skin marker **(as seen in Figure 1)** if this pathway became necessary while the patient was in the department. The decision to delay intubation proved most advantageous for patient care, but the decision must be viewed within the limitations of the uniqueness of this case.

References available online.



Navigating the Diagnostic Challenge: Recognizing Spinal Epidural Abscesses in Emergency Medicine

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The incidence of SEA is approximately 2 to 8 cases per 10,000 hospital admissions, and rising as our diagnostic capabilities have improved. Delay in diagnosis and treatment can result in permanent neurological damage, significant morbidity, and medicolegal cost.

As a future or current emergency physician, you will frequently encounter patients presenting with back pain. A recent AHRQ review identified back pain as the fifth most common reason for visits to the ED.[1]

According to a meta-analysis in BMC Musculoskeletal Disorders,[2] up to 17.1% of all ED visits are related to back pain. In the United States, 8 out of 10 people will experience back pain at some point in their lives,[3] and of those, 5% will go on to suffer from chronic back pain.[4] Back pain-related visits are also quite common in clinical settings, being the third most common reason for seeking medical care.[1] This results in a staggering cost of $635 billion annually to the U.S. economy, encompassing direct medical costs, lost wages, and employer-related charges.[1]

However, a small percentage of these patients have a more serious underlying cause for their back pain than a simple musculoskeletal diagnosis. These presentations do not always follow a classic pattern, and providers must maintain a high index of suspicion. Among these, spinal epidural abscess (SEA) remains one of the most challenging diagnoses, especially early in the disease process. If undiagnosed, SEA carries a significant risk of disability.

So, on our next shift in the ED, when should we be more concerned? Are there certain associated conditions that should catch our attention? What are some red flag signs that warrant further workup, and how can we recognize these serious causes, especially early on, when they are the most difficult to diagnose?

EPIDEMIOLOGY

Spinal epidural abscesses can be extremely difficult to diagnose. We are taught to look for the "classic triad" of spine pain, fever, and neurologic symptoms; however, only about 10% of patients with confirmed SEA present with all three. SEA is progressive in nature, and as the abscess grows, it will impact the nerve roots and spinal cord more significantly. Thus, the later in the development of these symptoms, the more likely it is that all three "classic" signs will be present. Early on, you will face the diagnostic challenge of back pain alone and must look for risk factors to stratify patients into low, moderate, and high-risk categories. These cases are particularly challenging because, although the incidence is low, the morbidity and mortality are extremely high.

Due to the complexity and urgency of diagnosing SEA, many studies have identified key diagnostic findings to help narrow the differential and spot these elusive conditions in time to preserve the patient's quality of life.

CLASSIC PRESENTATION

Consider a 63-year-old male with a medical history of degenerative joint disease (DJD), psoriatic arthritis, and Type 2 diabetes mellitus (DM2) who presents to the ED with worsening low back pain, fever, and inability to ambulate. One week ago, he visited the ED with complaints of chronic low back pain and later went to an outpatient clinic where he received a steroid injection in his left hip. Today, the pain is

References available online.

sharp, located in the left lower back and flank, and radiates to his left groin.

UNDERSTANDING SEA

The rate of progression of SEA is not standard and depends on vertebral anatomy. For example, stenosis can lead to faster progression. Four stages have been described in the natural history of the disease:

- **Stage 1:** Back pain, fever, or spine tenderness
- **Stage 2:** Radicular pain and nuchal rigidity
- **Stage 3:** Neurological deficits
- **Stage 4:** Paralysis

The goal is to catch SEA before it reaches stage 4, ideally in stage 1 or 2. The mechanisms of formation and spread of SEA primarily include hematogenous dissemination (over 50%), contiguous spread (10-30%), and direct inoculation (15%). The remainder of cases are idiopathic.[5]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for back pain includes musculoskeletal issues, disc-related problems, infections, and systemic conditions.

DIAGNOSTIC WORKUP

History and Physical Examination

The physical exam in these patients is frequently exceedingly difficult due to pain. Additionally, many of these patients may suffer from ongoing or chronic pain from previous back surgeries or other medical issues. It is crucial to determine which symptoms, if any, are new and which are chronic. A disciplined and detail-oriented approach to the history and documentation of the physical exam is vital. This is not only important from a documentation standpoint but also helpful medicolegally. Some neurologic functions lost due to SEA may not be recovered, underscoring the importance of thorough documentation at the time of arrival.

Engage EMS in patient care, as they frequently see patients move or ambulate prior to arrival. Did the patient require assistance? Did you witness them ambulate, and if so, did they limp or stumble? Was any family present on the scene who mentioned new symptoms, trauma, fever, or recent surgery?

Work with the patient. As mentioned, these patients are often in a great deal of pain, making it challenging to obtain a detailed history. Treat their pain, then revisit the history. Ask about symptoms, onset, weakness, numbness, tingling in extremities, anticoagulant use, regular back pain, and how this episode differs.

Two key pieces of history that can be easily overlooked are recent ED visits related to these symptoms and incontinence. Patients with SEA often have a history of recent ED visits that initially seem non-specific. However, repeat visits with progression of symptoms should alert the provider. Unpack incontinence, as it can have multiple causes unrelated to SEA or cauda equina, such as stress incontinence, pelvic floor dysfunction, or mixed etiology secondary to illness like a urinary tract infection.

You will need to keep a sharp eye out for new or developing symptoms and document the timeline of symptom development and previous encounters.

IMPORTANT HISTORICAL QUESTIONS

For EMS

- Could the patient ambulate?
- Did they require lift assist, and if so, how much?

For Family

- What have you noticed regarding symptom progression over time?

At Bedside

- What is new?
- Timing and onset
- How long has it been present?
- Document neuro deficits

When assessing a patient with back pain with SEA in mind, carefully look for the following:[6]

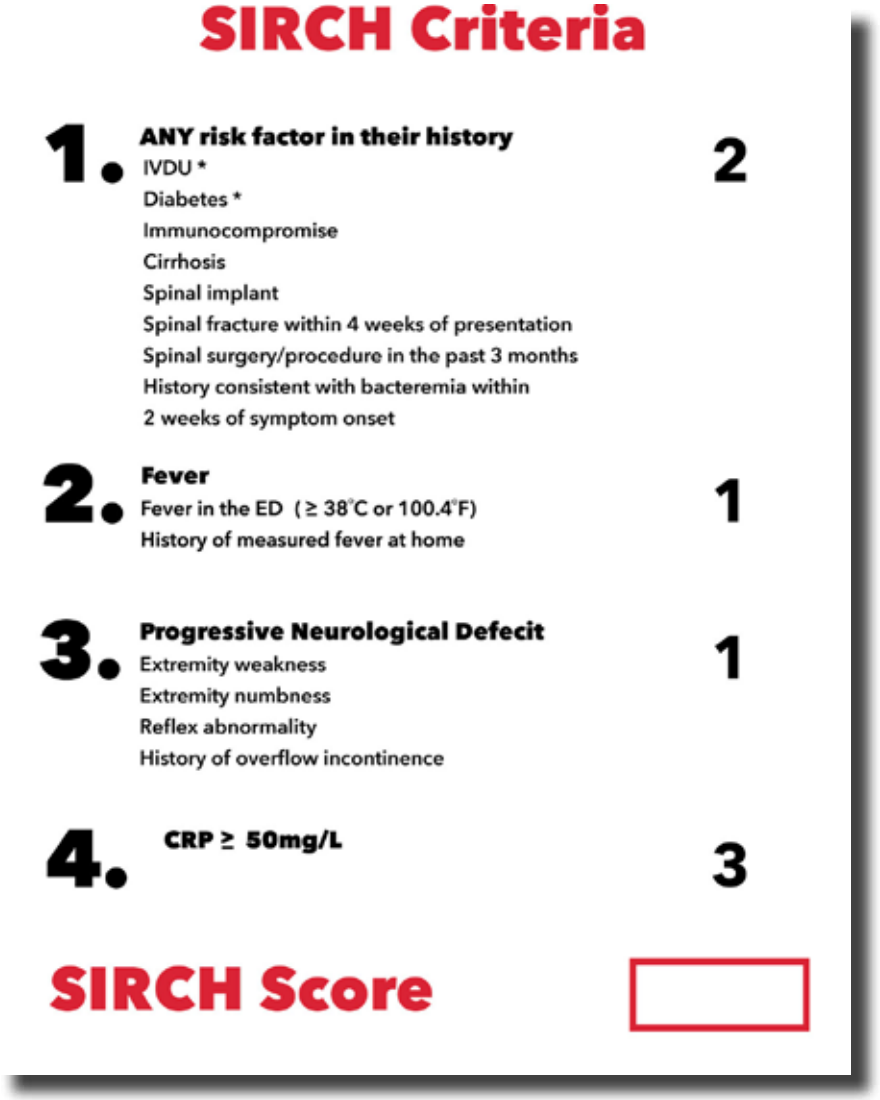
- Percuss the spinous processes for tenderness. This is important for documentation, but in the setting of worsening or progressing symptoms, lack of tenderness should not prevent further workup. Point tenderness is a red flag for infection and fracture.
- Test for saddle anesthesia by examining the sacral nerve roots. Sensation to light touch and pinprick in the perineum, posterior thigh, and perianal region is supplied by the S2-S4 dermatomes. Early in the disease process, patients may have minimal decrease in sensation.
- Perform a digital rectal exam to assess rectal tone and sensation. Document the presence or absence and any associated sensory loss.
- Assess for fever (>100.4°F) or signs of infection.
- Check for bilateral or multi-level neurologic findings in the lower extremities and assess for gait disturbances.

Inflammatory markers should be examined in the appropriate clinical setting. WBC is often not elevated and nonspecific. If clinical suspicion is high, continue with the workup despite normal WBC levels. ESR/CRP (when available) are helpful in the risk stratification process but should only be used without other ongoing inflammatory or infectious processes.[5]

The gold standard for diagnosing a spinal epidural abscess remains MRI with contrast. It is advised to obtain a pan view of the spine to look for skip lesions, which may occur in up to 13% of patients. Although imaging is necessary for a complete diagnosis, it is up to the clinician to decide which patients need imaging.

A study by Shroyer et al. in 2021[8] aimed to address the diagnostic challenges of spinal epidural abscesses by developing a tool called SIRCH (Spine Infection Risk Calculation Heuristic). The SIRCH assessment score can be used at the bedside to determine if the patient

References available online.



fits the criteria for suspected pyogenic spinal infection that requires imaging. The tool was derived from a two-part observational cohort study over six years, involving patients with low back pain. The SIRCH score uses four clinical variables to predict SEA or other spinal infections. If a patient has a SIRCH score of ≥ 3, it is best to obtain a complete spine MRI with contrast of the patient. A lower SIRCH score lowers the suspicion of a spinal abscess.

Improving the early diagnosis of spinal epidural abscesses in clinical practice involves several strategies aimed at increasing awareness, enhancing diagnostic accuracy, and ensuring timely intervention. Here are some key approaches:

1. INCREASE AWARENESS AND EDUCATION

- **Training and Continuing Education:** Regular training sessions and continuing medical education programs can help increase awareness about SEA, its risk factors, and its clinical presentation.[9]
- **Clinical Guidelines:** Disseminating updated clinical guidelines and protocols for the diagnosis and management of SEA can help standardize care and improve early recognition.[9]

2. RISK STRATIFICATION

- **Identify High-Risk Patients:** Recognize patients with risk factors such as diabetes mellitus, intravenous drug use, recent spinal surgery, or immunosuppression. These patients should be closely monitored for signs of SEA.[10]
- **Use of Scoring Systems:** Implementing scoring systems like the SIRCH (Spine Infection Risk Calculation Heuristic) can help stratify patients based on their risk and guide the need for further diagnostic workup.[9]

3. THOROUGH CLINICAL EVALUATION

- **Detailed History and Physical Examination:** A comprehensive history and physical examination are crucial. Pay attention to symptoms like severe localized back pain, fever, and neurological deficits, even if they are subtle.[10]
- **Engage EMS and Family:** Involve EMS personnel and family members in the history-taking process to gather information about the patient's symptoms and functional status prior to arrival.[9]

4. UTILIZE DIAGNOSTIC TOOLS EFFECTIVELY

- **Inflammatory Markers:** Use inflammatory markers such as ESR and CRP to aid in the risk stratification process. While these markers are not specific, they can support clinical suspicion.[10]
- **Imaging:** MRI with contrast is the gold standard for diagnosing SEA. Ensure timely access to MRI for patients with high clinical suspicion.[11]

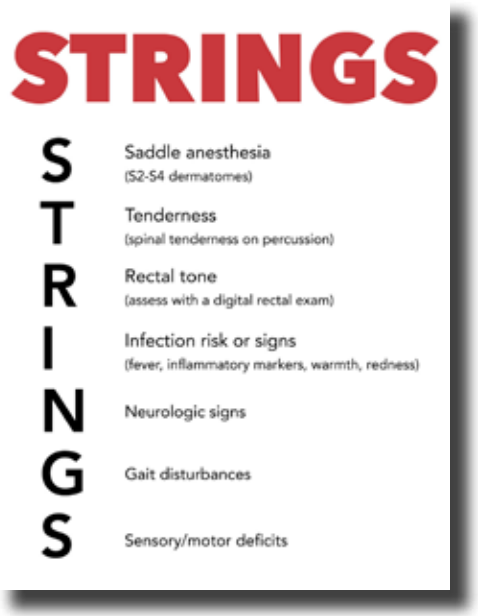
5. MULTIDISCIPLINARY APPROACH

- **Collaboration:** Foster collaboration between emergency medicine, radiology, infectious disease, and spinal surgery specialists. Early consultation with these specialists can facilitate prompt diagnosis and treatment.[9]
- **Case Discussions:** Regular multidisciplinary case discussions and reviews can help identify missed opportunities for early diagnosis and improve future practice.[9]

6. PROMPT TREATMENT AND FOLLOW-UP

- **Early Intervention:** Initiate appropriate treatment, including antibiotics and surgical intervention as soon as SEA is suspected. Prompt treatment can significantly improve outcomes.[11]

References available online.



- **Follow-Up:** Ensure close follow-up for patients at risk of SEA, especially those with recent ED visits for back pain or those with ongoing symptoms.[10]

The STRINGS mnemonic can be utilized during the physical exam to fully gather all the necessary information.

By implementing these strategies, clinicians can improve the early diagnosis and management of SEA, ultimately leading to better patient outcomes.

TREATMENT APPROACHES

Discuss the multidisciplinary approach to treating SEA, involving both surgical and pharmacological interventions. Statistics on time to diagnosis versus clinical outcomes.

CONCLUSION

- Early detection is crucial for favorable outcomes in SEA cases.
- Maintain a heightened awareness of the classic triad and associated risk factors.
- Remember that diabetes is a major risk factor, even more so than IVDA.
- A thorough differential diagnosis, including consideration of SEA, is essential in back pain cases.
- Collaborative decision-making between emergency medicine and other specialties is vital for optimal patient outcomes.

Getting Down to Business:
A Residency-Based Business Curriculum for Emergency Physicians

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The health care delivery system is a complex industry in which physicians simultaneously act as clinicians, participants, and managers.[1] As such, it is becoming increasingly critical that physicians understand basic business principles to successfully advocate for patients and create impactful changes in health systems. Highlighting the importance of business education during physicians' training, many of the Accreditation Council for Graduate Medical Education (ACGME) core competencies for resident physicians involve management and health systems knowledge.[2] However, a recent national needs assessment of residents demonstrated that many training programs provide minimal education in these competencies.[3] Consequently, trainees report having weak exposure to critical topics like negotiations and billing, and attending physicians corroborate a lack of training in critical business principles throughout all stages of their careers.[4]

CRITICAL LEARNING GAP

To help address this critical learning gap that increasingly affects physicians' abilities to advocate for patients and create impactful change, our team used Kern's six-step approach to curriculum development to build a pilot business curriculum for resident physicians.[5] Our goals in this process were to 1) educate learners in basic business principles as they relate to the practice of medicine; 2) assess learning through a series of surveys and interviews; and 3) develop a sustainable curriculum model that can be adopted across organizations.

Given the increasing importance and overall lack of current avenues for education in critical business principles of medicine, we are sharing our business curriculum development process to help others adapt similar programs. Here, we describe the creation, implementation, and assessment of this pilot business curriculum at a 4-year Emergency Medicine (EM) training program in the United States, while highlighting its potential for broad applicability across organizations and specialties.

Session	Topic	Time Allotted & Structure
1a	Personal Finance	100 minutes Lecture
1b	Models of EM Practice	90 minutes Lecture & Alumni Panel
1c	Negotiations	120 minutes Lecture & Mock Case Breakout Groups
1d	Billing & Coding	90 minutes Lecture
2a	Malpractice	270 minutes Lecture & Small Group Breakouts
2b	Operations	60 minutes Lecture & Operations Tech Demo
2c	Emotional Intelligence (EQ)	90 minutes Emotional Intelligence Quiz & Lecture
2d	Change Leadership	90 minutes Lecture & Panel

Figure 1. Eight-session business curriculum topics and time dedicated

CURRICULUM DEVELOPMENT & DESIGN

To construct the foundation of the curriculum, our team first conducted an extensive literature review of existing business curriculum proposals in medicine.[6-17] Next, our team of business-trained physicians worked closely with education experts at our institution to combine the topics and lessons from this literature review with data from a 2019 national needs assessment of business training in EM.[18] Through a consensus-driven process, we honed in on eight business topic areas that are critical for modern physicians: personal finance, models of practice, negotiations, billing and coding, emotional intelligence (EQ), operations, malpractice, and change leadership.

References available online.

Ultimately, we developed these topic areas into an eight-session curriculum that we implemented over 18 months (August 2020–February 2022) at a 4-year EM training program in the United States. The sessions were included as part of the residency's weekly didactics and varied in length from 60-90 minutes for lectures or panel-based discussions to 240 minutes for an immersive malpractice experience (Figure 1).

Due to the ongoing COVID-19 pandemic, all the sessions were held virtually. We utilized our institution's network of faculty and alumni, as well as national experts, to serve as presenters for each session. We provided each speaker with objectives—developed through iterative discussions by our team in conjunction with education expert consultation and focused on the upper tiers of Bloom's taxonomy—to ensure all critical aspects of each topic were covered.[19] Prior to each session, one of our team members met with the speaker(s) to review content and ensure alignment with the objectives. We recorded most of the sessions to enable asynchronous review, in addition to developing an open-access reference document with high-yield notes from each session.

To assess impact and further refine the curriculum, we conducted a series of IRB-approved surveys and interviews with learners. All survey and interview content was developed through iterative discussions among our team in conjunction with institutional education experts. Surveys were administered electronically via Qualtrics; they were made available by email link and a QR code during individual education sessions. The curriculum was bookended with identical pre-intervention and post-intervention surveys to assess learners' comfort levels with each of the curriculum topics via a 4-point Likert scale. We also conducted pre- and post-session surveys for each of the eight sessions; these surveys combined 4-point Likert-based comfort level questions with several multiple-choice knowledge assessment questions that were developed based on session objectives with education expert review. Respondents who completed both the pre- and post-session surveys for at least one session were matched and paired, and those responses were then used for significance testing; non-paired responses were excluded. Wilcoxon Signed-Rank was used to compare pre- and post-session Likert comfort level questions, and McNemar's test was used to evaluate the non-parametric pre- and post-session binary knowledge assessment variables.

In addition to surveys, we also conducted focused interviews to gain deeper insight into the curriculum's impact on learners. One of the authors interviewed five trainees, selected via convenience sampling among session attendees, over a 2-month period following a semi-structured protocol. Interview questions were created through iterative discussions among the team and examined participants' prior experiences before exploring how the curriculum impacted them, including which sessions were particularly helpful and less impactful, and why. Themes and direct quotations were recorded in real time. Through iterative rounds of discussions, our team derived themes categorizing learners' experiences with the curriculum. Exemplar surveys and the interview guide are available in Figures 2-4 in the supplement.

I can skillfully manage my personal finances, including budgeting, saving, and insurance.
Strongly Disagree 1 2 3 4 Strongly Agree

I can articulate different EM models of practice and compensation structures.
Strongly Disagree 1 2 3 4 Strongly Agree

I can navigate negotiations processes, especially as they relate to employment contracts.
Strongly Disagree 1 2 3 4 Strongly Agree

I can explain the intricacies of EMR documentation, levels of service and coding.
Strongly Disagree 1 2 3 4 Strongly Agree

I can prepare for a malpractice lawsuit.
Strongly Disagree 1 2 3 4 Strongly Agree

I can describe the importance of operations management for efficient ED function.
Strongly Disagree 1 2 3 4 Strongly Agree

I can employ strategies to empower myself and others as a leader in the ED.
Strongly Disagree 1 2 3 4 Strongly Agree

I can utilize change leadership strategies to help improve my workplace.
Strongly Disagree 1 2 3 4 Strongly Agree

The business aspects of my life and career cause me stress.
Strongly Disagree 1 2 3 4 Strongly Agree

Figure 2. Exemplar survey 1 used to assess the business curriculum

1. **Confidence Assessment** *(Please answer the following question by circling a number on the scale (1 – 4) to indicate your response for the following area.)*
   - **I can skillfully manage my personal finances, including budgeting, saving, and insurance.**
     Strongly Disagree 1 2 3 4 Strongly Agree
2. **Knowledge Assessment** *(Please answer the following questions by circling the most correct answer.)*
   - **Which of the following factors influence one's credit score?**
     A. Income & payment history
     B. Age & credit history length
     C. Debit card usage & income
     D. Payment history & credit history length
   - **What is the "Rule of 72" as it relates to personal finance?**
     a. It helps estimate the number of years it will take for an investment to double.
     b. It relates retirement age to necessary retirement savings.
     c. It helps demonstrate the inverse relationship between spending and savings.
     d. There is no "Rule of 72" in personal finance.
   - **When are the funds in a Roth IRA account subject to tax?**
     a. Upon withdrawal of money from the account during retirement.
     b. Prior to contributing money to the account.
     c. Both prior to contributing money to the account and upon withdrawal of money from the account.
     d. Never subject to tax, either before or after contribution to the account.
   - **A "Rider to Increase", in the context of disability insurance:**
     a. Offers one the ability to increase coverage at set periods to keep pace with rising income.
     b. Requires one to increase coverage at set periods of time.
     c. Automatically increases one's coverage to keep pace with inflation.
     d. Allows one to switch insurance companies without penalty.

Figure 3. The second exemplar survey used to assess the business curriculum

- Please tell me a bit about your background, including any prior careers, and what you hope to do after residency.
- How, if at all, has the Business of EM curriculum impacted you?
- What behavior changes in your personal or professional life, if any, have you made after attending the Business of EM sessions?
  - If needed, consider prompting: personal savings/retirement, documentation, career plans
- Which sessions of the Business of EM curriculum have most influenced your behavior and why?
  - If needed, list topics: Personal Finance (Sept 2020), EM Models of Practice & Compensation (Oct 2020), Negotiation & Contracts (Jan 2021), Documentation & Billing (May 2021), Interpersonal Communication (Aug 2021), Operations Management (Sept 2021), Malpractice (Nov 2021), Change Leadership (Feb 2022)
- What about the structure or content of the Business of EM curriculum particularly worked well for you?
- What could be improved with the structure or content of the Business of EM curriculum?
- What additional topic(s) would you like to see covered in the Business of EM curriculum? Are there any additional structures or learning opportunities you would like to see?

Figure 4. An interview guide assessing business curriculum

References available online.

PILOT CURRICULUM IMPACT

The pilot curriculum was offered to 58 EM residents, and nearly every resident participated in some portion of the curriculum over its 18-month implementation; an average of 35 learners were present at each individual session. Twenty-seven learners completed the pre-curriculum survey (response rate=46.7%), and 14 completed the post-curriculum survey (response rate=24.1%). 12.5% of learners reported some level of prior business training, such as a master's degree. In the pre-curriculum survey, 26% agreed or strongly agreed that they felt comfortable with the curriculum topics; this rose to an average of 73% in the post-curriculum survey.

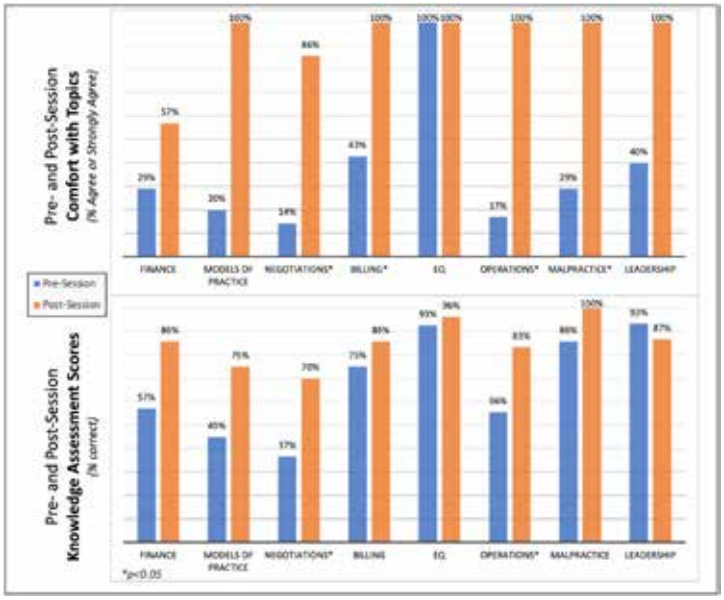

Fig. 5. Resident confidence in business acumen, pre- and post-curriculum

Across the 8 sessions, 10-24 learners completed the pre-session survey, and 6-19 learners completed the post-session surveys. An average of 7 learners per session submitted both the pre- and post-session surveys. Seven of the eight sessions demonstrated an increase in comfort levels and knowledge scores (Figure 5). On average, 37% of participants agreed or strongly agreed that they were comfortable with topic areas before each session; this increased to an average of 93% after the sessions. Knowledge scores rose from an average of 68% correct pre-session to an average of 85% correct post-session. Four sessions showed statistically significant rises in comfort levels: Negotiations, Billing and Coding, Operations, and Malpractice ($p<0.05$). Half of the sessions demonstrated rises in knowledge scores by over 25%, and the Negotiations and Operations sessions showed statistically significant increases in knowledge scores ($p<0.05$).

Interviews revealed that, in addition to presenting foundational concepts, the curriculum created tangible impacts on learners' lives, with participants commenting that they "have started setting aside an emergency savings fund and saving more for retirement" and "feel more comfortable having a framework for how to think about potential job options in the future."

Ultimately, our team successfully created both a process and a pilot program for resident physicians that fills a critical education gap. By combining existing proposals in the literature with needs assessment data to identify critical topic areas, followed by developing session objectives, content, and assessments in collaboration with business and education experts, we developed a curriculum that increased learners' comfort with and knowledge of most session topics while receiving overwhelmingly positive reviews in interviews.

As part of a continuous improvement process, our team is incorporating survey results with feedback from interviews to improve future iterations of the course. Planned improvements include additional focus on specific takeaways and direct applications to life in medicine and beyond for each session.

LIMITATIONS

Although our pilot curriculum has broad potential applicability across institutions and specialties, both our curriculum and this project have limitations. First, the curriculum was implemented at a single EM residency training program, with a relatively small number of participants and variable response rates to individual surveys. Second, the surveys included in the study relied heavily on self-assessment of comfort, when physicians have a documented limited ability to self-assess.[20] Finally, this work does not explore potential long-term applications of the demonstrated knowledge gains.

CONCLUSION

Basic business knowledge is a critical skill for every physician working in today's complex health system environment, and our pilot curriculum suggests that this knowledge can be achieved in part through a longitudinal business curriculum during residency training. The curriculum we developed is effective at increasing resident physicians' comfort with and knowledge of critical business aspects related to the practice of medicine. Notably, since the original development of this pilot curriculum in 2020, several authors have introduced targeted business education as well; the recent emergence of numerous educational programs in this realm reaffirms the importance of this topic.[12-15] Building on our process, other organizations may be able to implement or enhance similarly impactful educational programs. Future work is needed to adapt and refine the curriculum, expand across institutions and specialties, and explore long-term applications.

References available online.

INTEGRATED WEALTHCARE

Perspective Matters

"As emergency physicians, you live the reality that health is unpredictable. Take steps during training to protect your financial health over the life of your career."

M. Shayne Ruffing
Integrated WealthCare

After almost 30 years of immersion in the financial side of medicine, there are a couple of relevant truths that should be appreciated, as you move through training and look ahead to attendinghood.

SPECIFICALLY:

Cash is still king — It is tempting to use all available cashflow to reduce debt, or invest for growth, but both of these take a back seat to having adequate savings. Having a personal reserve provides financial and psychological security. Build a reserve of cash in the bank. This can be a savings account, CD, money market or any other cash equivalent.

Protecting your income is best done early – As emergency physicians, you live the reality that health is unpredictable. Adequate disability insurance is critical for early practice physicians. It insures and ensures that the significant investment that you have made in your career pays you what you have invested in, regardless of your ability to continue in the ED. There are currently five companies with an extremely competitive disability contract for an emergency physician. The differentiator is in the pricing and sometimes in the way you qualify. Not one of the five companies is meaningfully different or better than the other, despite what they will each tell you. Put them all side by side for the most informed decision.

Taxes will likely always be higher in the future – As a resident, you are in the lowest income tax environment you will (hopefully) ever be in again. Take advantage of it by putting as much money into a Roth IRA as you can, every year. While it may seem counterintuitive, today is the time for you to pay taxes, particularly when you can put after-tax money into a future tax-free account. With a Roth IRA, you pay taxes on the contribution in a +/- 16% tax world and take the money out tax free when it would be a +/- 30% tax environment. Invest the IRA for aggressive growth and watch your financial security grow every day.

Fund your 403(b), even if you don't get a match – Statutory employees (house staff) generally are not able to get GME money into a retirement plan. But that doesn't and shouldn't prevent you from contributing as much as possible to these accounts, after building up savings and funding your Roth IRA. These accounts are tax deductible today, reducing the tax cost on your Roth contribution, and will grow tax deferred over time. A quick example: If you only contribute $10,000 per year to your 403(B) and it appreciates at a net 6.5% annually, the value in 30 years will be $929,229. That's a well-funded retirement party. :)

Invest appropriately – If you don't know how to do this, pay someone for advice. A fiduciary investment

References available online.

advisor can structure your accounts, cashflow, and investment holdings for maximum tax efficiency, growth and risk management.

Have a loan repayment plan – The future of the PSLF program continues to be uncertain. There will likely be some level of phaseouts, revenue ceilings, or other factors that reduce the benefits of this program. Take the time to understand or to retain student loan counselors to develop and maintain a prudent debt strategy, but not at the expense of the previous considerations. The debt got you here, but it doesn't have to keep you here. Create a plan that accomplishes all of your goals and set a reasonable review schedule to stay on track. Generally, twice per year is sufficient.

At the end of the day, your training has prepared you to be a good doctor and earn a good living. Your goals can be accomplished, with some forethought and intentionality. If you are comfortable doing all of this yourself and managing your practice and raising your family, that's great; you should do that. In my lengthening experience, however, it is a small percentage of physicians who can and want to do this themselves.

If you are in the majority, find and establish a relationship with a multi-faceted firm who can help you understand the relevant considerations, develop and quarterback an action plan, and manage the results over time. The relatively nominal expense that you will pay as a percentage of your income should be well worth the confidence and success of achieving your goals.

Thank you for your service to the greater good.

Shayne

M. Shayne Ruffing is the founder and CEO of integrated WealthCare (IWC), an SEC Registered Investment Advisory. Headquartered in Durham, North Carolina, IWC provides comprehensive financial and wealth management to physicians and medical practices across the United States. IWC has two primary advisory offices: One in Durham, NC, one in Kona, HI, and a soon to be presence in the greater Orlando, FL, area.

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References available online.

POCUS for the Win: Ultrasound-Guided Pericardiocentesis for Cardiac Tamponade

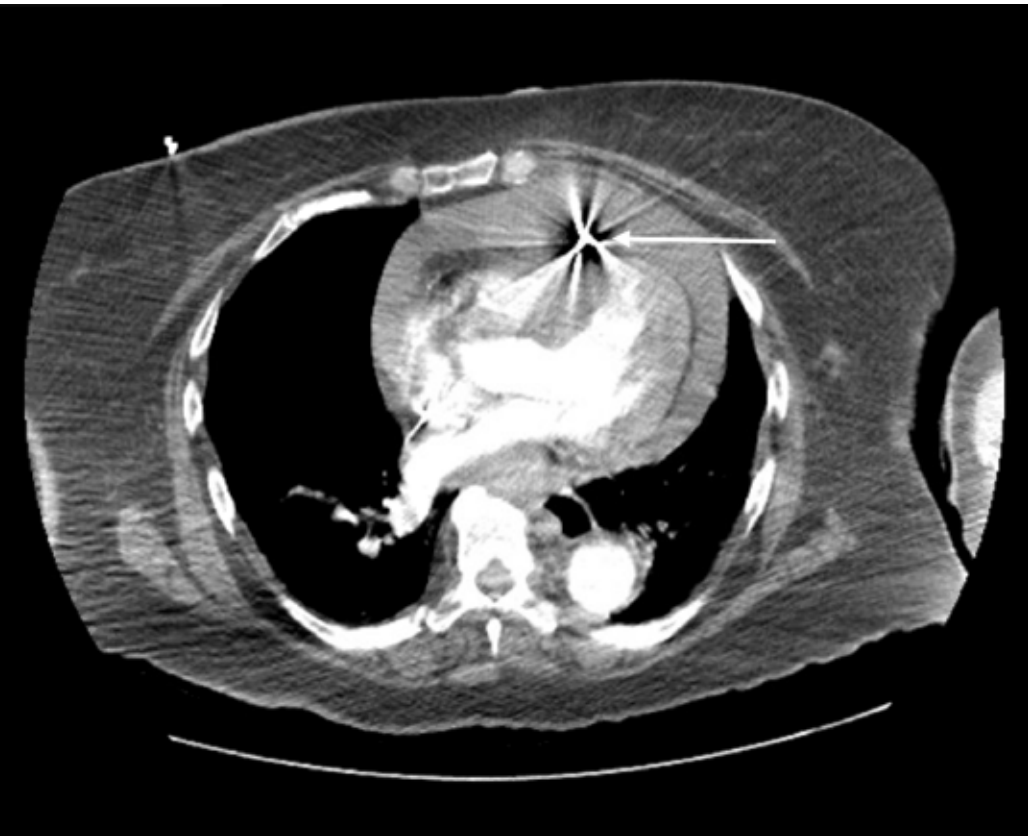

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Figure 1. CTA chest axial with pacemaker lead perforation (white arrow) through the anterior right ventricle wall and hemopericardium.

HISTORY OF PRESENT ILLNESS

A 66-year-old female, status post single-lead implantable cardioverter defibrillator 3 weeks ago, with history of hypertension, hyperlipidemia, and heart failure with reduced ejection fraction (25%) secondary to non-ischemic cardiomyopathy presents to the ED by ambulance with severe epigastric pain, radiating to her back that started 2 hours prior to arrival.

PHYSICAL EXAMINATION

Vitals are: temperature 36.4°C, heart rate 97 bpm, blood pressure 44/23 mmHg, respirations 28/min, oxygen saturation 95% on room air. Examination reveals a lady who is pale, diaphoretic, and anxious-appearing, with a GCS of 14, losing 1 point for verbal. Cardiac and respiratory examination is only significant for diminished lung sounds. She has no peritoneal signs, no focal abdominal tenderness/masses, and gross rectal examination is without blood. The patient is given 1 liter of lactated ringers and 2 units of uncrossmatched blood, resulting in an increase in mean arterial pressure to >65 mmHg and improvement in her mental status. Broad-spectrum antibiotics were also started.

LABS AND IMAGING

The patient's bedside glucose was 212 mg/dL. Hemoglobin prior to transfusion was 10 g/dL, lactate was 4.2 mg/dL with a mild anion gap metabolic acidosis. Otherwise, labs such as the comprehensive metabolic panel, coagulation panel, high sensitivity troponin, and brain natriuretic peptide were unremarkable. A computed tomography angiography (CTA) of her aorta through the chest/abdomen/pelvis (Figure 1) was emergently obtained, which revealed a pacemaker lead perforation through the right ventricle with resulting hemopericardium. The patient's blood pressure continued to decline, prompting 2 more units of uncrossmatched blood, norepinephrine, and vasopressin to maintain MAP goals.

POCUS

While emergent cardiac surgery consultation was being obtained, the ED team performed a parasternal-approach, ultrasound-guided ED pericardiocentesis (Figure 2) under procedural sedation

References available online.

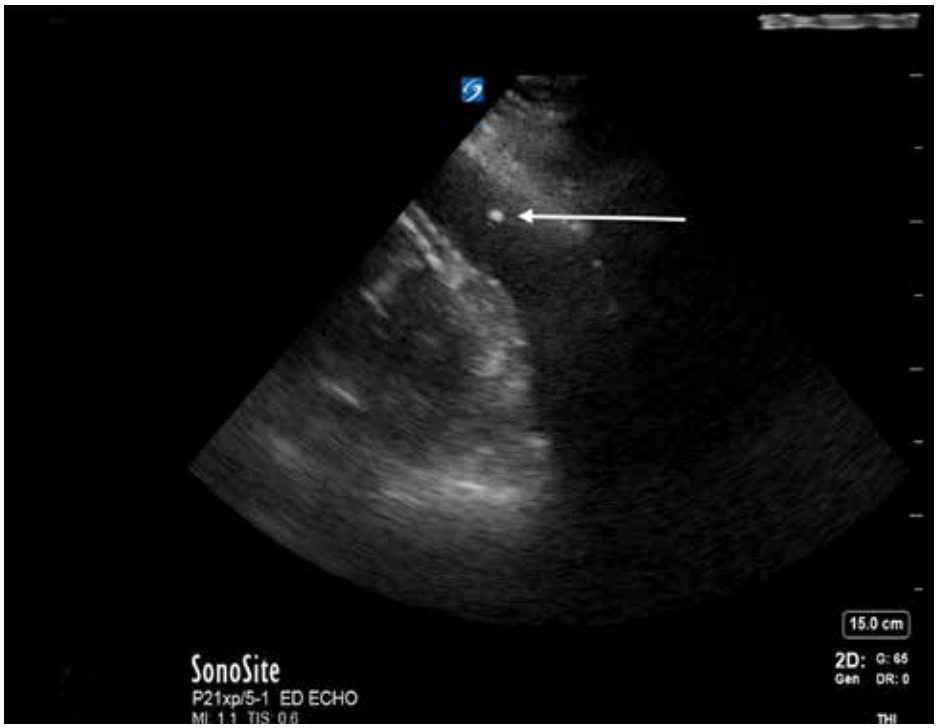


Figure 2. Bedside echocardiogram with spinal needle tip (white arrow) visualized in the hemopericardium during therapeutic pericardiocentesis.

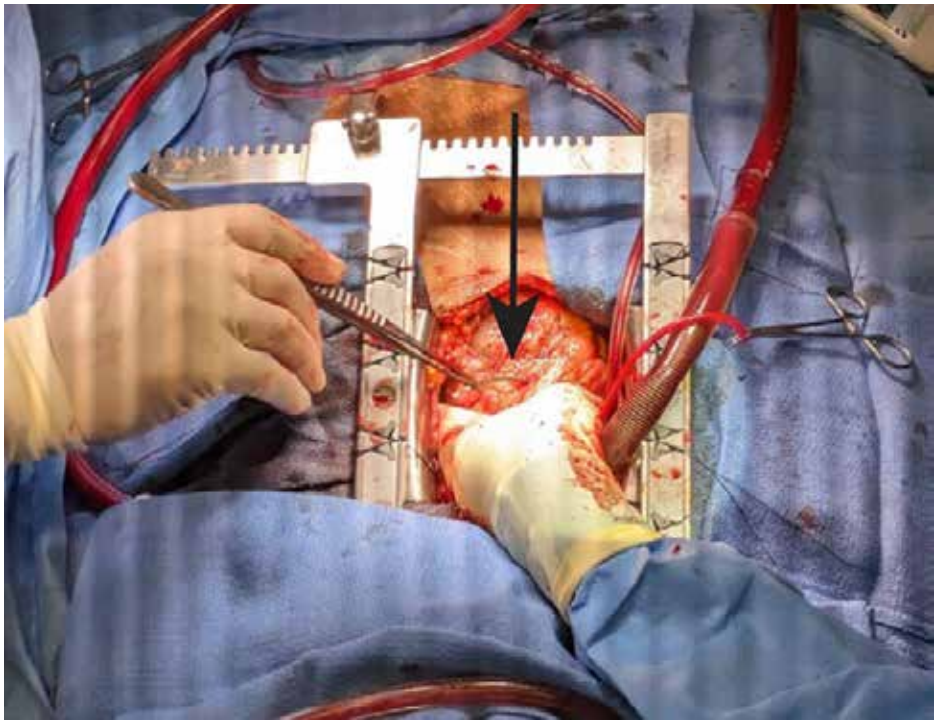


Figure 3. Intraoperative photograph of perforating pacemaker lead, grasped by forceps (arrow)

with ketamine. A subxiphoid approach for pericardiocentesis was deferred as the patient had a large liver cyst obstructing the path and the largest fluid pocket was visualized in the parasternal long view.

The removal of 90 mL of sanguinous fluid immediately stabilized the patient's vital signs, improved mental status, and ultimately negated the need for vasopressors.

DISCUSSION

The patient had cardiac tamponade secondary to hemopericardium from pacemaker and defibrillator lead right ventricular free wall perforation. Lead perforation is considered a rare complication with a majority occurring within the first year of implantation. The incidence rate is estimated to be approximately 0.1%-1%.[1,2] Lead perforation of the myocardium can lead to life-threatening complications such as cardiac tamponade.

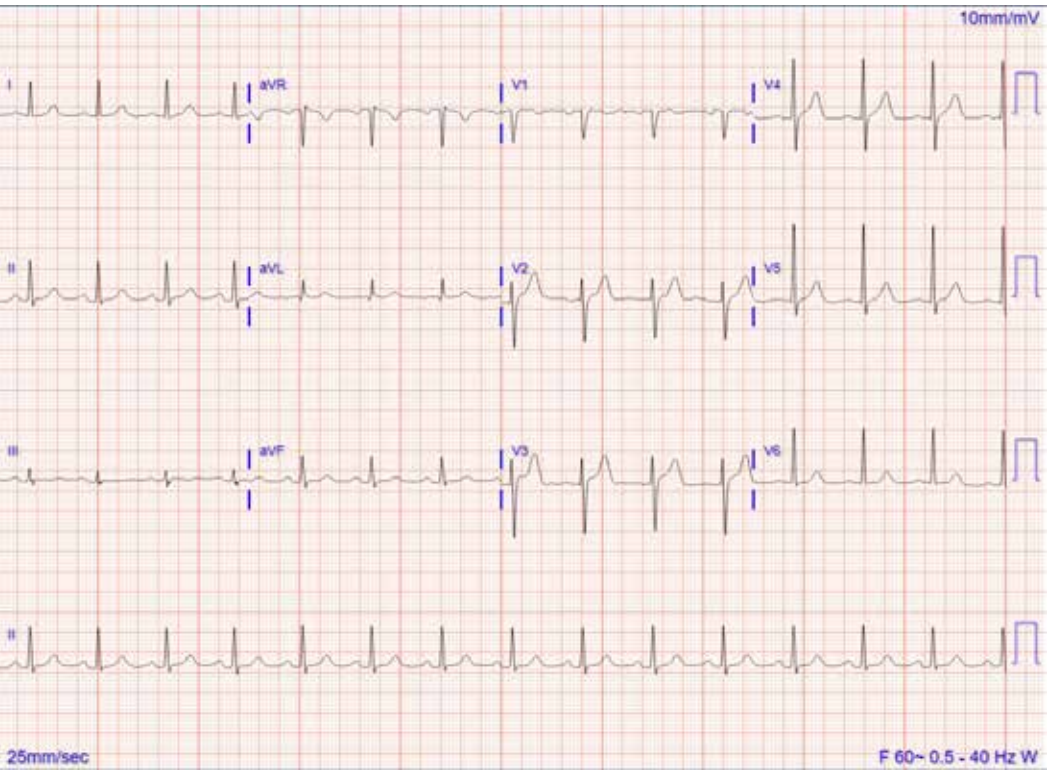
POCUS is a key diagnostic tool that can allow emergency physicians to diagnose cardiac tamponade with the presence of a pericardial effusion with right ventricular collapse during diastole and/or right atrial collapse with systole. There can be other findings on echocardiogram to include a plethoric inferior vena cava (>20 mm), which can be up to 92% sensitive, but not necessarily specific.[4] Also, septal "bounce," described as intraventricular septal deviation towards the left ventricle during inspiration can be another finding of tamponade physiology. In an emergent setting with hemodynamic instability, an ED pericardiocentesis can be performed to allow ventricular filling. With ultrasound-guidance, major complication rates of pericardiocentesis can be lowered from 25% to 3%.[4-6]

HOSPITAL COURSE

The cardiac surgery team performed a sternotomy and pericardiotomy, revealing a lead perforation of the right ventricle and an innominate vein injury (Figure 3). The lead was removed and repair of the ventricle and vein performed. The hospital course was prolonged with the complication of a sternal wound infection, but the patient improved on intravenous antibiotics and was discharged home with a wearable defibrillator. She attended her outpatient follow-up appointments with cardiac surgery and cardiology without complications.

References available online.

Nitroglycerin's Hidden Trigger: The Bezold-Jarisch Reflex Revealed



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Quick recognition of the Bezold-Jarisch reflex triad of hypotension, bradycardia, and apnea is important for any physician who administers nitroglycerin. This knowledge can ensure positive patient outcomes and avoid cardiopulmonary collapse.

CASE PRESENTATION

A 59-year-old male presented to the emergency department through the lobby with general malaise, dizziness, and hypertension for 6 days prior. In triage, the patient endorsed acute subtle chest pressure with no associated pain or radiation. Patient denied shortness of breath, nausea, abdominal pain, jaw pain, arm pain, or headache. No pertinent family or past medical history was reported.

An initial EKG showed normal sinus rhythm at 87 bpm with no ST-segment or T-wave abnormalities. Initial vital signs were notable for a BP of 208/114 with no other abnormalities. Cardiac exam showed normal rate and rhythm, with no murmur heard. No known drug allergies were confirmed with the patient.

The initial treatment modalities for hypertension and chest pain included 0.4 mg sublingual nitroglycerin and 324 mg chewable aspirin. Five minutes after medication administration, the patient became unresponsive and diaphoretic, with pulse in the 20s and a blood pressure of 50/30s. The patient became unresponsive to painful stimulus, with no palpable carotid pulse, and CPR was initiated. The patient immediately woke up with initiation of CPR but was lethargic, with a weak carotid pulse in 30s. Further treatment with 1mg atropine IV and a normal saline bolus was started. Shortly after, the patient's pulse improved to 80s and the blood pressure improved to 80s/60s. The patient fully returned to baseline mental status 5 minutes after atropine administration, with no pertinent findings on repeat exam.

Labs showed hyponatremia at 126 mmol/L, negative BNP, and high-sensitivity troponin of 22ng/L with repeat delta troponin at 20 ng/L. The patient was admitted for continued monitoring. Echocardiogram showed normal systolic function with normal LVEF 65%, no wall motion abnormalities, and no valvular abnormalities. No further cardiac events

References available online.

were observed, and serial troponin markers remained negative. A work-up for acute coronary syndrome (ACS) was negative.

Cardiology was consulted and noted that the initial reaction was likely Bezold-Jarisch reflex. The patient's presenting symptoms were attributed to poorly controlled hypertension. His medications were adjusted, and he was ultimately discharged in stable condition.

DISCUSSION

The case underscores the clinical significance of the Bezold-Jarisch reflex (BJR). Pertinent chart reviews and case studies revealed few clinically relevant cases involving nitroglycerin induced BJR. In prior cases where Bezold-Jarisch reflex was observed, those patients typically had underlying cardiac pathology.[2,3] The BJR has rarely been documented in patients who have a non-cardiac etiology of their symptoms or have no prior cardiac history.

This case demonstrates that Bezold-Jarisch reflex can be seen in patients without underlying cardiac pathology. Given the prevalence of nitroglycerin administration in the ED, it is important for physicians to recognize this as a medication side effect. Quick recognition of the Bezold-Jarisch reflex triad of hypotension, bradycardia, and apnea is important post administration of nitroglycerin to ensure positive patient outcomes and avoid cardiopulmonary collapse.

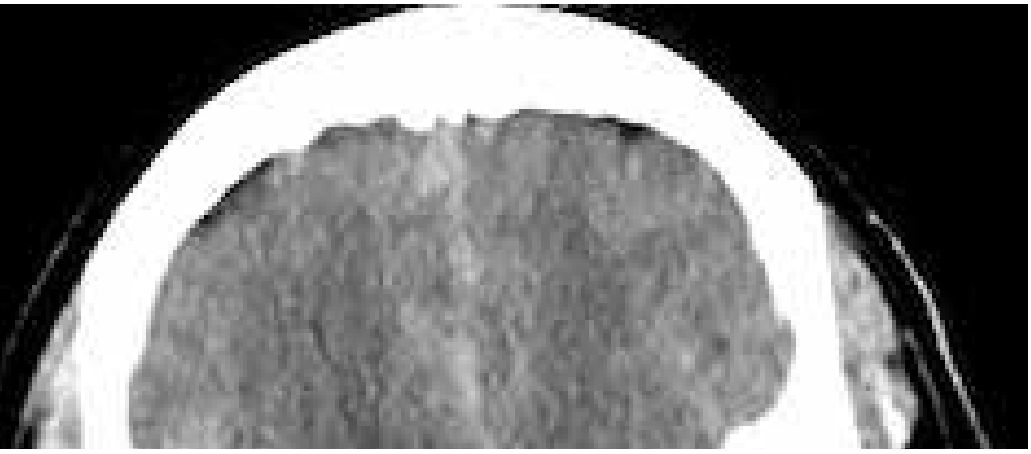
In this case, the patient's bradycardia was effectively treated with atropine and their hypotension was managed with fluid resuscitation. While research on this reflex is minimal due to the isolated nature of this reaction, it warrants consideration in patients showing unexplained apnea, hypotension, and bradycardia. The management above, atropine and IV fluid resuscitation, may be considered if this reaction is witnessed.

It remains crucially important for physicians to maintain vigilance for a rare but life-threatening reaction that can occur from a frequently utilized medication.

References available online.

NICE to Know Series: Aneurysmal SAH Care in the ED and Beyond

(Neurological Interventional Care in Emergencies)



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Aneurysmal subarachnoid hemorrhage (aSAH) is a must-not-miss diagnosis in the emergency department (ED). An aneurysm is a localized dilatation or ballooning of a blood vessel wall. Risk factors include hypertension, age >50, smoking, and genetics (i.e., polycystic kidney disease, family history).[1] The rupture of an intracranial aneurysm leads to bleeding within the subarachnoid space, where cerebrospinal fluid circulates, which leads to various pathophysiological consequences, such as hydrocephalus, inflammation, vasospasm, and ischemic injury.

The classic presentation for patients suffering from an aSAH is a thunderclap headache or a sudden, severe headache that reaches its maximum intensity within minutes. Some patients may have had a milder sentinel headache that occurred days or weeks earlier. Other symptoms may include neck pain or stiffness, photosensitivity, nausea/vomiting, loss of consciousness, and neurological deficits.[2] This description typically prompts the evaluation for subarachnoid bleeding; however, up to 12% of presentations are still missed.[3,4] Kowalski (2004) analyzed 56/482 SAH patients that were missed in the ED and looked at the predictors associated with missed diagnosis. One of the interesting associations was a longer interval between the onset of symptoms and presentation in missed cases.[5] This points to a bias among providers who might assume that patients with aSAH would present on the day of aneurysm rupture.

SAH is a devastating condition with high morbidity and mortality; roughly two-thirds of untreated SAH patients die or have serious neurologic disabilities consequently.[6] In the ED, clinicians are tasked with promptly identifying an aSAH and implementing treatments that are geared towards the prevention of aneurysm rebleeding and the deleterious effects of acute hydrocephalus. Here we will dive into how the ED clinician can effectively diagnose aSAH, treatments, and what comes next once the patient leaves the ED.

CASE PRESENTATION

A 61-year-old female with a history of hypertension presented to the emergency department after a syncopal episode. She was shopping at the mall when she suddenly collapsed. There were no reports of head trauma. Upon arrival at the ED, she was very lethargic and slow to answer questions. She reported having an intense headache that came on suddenly prior to her collapse. She is a current smoker. She is not taking any anti-platelet or anti-coagulant medications. She was maintaining her airway and aside from her drowsiness, her neurological examination was non-focal.

OK, I AM GETTING A CT SCAN—WILL IT PROVIDE ME WITH THE ANSWERS TO MY BIGGEST QUESTION?

A non-contrast head computed tomography of the head (NCHCT) is the initial test for evaluating any intracranial hemorrhage **(Image 1)**. The sensitivity of detecting subarachnoid blood is 100% within 6 hours of symptom onset and up to 90% within 24 hours.[7] Once identified, it is important to take note of a few key radiographic features: the presence of hydrocephalus, distribution of blood, intraparenchymal involvement, and the presence of global cerebral edema.[7]

References available online.

Hydrocephalus arises from obstruction of the brain's normal cerebrospinal fluid (CSF) flow dynamics. An increase in CSF due to obstruction will lead to an increase in intracranial pressure (ICP) and patients can quickly decline. Temporal horn enlargement, trans-ependymal edema, and bowing of the third ventricle are CT findings suggestive of obstructive hydrocephalus.[8] These findings in conjunction with the patient's mentation and neurologic examination are an indication of an ICP crisis and necessitate immediate intervention.

Mild hydrocephalus can occasionally be tolerated if the patient remains easily arousable, although very close neuro monitoring must be maintained in an ICU level setting due to the risk for rapid worsening of hydrocephalus. The treatment for acute hydrocephalus in the setting of aSAH is emergent ventricular drain (EVD) placement. This procedure is performed by a trained neurosurgeon but may occasionally be done in the emergency department if necessary. Intubation is not absolutely necessary prior to EVD placement, but should be performed at the discretion of the ED attending.

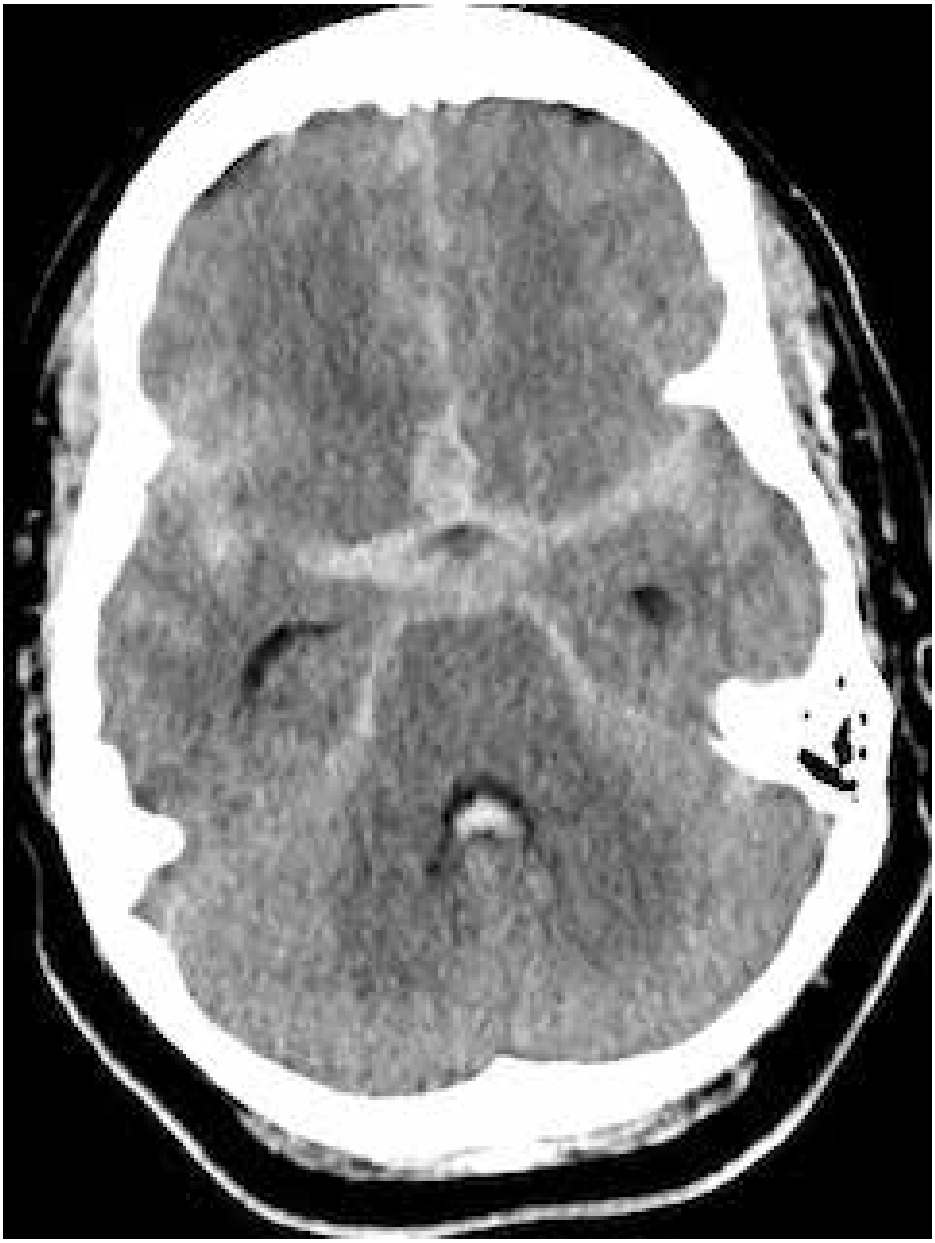

Image 1

The distribution of blood on a NCHCT may help discern the underlying etiology or location of the patient's vascular insult. However, this will need to be further assessed with a CT angiography (CTA) of the brain. CTA has a 98% sensitivity and specificity for the detection of aneurysms >3mm in size.[1] CTA is helpful for our neurosurgical and neuro-interventional colleagues in planning an open craniotomy for aneurysmal clipping or endovascular coiling, respectively. The presence of associated intraparenchymal hemorrhage or diffuse cerebral edema indicates a higher severity of brain injury and carries a high rate of morbidity and mortality.[1]

TO TAP OR NOT TAP...THAT IS THE QUESTION!

Performing a lumbar puncture (LP) to exclude SAH when there is a negative NCHCT remains the standard of care. A hemorrhage on NCHCT may begin to appear less hyperdense, making a subarachnoid bleed difficult to diagnose. A LP is used to evaluate for the presence of red blood cells and xanthochromia, or the yellowish discoloration in the CSF. Xanthochromia is a result of red blood cell (RBC) breakdown, develops within 6-12 hours after aSAH, and has a 93% sensitivity and 95% specificity when present for aSAH.[9,10]

Despite advanced imaging techniques (i.e., CTA, MRI/MRA, diagnostic angiography) becoming more readily available, the presence of an aneurysm on CTA after a negative NCHCT does not confirm rupture. LP can yield false-negative results, partially if performed too early after symptom onset or if the blood is localized in a small area of the subarachnoid space.[11] Mark (2015) reported 49 of 58 patients undergoing LP had either visible xanthochromia or a CSF RBC count greater than $2000x10^6$/L which safely identified an aneurysmal cause of subarachnoid hemorrhage.[12] Both LP and CTA are great diagnostic tools when evaluating aSAH; however, LP remains the best test in diagnosing a ruptured aneurysm because the treatment for an unruptured aneurysm is different.

References available online.

HOLY CRAP...THAT IS A BIG BLEED. WHAT COMES NEXT?

So, you have cracked the case! You have identified the patient has an aSAH. Now what? As you begin to mobilize the cavalry (neurosurgery, neurology, neuro-ICU, neuro-interventional radiology) the ED provider must remember these key management considerations:

ABCS ALWAYS COME FIRST...

Blood pressure control

Blood pressure control is the single most important thing the ED physician can do in the setting of a ruptured aneurysm. Failure to adequately control the BP may lead to aneurysm re-rupture, which drastically worsens both neurological and mortality statistics. BP elevation in the setting of a ruptured aSAH is often due to pain and sympathetic activation.[13] Analgesics may be effective in blood pressure control; however, anti-hypertensive medications may be necessary. The treatment of blood pressure is a balancing act. It is important to maintain adequate cerebral perfusion pressure (CPP) by preventing hypotension and lower the risk of aneurysm re-rupture by preventing hypertension.

An elevated blood pressure after a ruptured cerebral aneurysm can be treated with a dose of labetalol; however, clinicians should be cautious of its bradycardic effects. The ideal agent of choice is a continuous titratable infusion with nicardipine or clevidipine with a systolic blood pressure goal of 120mmHg–140mmHg.[13] After the aneurysm is secured, blood pressure goals are liberalized (SBP <200mmHg) to reduce the likelihood of vasospasm and delayed cerebral ischemia.[7,13]

Anticoagulation reversal

Some patients may present with aSAH and concomitantly take antiplatelet or anticoagulation agents for various reasons (e.g., atrial fibrillation, coronary artery disease, cardiac stents). In the setting of most intracranial bleeding, more specifically aSAH, it is important to discontinue all anticoagulants and reverse them with the appropriate agents. Patients taking antiplatelets (i.e., aspirin, clopidogrel, ticagrelor) should be given desmopressin (DDAVP), and a transfusion of platelets should be considered.[14] For those taking the vitamin K antagonist, warfarin, and found to have an elevated international normalized ratio (INR), 4-Factor Prothrombin Complex Concentrate (4-PCC), or K-Centra, should be used as a reversal agent.[15] The newer direct oral anticoagulants (DOACs) apixaban and rivaroxaban should be reversed with Andexxa, or 4-PCC if the former is not available. If patients are taking dabigatran, the appropriate reversal agent would be Pradaxa.[15] There should be a discussion with your neurosurgical team surrounding the appropriate reversal agent.

Antiepileptics

Seizures may be most frequent at the onset of aSAH.[7] Several studies reported seizure activity in 6-26% of patients after aSAH.[16] Seizure prophylaxis should be initiated and continued at least until after the aneurysm has been secured. The agent of choice is levetiracetam, with a loading dose of 60 mg/kg upon arrival and a maintenance dose as patients transition to the ICU. Seizure activity in the setting of an unsecured aneurysm increases the risk of aneurysm re-rupture, carrying a high rate of mortality.[16] Historically, phenytoin has also been used for seizure prophylaxis, but recent data suggest that phenytoin is associated with worse neurologic and cognitive outcomes and should be avoided.[17]

External Ventricular Drains

An external ventricular drain (EVD), or ventriculostomy, is an invasive monitoring and therapeutic device that is inserted by a neurosurgeon. Placement of an EVD most frequently occurs in the ICU setting; however, there are cases where an increase in ICP and a decline in the patient's mental status because of acute obstructive hydrocephalus (AOH) necessitates emergent placement in the ED. This device is carefully placed into the third ventricle by way of Kocher's point (11cm posterior from the nasion and 3cm from the midline with a trajectory toward the ipsilateral medial canthus and a line extending coronally from the ipsilateral tragus).[18] An EVD is used to decrease ICP by diverting CSF from the intracranial compartment to an external drainage system. It is also used to quantify the ICP via a pressure transducer, which allows providers to swiftly treat any ICP elevations. This apparatus is closely managed by the neuro-intensivists and neurosurgical team.

During EVD placement, the ED attending has a critical role to play. BP management is paramount, as a spike in blood pressure during EVD placement may precipitate re-rupture of the aneurysm. For this reason, pain control, sedation, and BP management are essential, in addition to airway control if necessary. ED management of these parameters allows the neurosurgeon to focus on proper placement of the ventricular drain.

Re-rupture

Re-rupture of an aneurysm and rebleeding occurs in ~15% of patients and carries a high mortality rate (50%).[7] The risk of rebleeding increases over time in the setting of an unsecured aneurysm, but highest in the first 24 hours. Factors associated with rebleeding include noxious stimuli, agitation, rapid drainage of CSF during ventriculostomy placement, hypertension, and seizure activity.[19] Only aneurysm securement is effective in *definitively* preventing rebleeding and should be performed as soon as possible.

Delayed Cerebral Ischemia (DCI) & Vasospasm

Vasospasm is the leading cause of morbidity among patients who survive their initial SAH.[20] The volume of blood located in the subarachnoid space can irritate the large arteries that reside there. This irritation can lead to vasospasm and decreased blood flow through the vessel, causing ischemia to the downstream neuronal tissue. Nimodipine, a dihydropyridine calcium channel blocker, has a selective affinity for the calcium channels present in

References available online.

the smooth muscle cells of cerebral blood vessels. The vasodilation induced by nimodipine improves neurologic outcomes, likely through a neuroprotective mechanism. DCI may occur after 3-14 days with a peak around days 7-10.[13,19] Serial neurological examinations, transcranial Doppler, and diagnostic angiography are methods used for DCI surveillance.

Disposition and Beyond

Patients with aSAH will always require the highest level of care in an ICU or a dedicated neuro-ICU. There are situations where patients will go directly to the operating room or the interventional radiology suite for securement of the ruptured aneurysm prior to admission to the ICU. Endovascular coiling of aneurysms has gained favor over neurosurgical clipping in recent years due to its safety profile and superior functional outcomes.[2,21] To note, aneurysms associated with intraparenchymal hematomas, broad neck aneurysms not amendable to coiling, and patients with diffuse cerebral edema *may* benefit from *open* neurosurgical intervention.[2] Patients typically remain in the ICU for up to 21 days to closely monitor for DCI and are treated for other systemic sequelae of ruptured aSAH, such as cerebral salt wasting, stress cardiomyopathy, neurogenic pulmonary edema, and central fevers.

I AM AN EMERGENCY PHYSICIAN...WHAT DO I NEED TO KNOW?

- A good neuro-examination should be obtained upon arrival before any diagnostic or therapeutic interventions.
- A CT is 100% sensitive in detecting SAH within the first 6 hours, followed by a CTA if bleeding is present, to help identify the aneurysm and guide treatment planning.
- After 12 hours of headache symptom onset and a negative CT, a lumbar puncture remains a useful diagnostic tool in revealing aSAH.
- Until the aneurysm is secured, there is a high risk for re-rupture. To avoid rebleeding, the ED physician should:
  - Treat pain aggressively
  - Obtain tight blood pressure control (goal SBP 120 mmHg–140 mmHg)
  - Administer anti-seizure prophylaxis
  - Obtain immediate neurosurgical consultation for EVD placement in the setting of acute obstructive hydrocephalus
  - Reverse coagulopathy
- Oral nimodipine should be used to prevent aSAH sequelae such as vasospasm and associated delayed cerebral ischemia, but this is rarely a cause of patient deterioration in the emergency department.

CASE CONCLUSION

Our patient was quickly transferred to the ICU where she received an EVD and subsequently underwent coiling of an anterior communicating artery aneurysm. She remained in the ICU for 21 days where close monitoring of her ICP was performed with the EVD in place and delayed cerebral ischemia was surveilled with daily transcranial dopplers (TCDs) and avoided with the use of nimodipine. She went home on hospital day 24 and has made a tremendous recovery.

The Ice Pack Test: A Diagnosis of Myasthenia Gravis in the Emergency Department

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Myasthenia Gravis (MG) is an autoimmune neuromuscular disorder characterized by the formation of autoantibodies against postsynaptic acetylcholine receptors at the neuromuscular junction resulting in fluctuating weakness of the extraocular, bulbar, limb, or respiratory musculature. At cold temperatures, neurotransmission is believed to be improved due to inhibition of acetylcholinesterase.[1] Patients presenting with ocular features of MG including ptosis and diplopia often report temporary relief after application of an ice pack over the eyes, and clinicians may assess for improvement of ocular symptoms after cooling as part of the initial workup of suspected MG. We present a case in which the clinical diagnosis of MG was successfully made in the emergency department by the ice pack test to draw attention to the utility of this inexpensive, noninvasive, and highly specific diagnostic test in emergency department and primary care settings.

INTRODUCTION

Myasthenia Gravis is the most common disorder of neuromuscular transmission.[2] The prevalence of MG in the United States is estimated to be 37 per 100,000[3] with estimated incidence ranging from 4.1 to 30 cases per million person-years.[4] The disease most commonly presents in females under the age of 40 and men over the age of 60. MG may present at any age in either sex, although it is rare in children.[3] MG has a strong association with thymic hyperplasia and thymoma. It is estimated that 13% of patients with MG have one or more co-existing autoimmune diseases, most commonly affecting the thyroid gland. There is an estimated rate of co-occurrence of 7% with Graves' disease and 3% with Hashimoto's thyroiditis, which may contribute to symptoms of ophthalmopathy and weakness if present.[5]

Most patients initially present with intermittent flares of ptosis, extraocular muscle weakness, and diplopia. The second most common presentation involves bulbar muscle weakness, which can present in the form of dysphagia, dysarthria, and frequent aspiration.[6] A key feature in diagnosis is weakness that worsens with repeated muscle use and improves with rest, which explains the symptom correlation as typically better in the morning and worse in the evening. Left untreated, weakness may spread and become generalized.

In severe flares, known as myasthenic crisis, the diaphragm and accessory muscles of breathing may become significantly weak, requiring noninvasive positive airway ventilation or intubation with mechanical ventilation. For this reason, prompt diagnosis and initiation of treatment are vital for individuals experiencing their first MG flare.

CASE PRESENTATION

This patient is a 52-year-old male with past medical history of hypertension and hyperlipidemia who presented to the ED with approximately one month of progressive bilateral ptosis and one week of progressive bilateral diplopia and photophobia. The patient reported that these symptoms were more noticeable later in the day and worsened with reading and typing. The patient's gaze disturbance was first noted by his wife, and the patient agreed to be evaluated by an optometrist. The patient was seen and instructed to report to the ED for concern of neuromuscular or intracranial pathology.

After an unremarkable initial workup at an outside ED which included head CT/CTA, basic blood work, chest radiograph, and ECG, the patient was transferred to our tertiary care center for further neurological evaluation.

The patient's vital signs were within normal limits aside from mildly elevated blood pressure. Review of systems was positive for headache, diplopia, and

References available online.

Image 1: Forward gaze on presentation

Image 2: Upward gaze on presentation, note frontalis muscle compensation to lift eyelids

Image 3: Administering the ice pack test

photophobia. The patient denied recent fever, seizures, vertigo, limb weakness, stiffness, or pain. Reassuringly, the patient denied having any respiratory complaints. On physical exam, the patient exhibited bilateral ptosis **(Image 1)**.

Extraocular range of motion was intact, but he had some difficulty with vertical gaze, which was easily fatigable and accompanied with noticeable compensation by the frontalis muscle **(Image 2)**. Additionally, he had marked photophobia, which prevented pupillary light reflex testing due to the patient squeezing his eyelids tightly in response to bright light. The patient was unable to effectively complete visual field testing due to diplopia.

The remainder of his physical exam, including his neurologic exam, was unremarkable. Due to concern for MG based on his history and exam, the ice pack test was performed. A plastic bag was filled with ice and held over his eyes for 2 minutes **(Image 3)**.

The ice pack was then removed, with noticeable improvement of symptoms and exam, including resolution of his ptosis **(Image 4)**. Vertical gaze improved **(Image 5)**, and the patient reported resolution of diplopia and photophobia.

Pupillary light reflex was assessed and found to be normal. This effect lasted for approximately 90 seconds before ptosis reappeared. The test was conducted twice, approximately 15 minutes apart, with identical results, and the presumptive clinical diagnosis of MG was made.

The patient was admitted for additional neurological work-up and diagnosis confirmation.

Blood samples were sent to an outside lab to check for the presence of anti-acetylcholine and anti-MuSK antibodies. Soon after arriving on the floor, the patient was evaluated by the staff neurologist who agreed with our diagnosis and initiated prednisone and pyridostigmine. A chest CT was unremarkable for any thymic or respiratory abnormalities and brain MRI was similarly unremarkable. Approximately 24 hours after first arriving in the ED, the patient reported that his symptoms had greatly improved, and on repeat exam, exhibited no ptosis or vertical gaze deficit.

Image 4: Forward gaze after ice pack test

Image 5: Upward gaze after ice pack test

Images included with written patient consent

DISCUSSION

The mechanism of ptosis in myasthenia gravis is believed to be due to the inhibition of the levator palpebrae superioris muscle at its lower motor neuron synapse with the oculomotor nerve. This is caused by the blockade of nicotinic acetylcholine receptors by autoantibodies. Several mechanisms have been proposed to explain the temporary improvement of ocular MG symptoms after cooling.[1] Although colder temperatures have been demonstrated

References available online.

to decrease the speed of nerve conduction,[7] they also inhibit the action of acetylcholinesterase, resulting in a greater amount of neurotransmitter for a longer period to be present in the gap junction. This mechanism is also applied in the use of the acetylcholinesterase inhibitor pyridostigmine as first-line treatment for myasthenic flares.[6]

In addition to pyridostigmine, immunosuppression with glucocorticoids or azathioprine are indicated for acute myasthenic flares. Second-line immunosuppressive agents include methotrexate and cyclophosphamide. For patients experiencing myasthenic crisis, IVIG or plasmapheresis are indicated due to their rapid onset to prevent respiratory collapse. Thymectomy is recommended in patients with evidence of thymoma, seronegative disease, or anti-nicotinic-Ach subtype of disease if presentation occurs between the ages of 15 and 50.[6]

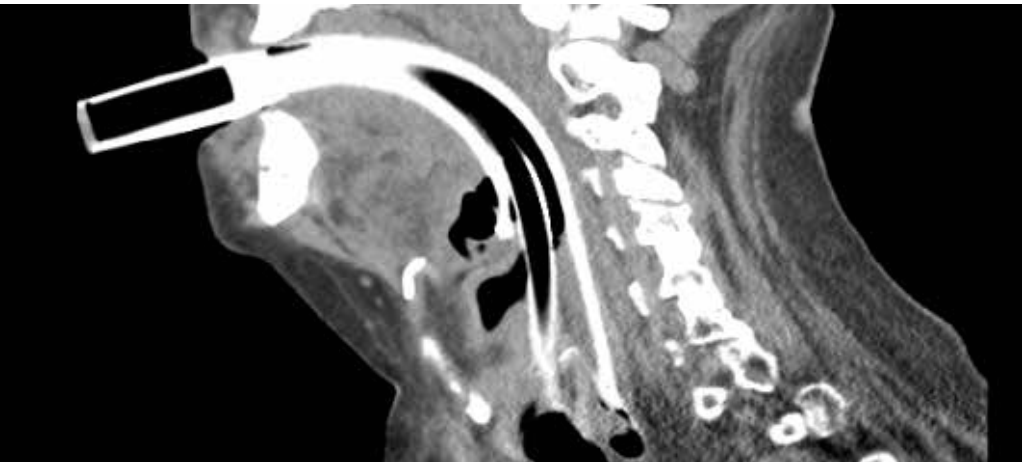
The initial diagnostic workup for patients with MG symptoms should include a complete neurological exam as well as imaging to rule out stroke or intracranial mass, which may present with similar symptoms. Patients should also undergo CT or MR imaging of the thymus as well as measurement of thyroid stimulating hormone to investigate concomitant autoimmune thyroid disease. Differential diagnosis may include disorders of the neuromuscular junction or the peripheral nervous system such as Lambert-Eaton syndrome, botulism, Horner syndrome, neuromyotonia, or Guillain-Barré syndrome. Diagnosis may be confirmed by nerve conduction study or by the presence of anti-acetylcholine or anti-MuSK antibodies, although a small percentage of patients are seronegative.[8]

Historically, checking for symptom improvement in response to intravenous edrophonium, a short-acting acetylcholinesterase inhibitor, was seen as the gold-standard diagnostic test for MG, often called the Tensilon test,[1] but it has since been supplanted by serologic and nerve conduction studies due to the concern for serious side effects including bradycardia and bronchospasm. The ice pack test has proven to be an effective initial method of diagnosis for ocular MG. When used to detect MG in patients presenting with diplopia, ptosis, or both, the ice pack test has been reported to demonstrate a sensitivity of 76.9% and a specificity of 98.3%.[9] The authors of this study found the study to remain highly specific, even in patients with co-existing thyroid dysfunction.

CONCLUSION

Patients presenting with their first MG flare require prompt diagnosis and treatment to prevent progression of symptoms. The initial diagnosis of MG is largely clinical, as confirmatory serologic testing such as Anti-AcH and Anti-MuSK serologic testing may take several days to result, and treatment should not be withheld for such confirmatory testing. In this patient's case, ice-pack testing could have easily been done at the first ED visit. A correct presumptive diagnosis was made quickly at our facility which allowed for confirmatory and prompt initiation of appropriate treatment. With maintenance immunosuppression, the prognosis of individuals with ocular MG is good, with most living a normal life expectancy with minimal disability.[8]

A Tricky Case of Peri-intubation Trismus



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When trismus occurs, unconventional methods might be required to achieve and maintain the airway. This case highlights emergency medicine's nimble approach to the difficult airway.

CASE REPORT

During a night shift, the ED team is called to the floor for emergent intubation of an admitted patient due to sudden hypotension, altered mental status, and inability to protect his airway. The patient is a 56-year-old male with a history of rheumatoid arthritis on prednisone and methotrexate, admitted 2 weeks ago for management of enterococcus faecalis bacteremia, COVID pneumonia, and influenza. Pre-intubation evaluation of the patient reveals an obese, edentulous man able to open his mouth greater than 3 finger widths.

PROCEDURE

Blood pressure demonstrated a MAP of 50 and therefore the patient was not immediately intubated but rather first resuscitated with norepinephrine. He was able to be oxygenated with bag valve mask ventilation and jaw-thrust maneuver throughout this process and was transferred from the floor to the ICU. After his blood pressure normalized, he was given etomidate and rocuronium for rapid sequence intubation. After waiting approximately 1 minute for the rocuronium to take effect, the patient became apneic but developed trismus and his mouth was unable to be opened, deviating from his previous airway exam.

With concern for IV infiltration causing failure of medication delivery, another dose of rocuronium was administered, this time via a separate IV site. Once again, despite waiting a minute, the patient demonstrated trismus, with a maximum jaw opening of approximately 20 mm. Palpation of the temporomandibular joint (TMJ) did not demonstrate any abnormalities in the mandibular fossa, although this assessment was challenging because of the patient's body habitus. Despite these challenges, ventilation and oxygenation were maintained via BVM, and therefore cricothyrotomy was not attempted.

Fiberoptic video bronchoscopy was then attempted. The bronchoscope was inserted through the lateral aspect of the mouth and revealed an edematous airway obscuring the view of the vocal cords. The patient's tongue was sutured to the side to allow insertion of a video laryngoscope, which again revealed a partial view of an edematous supraglottic airway. An ET tube could not be passed through the narrow jaw opening, and the video laryngoscope was removed. An LMA was then able to be inserted through the small opening. The connector cap was removed, and a video bronchoscope was inserted through the LMA with subsequent visualization of an edematous glottis partially obstructing the vocal cords. The disposable bronchoscope was then cut and used as an introducer to pass a size 6.0 ETT through the cords. Video laryngoscope was removed and both LMA and ETT were left in place. Follow-up chest X-ray demonstrated appropriate position of the ETT within the LMA.

Five days later, the ICU team was able to remove the LMA (but kept the ETT in place). The patient's trismus continued throughout the rest of his hospitalization, and he was not extubated. CT maxillofacial bones did not demonstrate any abnormalities of the TMJs or mandible. The oral maxillofacial surgery team was consulted with concern for trismus; however, prior to their evaluation, the patient unfortunately passed away due to worsening septic shock.

References available online.

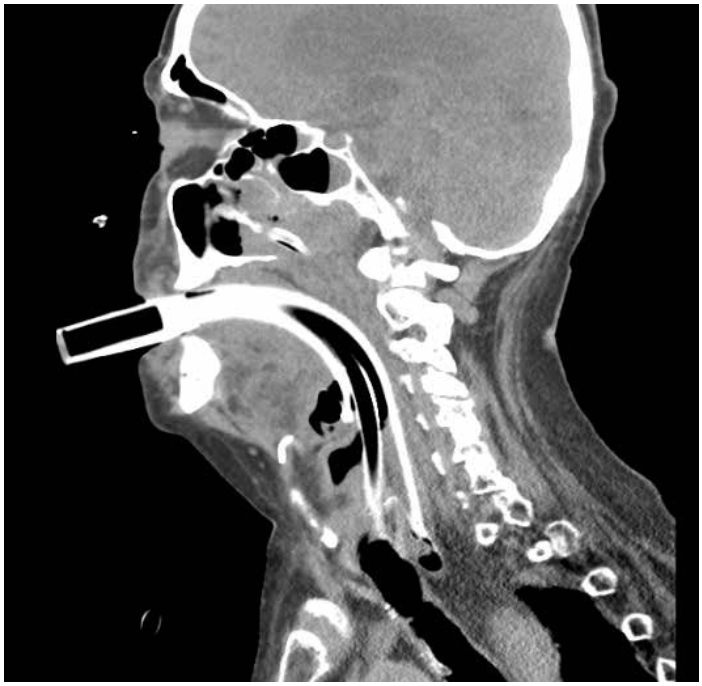


Figure 1. CT of the head and neck without contrast depicting an LMA with ETT in place

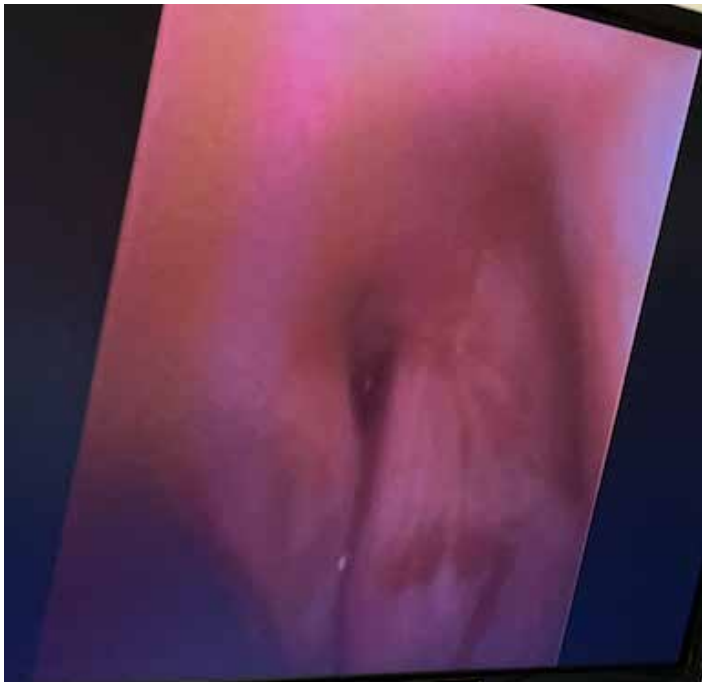


Figure 2. Video laryngoscope view of the supraglottic cavity with vocal cords obscured by swelling

BACKGROUND

"Trismus" is defined as limited jaw opening less than 35-55 mm. In a normally functioning jaw, the muscles of mastication (the medial pterygoid, lateral pterygoid, temporalis, and masseter muscles) attach to the rami of the mandible and contract to close the jaw via innervation by CN V3. Specifically, these muscles coordinate the hinge movement of the condyle in the mandibular fossa as the jaw begins to open. As the jaw opens further, the condyle glides anteriorly, resulting in a fully open mouth. Between the fossa and the condyle, an articular disc serves as cartilage. A problem with any one of these mechanisms may result in trismus.

The presence of trismus is assessed prior to intubation by placing 3 fingers in the patient's mouth. Trismus is considered to be present if the jaw cannot open wider than 3 finger widths. The evaluation of trismus is a component of the 3-3-2 rule, which serves as a quick method to predict a difficult airway. Apart from mouth opening, hyoid-mental distance is measured as 3 fingerbreadths, and thyroid mental distance is measured as 2 fingerbreadths. Any deviation in these brief measurements constitutes a difficult airway that may require cricothyrotomy, nasal intubation, or other advanced techniques that may be more safely done in the operating room.[2]

DISCUSSION

In this report, we discuss the hazard of developing trismus peri-intubation and unconventional methods used to intubate. Past case studies report on trismus before intubation or following extubation, but review of the literature reveals very few cases of trismus developing during intubation.

We considered many different etiologies of acute trismus. Rocuronium (of which we gave a total of 200 mcg) should have provided an adequate neuromuscular blockade to combat any sort of mechanism of trismus that occurred secondary to events at the motor end plates. These include upper motor neuron lesions (patient did have an additional history of strokes), and seizures. Hypocalcemic tetany was considered; however, ABG demonstrated a normal ionized calcium. Etomidate was considered, as side effects include spasm; however, the effects are usually transient and do not last for 5 days.

We also considered the possibility that succinylcholine was given in the place of rocuronium erroneously. This drug can cause malignant hyperthermia with associated muscle rigidity that can be refractory to neuromuscular blockade, but the absence of subsequent fever makes this diagnosis unlikely. A side effect of succinylcholine is also muscular spasms, but once again, we would have expected this side effect to have resolved several minutes following administration. There have been case reports of nondepolarizing agents, such as rocuronium, causing muscular rigidity, but these are very rare, and are thought to occur in patients predisposed to malignant hyperthermia via ryanodine receptor mutations.[3] Both drugs are present in the Emergent Intubation Medication box taken to floor intubation; however, inspection and medication counts confirmed rocuronium had been administered.

We considered extensive supraglottic swelling (believed to be as a result of COVID and influenza infection in this patient) could have contributed to the trismus, as trismus is a known chief complaint of supraglottic swelling.[4] An allergy to one of the intubation medications contributing to pre-existing soft tissue

References available online.

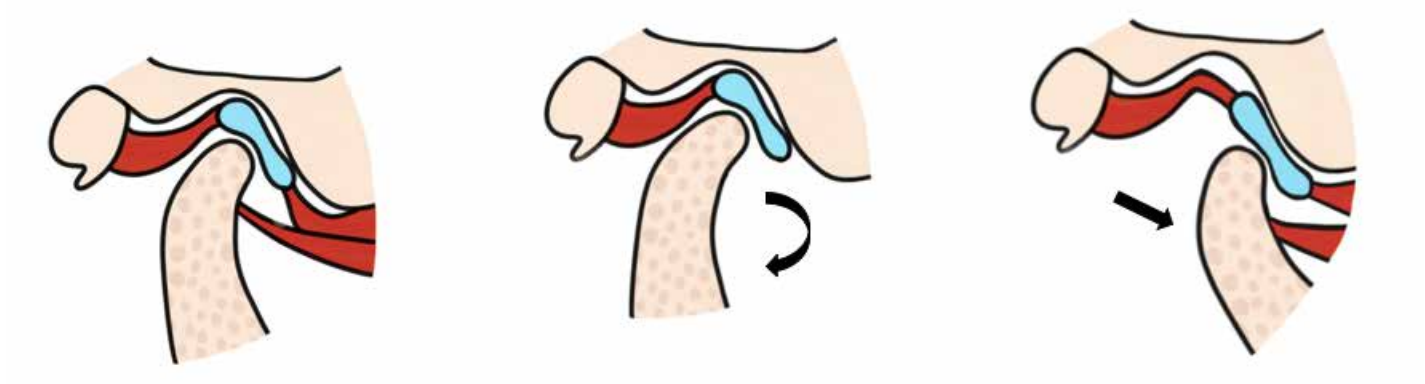


Figure 3. In normal jaw opening, the mandibular condyle sits in the mandibular fossa and initially rotates and then translates anteriorly over the anterior disc (blue)

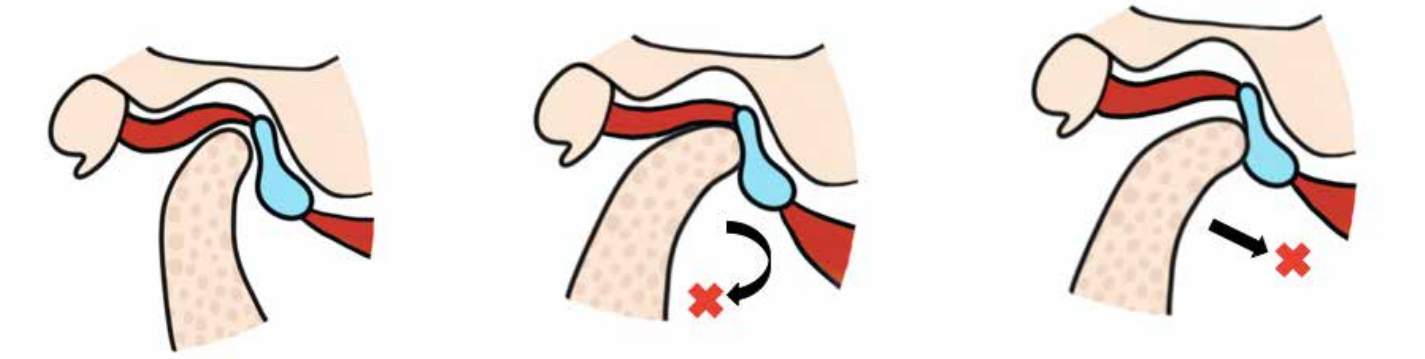


Figure 4. In trismus, the anterior disc can be deranged and the mandibular condyle cannot rotate or translate

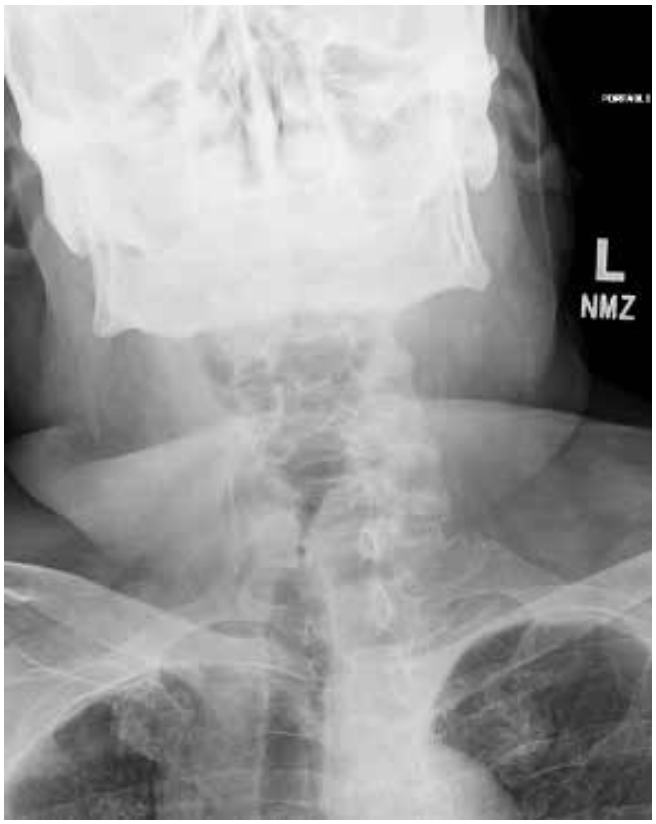


Figure 5. Narrowed airway seen on X-ray

swelling was also considered; however, this is unlikely as patient did not display other physical exam findings of an allergic reaction.

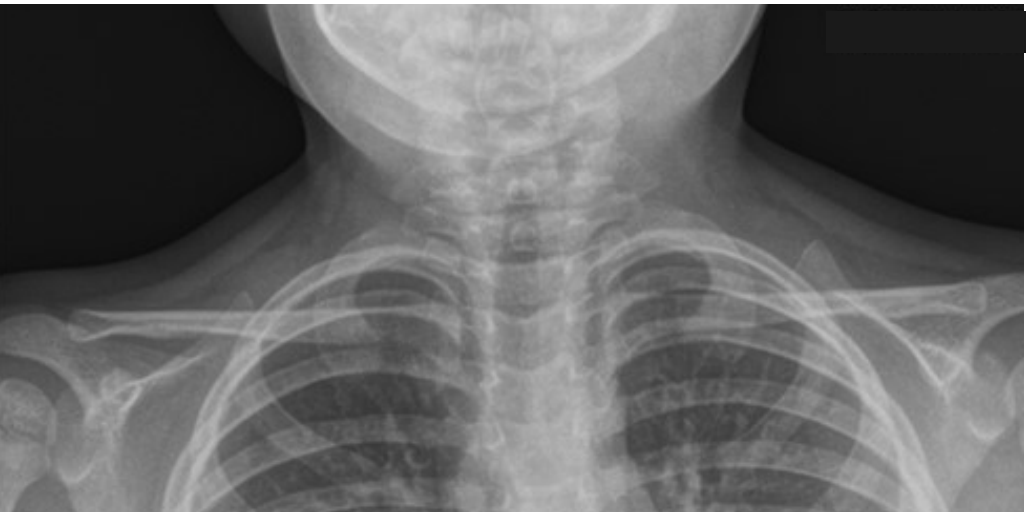
It is possible that the jaw-thrust maneuver, which was held for around 30 minutes to maintain oxygenation as the patient was resuscitated with norepinephrine and transferred to the ICU, caused a TMJ abnormality that resulted in trismus. Displacement of the anterior disc is a known mechanism of lockjaw, as are mandibular fractures. Pre-existing joint space disease caused by the patient's rheumatoid arthritis (the TMJ is affected in 22% of those with rheumatoid arthritis) may have predisposed the patient to sustaining excessive trauma via the jaw-thrust maneuver such that either one or both of the anterior discs were displaced.[5] A mechanical abnormality of the jaw is likely given the trismus did not resolve with neuromuscular blockade or the passage of time. CT maxillofacial was completed following intubation, and the radiologist did not comment on any TMJ bony abnormalities; however, MRI is the gold standard to evaluate the anterior discs as such small cartilaginous abnormalities may be missed on CT.[5]

CONCLUSION

This case highlights that trismus can occur during intubation and that unconventional techniques (e.g., an ETT through a laryngoscope) may be required to allow for a successful intubation. Emergency physicians should be aware that pre-existing rheumatoid disease may affect the TMJ and prepare for the possibility of a difficult airway in these patients.

References available online.

Partial Foreign Body Airway Obstruction (FBAO): Difficult Airway Planning and the Benefits of Interdisciplinary Care



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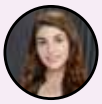

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During a previously quiet Saturday morning, EMS reports they are en route with a 3-year-old male choking on an unknown foreign body. They are 3-5 minutes out, and the patient is saturating 95% on room air, but in obvious distress.

When the patient arrives, he is in respiratory distress, tripoding, and not tolerating oral secretions. He is constantly moving, coughing, and trying to find a position of comfort. Staff begin attempting back blows while getting the patient connected to the monitor. Mom arrives with the patient and says he was found playing in a bag of makeup.

CASE QUESTIONS

- **What do you get ready to manage this patient before EMS arrives?**
- **When are back blows most effective for an airway foreign body?**
- **What if you can't intubate, can't ventilate?**

MANAGEMENT

Prior to administering any medications, video laryngoscope was set up in preparation for visualization and removal of a foreign body and for possible endotracheal intubation. In addition, the equipment needed for a needle cricothyrotomy was prepared as a backup. A chest x-ray showed a small circular foreign body just superior to the vocal cords **(Image 1)**, although later reported as normal by radiology.

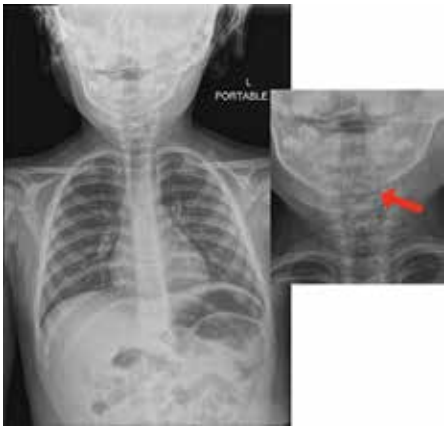


Image 1. CXR with nearly radio-lucent round foreign body sitting at the level of the vocal cords

Ketamine and midazolam were administered, and the patient was placed in partial supine position. Video laryngoscope revealed a lip balm cap sitting at the top of the patient's vocal cords. As the patient was placed further in the supine position, the lip balm cap fell posterior to the vocal cords. Since the foreign body was no longer obstructing the vocal cords, rocuronium was administered and the patient was successfully endotracheally intubated. After endotracheal intubation, the lip balm cap was removed with alligator forceps **(Images 2 and 3)**. Repeat chest X-ray confirmed appropriate tube placement and no additional foreign bodies or pulmonary findings **(Image 4)**.

Image 2. McGill forceps on top, shown for size comparison with alligator forceps below

References available online.

Image 3. Lip balm cap that was partially occluding the airway of the patient

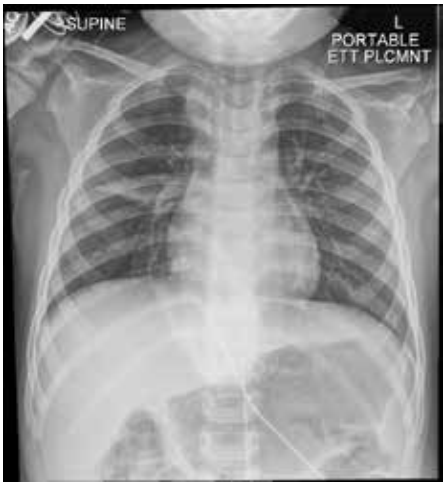


Image 4. Post intubation X-ray

The patient tolerated the intubation well, but developed coarse tight breath sounds as a result of the event. Glycopyrrolate and dexamethasone were administered to reduce secretions and decrease the inflammatory response. Anesthesia was at bedside and aided with rocuronium reversal with sugammadex. The patient was extubated after approximately 1 hour. The patient was then observed in the ED for 4 hours following extubation. After passing a PO challenge, he was discharged home with family.

CASE DISCUSSION

What do you get ready to manage this patient before EMS arrives?

This case highlights the importance of having a strategy that includes a backup plan to manage a potentially difficult airway, including not just equipment, but also interdisciplinary care teams for a difficult airway (anesthesia and surgery). This case could have quickly evolved into a can't-intubate/can't-ventilate scenario with devastating consequences if endotracheal intubation was unsuccessful or if the foreign body obstructed passage of an ET tube.

Given the patient's young age and small upper airway, needle cricothyrotomy would have been the required backup procedure in a can't-intubate/can't-ventilate scenario. Generally, in patients younger than 8 years old, a needle cricothyrotomy would be performed as opposed to standard cricothyrotomy.[1,2] Standard cricothyrotomy is contraindicated in younger patients due to theoretically increased risk of subglottic stenosis[3] and small cricothyroid membrane.[4] If time allows before managing a difficult airway, it is recommended to identify the cricothyroid membrane via palpation or with ultrasound to prevent device misplacement, injury to other structures, and airway trauma.[3] There are several options to set up a needle cricothyrotomy **(Image 5)**.

When are back blows most effective for an airway foreign body?

Back blows are still taught as part of a BLS technique to dislodge a foreign body in a pediatric patient under the age of 12 months; there may be some benefit if the child is small enough to hold in a head-down position. However, it is recommended not to perform back blows if the patient is still able to cough or cry.[5,6] In these cases, the obstruction is incomplete, and the patient may be able to dislodge the object by coughing. Although that did not occur in this case, forceful inhalation could allow for aspiration and complete occlusion of a previously partial airway obstruction.

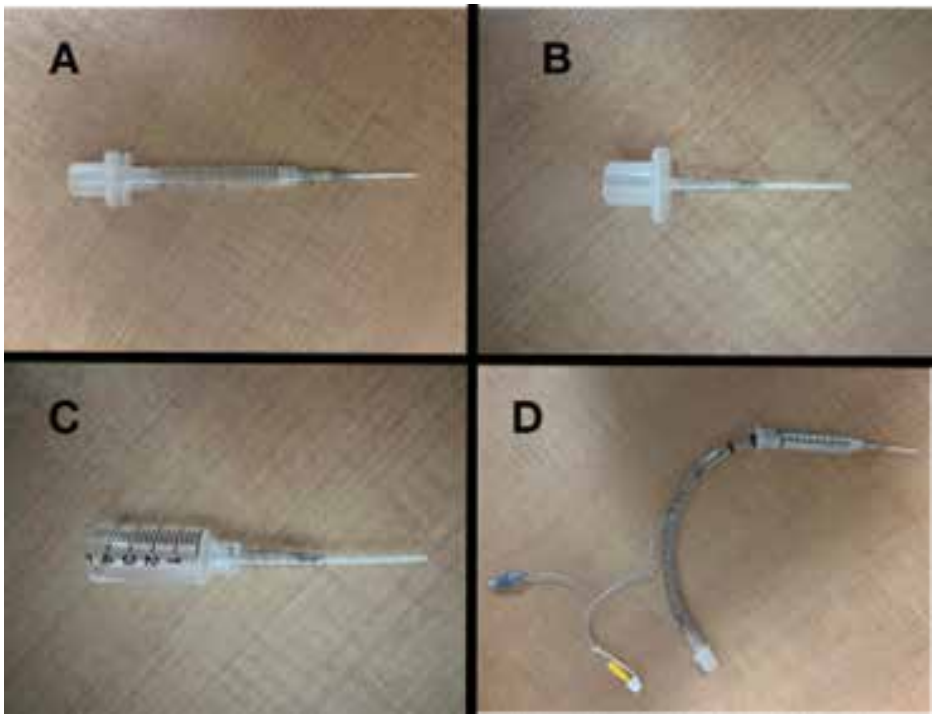


Image 5a. Large-bore angiocath (preferably a 14 or 16 gauge catheter) connected to a 3cc syringe and a size 7 ET tube connector. The size 7 ET tube adapter should fit snugly in the top of the syringe with the plunger removed.
Image 5b. Large-bore angiocath connected to a size 3 ET tube connector. The 3-0 ET tube adapter should fit tightly inside the luer lock connector of a standard angiocath device.
Image 5c. 10cc syringe that was cut in half and connected to a large bore angiocath. The outer diameter of many 10cc syringes is 15mm, thus fitting the universal outlet port of BVM or other airway equipment.
Image 5d. Large-bore angiocath connected to a 10cc syringe, inside of which is a 7.5 ET tube with the balloon inflated. This technique allows for variation in sizes between vendors of equipment but adds complexity and resistance due to length of tubing.

References available online.

What if you can't intubate/ can't ventilate?

Steps to perform a needle cricothyroidotomy[3]

1. **Stabilizing the larynx with your nondominant hand, identify cricothyroid membrane, apply tension to skin over the cricothyroid membrane by stretching skin in vertical direction.**
2. **Use a 3cc or 10cc syringe filled with water or saline attached to an angiocatheter and puncture skin over the cricothyroid membrane, aiming in a caudal direction at a 30-45 degree angle, apply negative pressure until air bubbles are detected in the syringe, stop advancing the needle.**
3. **Advance the angiocatheter and remove the needle.**
4. **Confirm intratracheal placement by aspirating air again.**
5. **Hold the angiocatheter at all times; do not rely on sutures.**
6. **Connect the catheter to high-pressure tubing (jet ventilation) or a bag valve mask with 100% oxygen. Ventilate at a rate of 10-12 breaths per minute, using an I: E ratio of approximately 1:4.**

It is important to keep in mind that jet ventilation requires an unobstructed upper airway to avoid hypercapnia and allow for expiration. Jet ventilation is not recommended for long term ventilation.[3] If using a BVM to deliver breaths, the physician needs to consider occluding or covering the pop off valve (if present) to allow delivery of high-pressure oxygen due to the narrowness of the angiocatheter.

COMPLICATIONS

A case by Okada et al. in 2017 describes a 3-year-old boy in cardiac arrest secondary to an upper airway obstruction from idiopathic anaphylaxis or acquired angioedema that led to a cannot intubate, cannot ventilate scenario. Video laryngoscopy failed and needle cricothyrotomy was considered, but the physicians were unable to identify the proper angle to insert the needle due to lack of space (i.e., small pediatric neck with a lot of subcutaneous fatty tissue). Physicians were concerned the posterior trachea would have been inadvertently punctured. In this case, they proceeded to standard cricothyrotomy with a cricothyrotomy kit. This failed, as the space between the cricothyroid cartilages was smaller than the cannula in the kit and the cannula kinked. Emergent tracheostomy was attempted and finally successful. Unfortunately, the patient died, and autopsy revealed the cricothyroid membrane was only 3 mm in size.[7]

Most common complications during cricothyrotomy include bleeding, injury to tracheal cartilage, perforation of trachea, false tract creation, infection, subglottic stenosis.[3] Neonates are particularly difficult to obtain emergent airway access due to their smaller anatomy. Even with proper positioning and placing their necks into extension, successfully palpating the cricothyroid membrane can be difficult. Once the cricothyroid membrane is identified, the steep angle needed to enter the membrane increases the risk of posterior tracheal penetration.[7]

If the physician is able to successfully perform a needle cricothyrotomy, jet ventilation carries complications including emphysema, pneumothorax, and lung injury.[1] Some experts even suggest that surgical cricothyrotomy or tracheostomy may be preferred in children younger than 6 years old because the trachea is so small and chance of posterior tracheal wall injury is high, but this is controversial.[1] In neonates, the mean length of the cricothyroid membrane is 2.6 ± 0.7 mm and a width of 3.0 ± 0.63 mm, compared to the average adult cricothyroid membrane 13.7 mm long and 12.4 mm wide.[8]

The Difficult Airway Society guidelines suggest that standard cricothyrotomy be performed if needle cricothyrotomy fails.[1] If ENT is emergently available, emergent tracheostomy can be considered as a first step. Emergent tracheostomy at the proximal trachea with scalpel, finger, bougie technique is another option to be considered if other options fail.[7]

TAKE-HOME POINTS

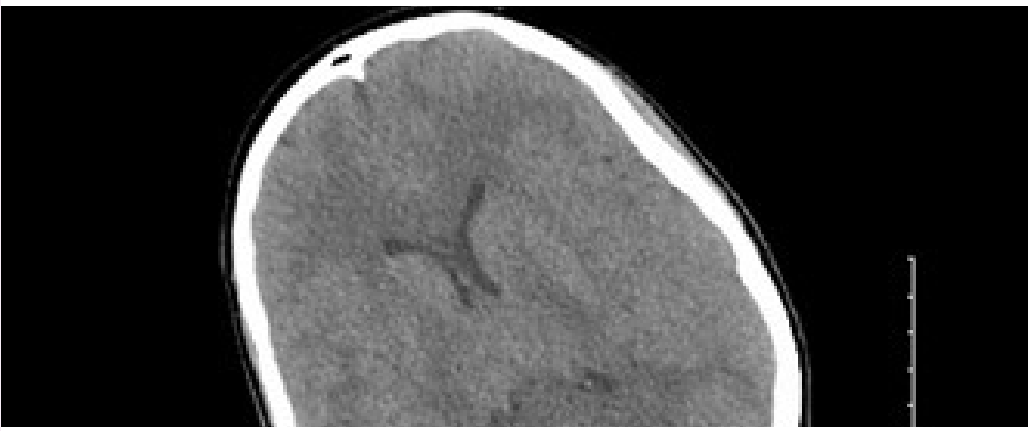
FBAO is an anxiety-provoking clinical encounter, especially in a pediatric patient. Planning ahead by having people and tools available to support anticipated care—including potential complications—is important, as is practicing for and simulating these types of high-risk situations.

One tool that was particularly useful in this case was the alligator forceps that allowed for removal of the foreign body. Often McGill forceps are considered for airway use, but in this pediatric patient, this tool was simply too large to work with the small airway and space available **(Image 2)**.

Additionally, the availability of the anesthesia team to support the reversal of rocuronium with sugammadex allowed this patient to be extubated in the ED, saving an ICU admission.

References available online.

A Case of Influenza A Encephalitis in an Adult with Addison's Disease



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Influenza is a common viral respiratory illness; however, in certain patient populations it can lead to more severe manifestations—including extrapulmonary complications. Patients at increased risk include children, older adults, and those who are immunocompromised.

CASE REPORT

A 31-year-old female with a past medical history of Addison's disease presented to the ED with altered mental status. She was febrile, tachycardic, and unable to follow commands. Initial evaluation demonstrated hyponatremia, positive influenza A nasal swab, and cerebral edema on head CT. Initial evaluation was suspicious for Influenza A encephalitis.

The patient was started on normal saline, prophylactic antibiotics, and stress steroids. Further evaluation with head MRI showed areas of acute infarction in splenium of corpus callosum and deep parietal white matter bilaterally, and areas of increased signal on FLAIR weighted images. This led to the patient's transfer to a tertiary care facility, where she received antivirals and supportive care. After 3 days, she was able to be discharged home with no lingering neurologic deficits.

SIGNIFICANCE

Physicians should recognize influenza encephalitis as a possible cause of altered mental status during influenza season. Furthermore, it is important to recognize Addison's disease as an increased risk for extrapulmonary manifestations of influenza.

INTRODUCTION

Influenza A is a common cause of respiratory illness that is typically self-limiting. However, in immunocompromised patients, influenza A can be more severe and lead to extrapulmonary manifestations such as encephalitis.[1] Influenza A encephalitis cases are usually seen in children and rarely does influenza cause cases of encephalitis in immunocompetent adults.[2] Adults affected by influenza A encephalitis typically recover but it can lead to lasting neurologic deficits.[2]

Addison's disease is a rare condition of adrenal insufficiency in which there is an inability to create glucocorticoids, mineralocorticoids, and androgens by the adrenal cortex. The prevalence is estimated to be around 5 new cases/million/year.[3] Typically, Addison's disease presents with symptoms such as fatigue, weakness, lethargy, muscle cramps, gastrointestinal complaints and hypotension. Addison's disease has several causes including autoimmune, infectious, neoplastic, and surgical.[3] Although not considered an immunocompromised state, patients with Addison's disease are considered clinically vulnerable.[4] Addison's disease has been shown to have a two-fold risk ratio for death and five times higher mortality rate from infections.[4] This increased morbidity and mortality is thought to be due to adrenal crisis caused by acute illness[3] and impacted natural killer cell cytotoxicity.[4]

Though uncommon, influenza can lead to more severe sequalae such as encephalitis and should be considered in patients presenting with neurologic symptoms during influenza season. Further, while not considered an immunocompromised condition, it is important to consider adrenal insufficiency as a risk for increased severity of illness and begin prompt treatment to avoid adrenal crisis.

CASE PRESENTATION

This is a 31-year-old female who presents via EMS to the Emergency Department for altered mental status and influenza-like illness.

References available online.

- **HPI: Patient's historian is her partner. He reports she has not been feeling well for the past couple days. She has not been eating or drinking much. She then began acting strangely and was not following commands which prompted her partner to call EMS. He notes she has Addison's disease for which she takes steroids daily. He does not recall her last known well.**
- **No Review of Systems performed due to altered mental status.**
- **Past Medical History: Addison's disease controlled with daily steroids**
- **Past Surgical History: none**
- **Fam History: none**
- **Social History: no alcohol, no smoking, no substance use**
- **Allergies: none**
- **Medications: prednisone - 5 mg in morning, 1 mg in afternoon, florinef (mineralocorticoid) - 0.1 mg daily**

Physical Exam

Vitals: 100.5, 107, 115/82, 18, 100%

Physical exam revealed an ill-appearing woman, with no scleral icterus, intact extraocular movements, no nystagmus and pupils equal and reactive to light. She was tachycardic but had no murmurs. She was not in acute respiratory distress and had no wheezes or rales. Her abdomen was soft and nontender. She had no cervical neck rigidity. She did not exhibit any abnormal muscle tone or seizure activity. She did not exhibit any facial asymmetry. She did appear confused and would inconsistently follow commands. Her GCS eye score was 4, verbal 3, and motor 5.

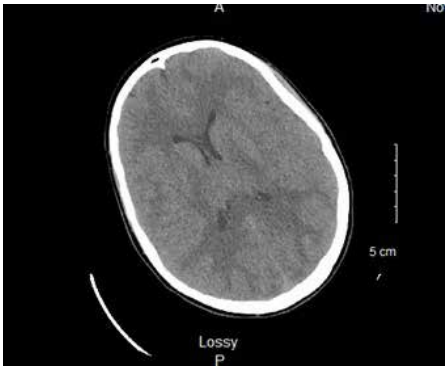

Figure 1. CT head read as small ventricles, effacement of third ventricle and bilateral cortical sulci, suggestive of cerebral edema. No herniation or midline shift. No acute ischemic process or intracranial process.

Labs/Imaging

Labs done upon initial evaluation included:

- **Cortisol: 150**
- **Viral Swab: flu A +, flu B –, RSV –, COVID –**
- **CMP: Na 126, K 3.7, Cl 96, CO2 20, Anion Gap 10, glucose 194, BUN 10, Cr 0.84, BUN/Cr 11.9**
- **Lactic Acid: 1.8**
- **CBC: WBC 9.24, RBC 5.05, Hgb 14.8, hct 41.5**
- **Blood cultures: resulted negative after 120 hours**
- **TSH: 3.543 #1 at 4:50 pm**
- **EKG: sinus at 90, normal intervals, no ST-T changes**
- **CXR: no acute cardiopulmonary disease**

Differential after Initial Evaluation

- **Based on the initial evaluation and physical exam, the differential included:**
  - Influenza encephalitis
  - Hyponatremia
  - Meningitis
  - Toxicity from unknown substance

Following evaluation included:

- **CSF:**
  - Cell Count/Differential
    - tube 1: 284 RBC, 1 nucleated cell, 23% neutrophils, 66% lymphocytes, 11% monocytes
    - tube 4: 1 RBC, 2 nucleated cells, 83% lymphocytes, 16% monocytes, 1% neutrophils
  - Gram stain/culture negative/no growth
  - CSF glucose 59 (normal), protein 28 (normal)
  - BioFire NAAT CSF negative for: cryptococcus gattii, CMV, enterovirus, Ecoli K1, H flu, HSV ½, HHV6, Human Parechovirus, listeria monocytogenes, strep pneumo, VZV
- **TSH: 1.209 #2 at 8:30pm**
- **Urine Na: 117**
- **Na: 130 (This repeat sodium was after 1 L of Normal Saline)**
- **Blood osmolality: 269**

DIAGNOSIS: INFLUENZA A ENCEPHALITIS

Management

Initially the patient was given 1 L NS for hyponatremia, which improved the sodium from 117 to 130. The initial evaluation led to a consult with Neurology, who suggested an MRI as well as a lumbar puncture. For management with the history of Addison's disease, Endocrinology suggested stress steroids. Teleneurology also recommended beginning antibiotics prophylactically. She was started on vancomycin and ceftriaxone.

Due to the patient's age and the infarcts seen on MRI, Neurology recommended transfer to a tertiary care facility.

Outcome

The patient spent 3 days at a tertiary center, where she received supportive care and antiviral influenza treatment. She was able to be discharged home and resumed her normal activities with no neurologic deficits.

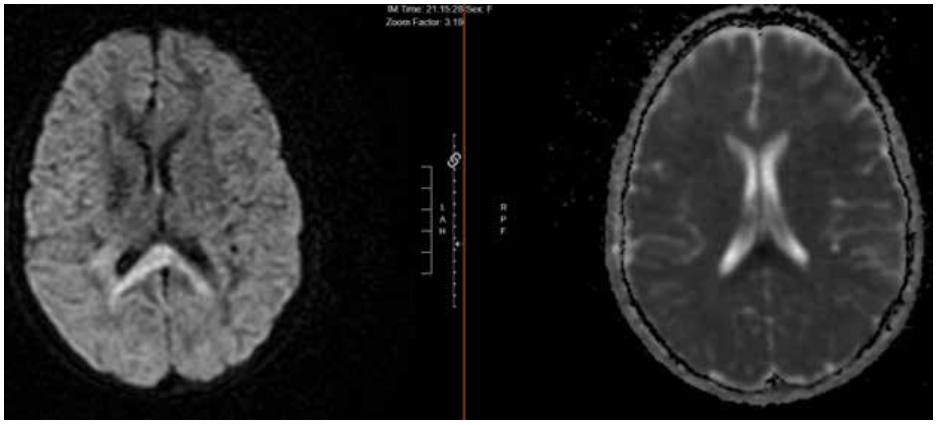

Figure 2. MR Brain w/w/o contrast read as areas of acute infarction in splenium of corpus callosum and deep parietal white matter bilaterally. Areas of increased signal on FLAIR weighted images are suspicious for encephalitis/meningitis given history.

References available online.

Discussion

The diagnosis of Influenza A encephalitis is uncommon, especially in immunocompetent people. Typically, influenza causes more severe complications in older adults or those who are immunocompromised. Extra-pulmonary complications of influenza include myocarditis, ischemic heart disease, encephalitis, Guillain-Barré, myopathy, and others.[5] Most cases of influenza encephalitis occur in children.[1] It has been shown that in patients with Influenza A encephalitis, 79% survive, but 25% had neurologic deficits including motor weakness and cognitive deficits.[5]

The diagnosis of influenza encephalitis is difficult given the lack of validated diagnostic criteria. The diagnosis should be suspected in patients with fever and neurologic symptoms, especially during influenza season.[6] There are no clear diagnostics but work-up includes nasopharyngeal swab/culture, blood work, CT, MRI, and lumbar puncture.[6] In those who had brain CT, 39% showed acute changes, 50% were normal, and 11% showed chronic changes.[5] All the patients who completed an MRI had abnormal findings.[5] Lumbar puncture findings including pleocytosis (17%), elevated protein (17%), and were normal in most cases (46%).[5] In this patient, CT and MRI both showed acute findings, but the CSF findings were normal. Brain MRI is more specific and the preferred method of diagnosis of encephalitis.[6] CSF fluid can be analyzed for influenza with PCR but has only been found to be positive in 16% of patients.[6]

A preventative measure from development of severe influenza illness is the influenza vaccine. Our patient had unknown vaccination status, and if they did not receive the annual influenza vaccine it may have also contributed to development of encephalitis. Influenza vaccination has an efficacy from 59-83%.[6]

There is no standard therapy for influenza encephalitis management.[6] Treatment for influenza encephalitis mainly consists of supportive care, antivirals (such as neuraminidase inhibitors and amantadine), glucocorticoids, antiepileptics in specific cases, and immunomodulatory medications such as gamma globulins and alpha interferon.[6] In the case of our patient, antibiotics were initiated prophylactically while test results were pending, and they were also started on antivirals. They also received supportive care. The role of glucocorticoids in this patient were more specific for Addison's disease.

For our patient, it is likely that Addison's disease played a role in the development of severe symptoms. Studies have shown that Addison's disease decreases the immune response and the ability to fight infection, which may have contributed to this patient's severe symptoms.[4] With a reduced immune response, the patient may have developed viral CNS invasion or increased cytokine release.[6]

The patient's hyponatremia may have further exacerbated the cerebral edema seen on imaging. The hyponatremia may have been caused by a degree of adrenal crisis; however, the patient was not hyperkalemic and had normal glucose, so it is more likely that dehydration was the cause, but it remains important to consider stress steroids in patients with Addison's disease to prevent adrenal crisis.[7]

Take-Home Points

This case highlights the need for including influenza encephalitis on the differential in presentations of altered mental status during influenza season. It is also important to perform comprehensive work-up for encephalitis due to the lack of diagnostic criteria.

This case also highlights that severity of illness can be increased in those with Addison's disease, and the need to initiate steroid management to prevent Addisonian Crisis.

References available online.

The New Playbook on Concussions: Updated Guidelines for Prevention, Management, and Recovery

Joshua Rayburn, MD
Creighton University, Phoenix

An estimated 1.6 to 3.8 million sports-related concussions occur annually in the United States, a number that has steadily risen over the past two decades.[1,2] This growing prevalence has driven continued advancements in concussion management, propelled by heightened awareness, mounting research, and increased media focus. Reflecting on the latest developments, the 6th International Conference on Concussion in Sport (Amsterdam 2022) unveiled an updated consensus statement, offering new insights and recommendations on the definition, prevention, screening, management, and recovery of sports-related concussions.[3] These updates aim to enhance safety, streamline care, and improve outcomes for athletes at all levels. This article will review those recommendations and explore their relevance in the context of emergency medicine care.

UPDATED CONCUSSION DEFINITION

The Concussion in Sport Group (CSG) redefined sports-related concussions as traumatic brain injuries caused by a direct blow to the head, neck, or body during sports activities, with forces transmitted to the brain. The injury is theorized to result in a cascade of neurological and metabolic changes that may cause a range of symptoms such as headaches, dizziness, or balance disturbances that may emerge immediately or evolve over hours and days. Concussions do not show abnormalities on routine imaging like MRI or CT scans. The CSG continues to work towards a unified and operational definition as the breadth of research on the topic and understanding of head injuries continues to grow. There are currently no universally accepted or standardized diagnostic criteria for concussion.

PREVENTION

The consensus guidelines emphasize preventative strategies and their importance in reducing concussion incidence and recurrence. Awareness of these strategies can help emergency medicine clinicians educate patient athletes, parents, and coaches and hopefully decrease the risk of concussions for athletes. Specific examples include:

- **Rule Changes:** Reducing collisions and high-risk actions has been associated with a decrease in concussion incidence. A meta-analysis and systematic review found that disallowing body checking in youth hockey was associated with a 58% reduction in concussion rates. Limiting the amount of full-contact practice in American football was associated with a 64% reduction in concussion rates.[4]
- **Protective Equipment:** Mouthguards are recommended for all levels of contact play. Use of mouthguards among hockey players of all ages showed a 28% reduction in concussion rates.[4] Helmets and headgear are likely protective as well; however, more research is needed to support recommendations on these.
- **Neuromuscular Training (NMT):** Warm-up programs focusing on neuromuscular control demonstrated lower concussion rates as well fewer musculoskeletal injuries. Rugby players who underwent the NMT training protocols three times per week had 59%

References available online.

fewer concussions compared to the control group.[5] NMT training included at least 20 minutes of integrated balance, resistance, and plyometric training with sport-specific maneuvers and verbal feedback and reinforcement from coaches. Further research is still needed across other sports and populations.

- **Optimal Management Strategies and Education:** Recommended strategies—including mandatory removal from play following concussions, concussion clearance protocols, and awareness campaigns focused on education of coaches, parents, and athletes—together have been associated with a 63% reduction in rates of recurrent concussions.[4]

While emergency medicine physicians may be more focused on the treatment and management of concussions after the initial injury, providing education and reinforcing these strategies can help prevent future concussions for athletes in the community.

SCREENING TOOLS

Revised concussion screening tools include the Concussion Recognition Tool 6 (CRT6), Sport Concussion Assessment Tool 6th Edition (SCAT6), and Child SCAT6 for field assessments as well as the Sport Concussion Office Assessment Tool (SCOAT6) for following clinical settings. These tools are most effective within 72 hours of injury and support serial evaluations throughout recovery. For example, the SCAT6 involves several steps that evaluate the athlete's background, symptoms, cognitive function (orientation, memory, and concentration), coordination and balance, and delayed recall.[6] This provides a framework for the athlete to receive standardized evaluations, which can be used to track the severity of their concussion and monitor improvement over time. Familiarity with these screening tools can aid physicians in diagnosing concussions in the ED and can help guide patient education and safe disposition planning.

MANAGEMENT

Updated recommendations emphasize early physical and cognitive activity with targeted treatments, with core concepts detailed below. Newer research has continued to support that strict rest is not as beneficial as early return to physical and cognitive activity at light intensity as tolerated,[7] which is a continued shift from concussion management in the past.

- **Immediate Removal:** Any athlete with a suspected possible concussion should be removed from play immediately. Initial signs may include loss of consciousness, confusion, amnesia, behavioral changes, seizure, impaired balance, or ataxia.
- **Early Physical Activity:** Relative but not strict rest is recommended after sustaining a concussion. Athletes should be encouraged to do their normal activities of daily living as tolerated within the 24 hours after the injury. Light aerobic exercise within 24-48 hours has been shown to improve recovery from concussion.[7] Examples include walking, stationary cycling, and other physical activities if they avoid physical contact, collisions, or falls. Activities should be symptom-limited, with intensity adjusted based on tolerance.
- **Gradual Increase in Exertion:** Anaerobic exercise, physical activity, and cognitive exertion should be gradually increased over time, and stopped if concussion symptoms are exacerbated. Recovery and return protocols are reviewed below.
- **Reduce Screen Use:** Athletes should reduce screen time on electronic devices during the first 48 hours after sustaining a concussion. Increased screen use during the initial recovery period has been associated with both an increased duration of recovery and increased symptom severity scoring.[8,9] Reduced screen use beyond 48 hours may not be effective but should be limited by symptom exacerbation.
- **Targeted Rehabilitation:** Cervicovestibular rehabilitation therapy is recommended and beneficial for athletes with persistent symptoms like headaches or dizziness that last for more than 10 days after the initial injury.[10] Referral to specialists is advised for complex cases involving migraines, cognitive challenges, or psychological issues lasting more than two weeks.

RECOVERY AND RETURN PROTOCOLS

Return to Learn (RTL): Most athletes resume academic activities within 10 days.[11] Many student athletes may return to school with minimal difficulty or limitations, while others may require a targeted stepwise symptom-limited return strategy. Initial adjustments should be communicated to the patient's educators, which may include limited screen use, reduced workload, curriculum and testing adjustments, modified school attendance, and frequent breaks from cognitive tasks. Strict rest or avoidance during the initial 24-48 hours should not be recommended. Duration of tasks may begin at 5 to 15 minutes at a time with incremental increases in cognitive load over time, ensuring no significant or prolonged symptom exacerbation. Mild symptom exacerbation is typically brief and has not been associated with delayed recovery.

Return to Sport (RTS): There is strong and clear evidence to suggest that strict rest is not beneficial in the immediate recovery phase after sustaining a concussion.[7,12] The proposed recovery strategy involves a stepwise approach detailed below. Each step generally takes a minimum of 24 hours, and if more than mild symptom exacerbation occurs, the athlete should stop and attempt to exercise the following day. All athletes should be evaluated by a medical provider and determined to be safe to return to sport prior to engaging in activities at risk of head impact including physical contact, collisions, or falls.

- **Step 1:** Symptom-limited activities may be started within the first 24 hours of injury. This generally includes daily activities of living and walking. Activities during any stage should

References available online.

not cause more than a mild or brief exacerbation of symptoms, defined as no more than a 2 on a 0 to 10 point scale (0=no symptoms, 10=worst symptoms imaginable).

- **Step 2A/2B:** Gradual progression generally starts with aerobic activity. Athletes should progress to light activity up to approximately 55% of their max heart rate (step 2A), then to moderate activity up to 70% of their max heart rate if tolerated (step 2B). Activities in this step may include stationary cycling, walking at a moderate pace, and light resistance training.
- **Step 3:** If tolerating aerobic activity, athletes then progress to individual sport-specific exercises such as running, change of direction, and individual skill drills. There should be no risk of head impact during this step. In general, athletes should be evaluated and cleared by a health care provider prior to progressing to step 4.
- **Step 4:** Athletes may be integrated into a team environment at this stage and may participate in non-contact training drills and exercises at high intensity. Exacerbation of concussion symptoms during activities in steps 4-6 warrants return to step 3 until there is full resolution of symptoms.
- **Step 5:** Athletes participate in full contact practice and resume normal training activities. Functional skills and appropriateness to advance should be assessed by coaching staff.
- **Step 6:** Return to normal game play. Athletes can expect at least 1 week to complete the full RTS rehabilitation strategy and may take longer, depending on individual characteristics and tolerance of activity at each step.

LONG-TERM CONSIDERATIONS

Research into the long-term effects of concussions—including depression, neurodegeneration, and traumatic encephalopathy syndrome (TES)—remains ongoing but is currently limited. There are increasing concerns that concussions and repetitive head trauma may cause long-term cognitive impairment or adverse neurologic outcomes; however, current data are limited and insufficient to suggest or confirm a causal relationship. TES is a clinical diagnosis involving exposure to repetitive head impacts with clinical features of cognitive impairment which follows a progressive course and is not explained by alternative conditions.[13] Chronic traumatic encephalopathy (CTE) is not a clinical diagnosis but rather is made via post-mortem neuropathology and should not be diagnosed by ED clinicians. Future studies may shed light and help identify the long-term impacts of concussions and repetitive head trauma in athletes.

CONCLUSION

The consensus guidelines from the 6th International Conference on Concussion in Sport provided new insights and helpful guidance in the management of concussions. Clinicians, coaches, and athletes can collaboratively promote safer sports environments and optimize recovery from concussions by adopting these updated strategies and integrating them into their conversations with patients. Emergency medicine clinicians play a key role in the diagnosis of concussions among athletes and other patient populations and can help to improve the time to recovery, reduce severity of symptoms, and prevent future concussions for these patients.

TAKE HOME POINTS

- **Concussion is a clinical diagnosis and there are no standardized diagnostic criteria. Head imaging is not indicated unless concern for alternative diagnoses.**
- **Consider reviewing screening tools such as SCAT6 or CRT6.**
- **Recommend avoiding unnecessary collisions when possible.**
- **Recommend mouthguards for all athletes.**
- **Recommend targeted neuromuscular training in warm-ups.**
- **Immediately remove any athlete with a suspected concussion from play.**
- **Early return to physical and cognitive activity should be advised, which should be increased gradually and limited by symptom exacerbation. Do not recommend strict rest.**
- **Reduce screen use in the first 48 hours.**
- **Stepwise protocols should be advised for returning to play and to school.**
- **Athletes should not return to full contact until cleared by a medical provider.**
- **Consider referral to a concussion specialist if symptoms persist more than 2 weeks.**

References available online.

empower
Sharing Our Stories

Morgan Sweere, MD, MPH
EMRA Board Secretary
EM Resident Editor-in-Chief
University of Florida — Jacksonville

A Deep Dive with Thom Mayer, MD, FACEP

We take a moment to learn from Thom Mayer, MD, FACEP, during this edition of EMPower. Dr. Mayer has been a leader in times of crisis for over 25 years, navigating significant challenges on the global stage.

Dr. Mayer is the Medical Director for the NFL Players Association, as well as an emergency physician-sports medicine leader of international renown and author of several books. He served as the Command Physician at the Pentagon Rescue/Recovery Operation on 9/11, Incident Commander for the inhalational anthrax outbreak in Washington, DC, that same year, and led a Team Rubicon Mobile Emergency Team in Ukraine following the outbreak of war. He is among the most widely respected leaders in times of crisis and is a highly sought-after speaker and consultant across many businesses and industries.

He was recently nominated to the Pro Football Hall of Fame in Canton, Ohio, and is a member of the Indiana Football Hall of Fame and the Hanover College Athletic Hall of Fame. *USA Today* named him one of the "100 Most Important People in the NFL." In sports medicine, his work at the forefront of changing concussion diagnosis and management in the NFL has changed the way these athletes are diagnosed and treated.

WHAT IS YOUR FAVORITE THING ABOUT EMERGENCY MEDICINE?

Everything! I have three boys, and I drove them to school every day. I would say to them that each day is "one more step in the journey of discovering where your deep joy intersects the world's deep needs." That's what my job gives me—deep joy! What we get to do every day is actually pretty profound.

IF YOU WERE RESTARTING RESIDENCY, WHAT ADVICE WOULD YOU GIVE YOURSELF?

Be more kind, thoughtful, and generous. You have the ability to make a difference in every patient encounter. In football, the players spend 3-5 times more time in the film room than on the football field. What does that mean? The preparation matters. When I look back at my training, I see opportunities where I could have been better. I would say that the preparation matters, and make failure your fuel to be better.

WHAT IS THE MOST IMPORTANT TRAIT FOR A LEADER TO HAVE?

To be kind. In fact, the first, second, and third most important traits are to be kind. As Reverend John Watson said, "Be kind, because everyone is fighting a great battle."

WHAT IS YOUR BEST TIME MANAGEMENT TIP?

Do it now. No to-do lists. Only to-don't lists.

FAVORITE CHIEF COMPLAINT

Trauma. I love things where I can immediately do something and see the effect. However, I think you have to see every complaint and love it and take advantage of its learning opportunity. I've worked in high-volume emergency departments my entire career, and there's something to learn from every patient.

WHAT IS THE MOST SIGNIFICANT RISK YOU HAVE TAKEN AND THE OUTCOME?

I've had multiple entrepreneurial adventures, including my own ambulance company and my own EM practice group. Shakespeare said, "Uneasy lies the head that wears the crown." When you start something and you're not sure it's going to work, it's scary, but it's part of life. Without risk, there is no reward.

WHAT IS THE BEST ON-SHIFT SNACK?

Swedish fish.

WHAT ARE YOU LEARNING ABOUT RIGHT NOW?

The most recent is escorting 40 D-Day veterans back to the Normandy beaches. While I've been a student of WWII history for years and grew up around my dad's friends who were veterans, being immersed in the history was staggering. It's translated differently when you see it with your own eyes. It helps you realize that every patient has a story, and we are the chief storytellers as emergency medicine physicians. We tell the patients the story of their visit, and we tell the consultants the stories of our patients.

WHAT IS YOUR FAVORITE SONG TO HYPE YOU UP BEFORE A SHIFT?

Jimmy Buffett's "Incommunicado."

WHAT MESSAGE WOULD YOU PASS ALONG TO THE READERS OF EM RESIDENT?

The leader you're looking for is you. Forget someday and embrace today. The answers are not above us; they are within and among us.

"Stop sucking up and start sucking down." When I was Command Physician at the Pentagon during 9/11, I had to work to secure the scene, so that we could get in there to recover people. I wanted to work with different people as part of a team so that we could go into that building and do it safely. The people who are "below you" now will be the people you need in the future.

Editor's note: Be sure to check out Dr. Mayer's books, Battling Healthcare Burnout, Leadership Is Worthless but Leading Is Priceless, and more – all available in Amazon.

References available online.

Announcing New EMRA Benefits

EMRA members, make life easier with two valuable new member benefits designed to help you be the best doctor you can be.

***BOUNCEBACKS!* YOUR GO-TO MEDICO-LEGAL BOOK**

The authoritative resource for medico-legal insight is back with a new anniversary edition, and **EMRA members get 20% off**, while simultaneously earning money for EMRA to reinvest in you. **TO GET THE DISCOUNT:** Scan the QR code and use discount code "EMRA" when purchasing.

***BOUNCEBACKS! EMERGENCY DEPARTMENT CASES: ED RETURNS* 10TH ANN. ED.** explores actual ED cases that went bad. Reading these cases helps develop your insight to avoid costly diagnosis errors and to improve your patients' safety.

AVOID SERIOUS MISTAKES IN THE EMERGENCY DEPARTMENT!

How ***Bouncebacks!*** helps you:

- **Thorough:** These cases cover many of the diagnostic pitfalls and risks associated with emergency medicine.
- **Practical:** These are real cases that went bad—see how better documentation could have improved patient care.
- **Authoritative:** Benefit from the insight of Michael Weinstock, MD, Risk Management Section Editor for EM:RAP.

For more insight, listen to our EMRA*Cast episode with Dr. Weinstock! (Find it on your favorite podcast platform or visit www.emra.org/emracast.)

RAPIDRADS MED ED: RADIOLOGY FOR EMERGENCY MEDICINE

How confident do you feel in your ability to interpret radiographic images?

Two emergency physicians, Chris Reilly and Gary Cook, understand the uncertainty, the pressure, and the consequences that come with not having strong imaging skills when every minute matters.

They designed ***RapidRads***, a video-based radiology education platform, to help you **build confidence and competence when reading radiographic images.**

EMRA programs get 3 free seats in this valuable platform! And EMRA members can access a 20% discount, courtesy of your EMRA membership.

The RapidRads curriculum includes 12+ hours of content on **adult and pediatric X-rays, CT interpretation, and EM board review prep**, all reviewed and approved by **board-certified radiologists** and aligned with EM clinical practice.

BOUNCEBACKS!

RAPIDRADS MEDICAL EDUCATION

Step Up as an EMRA Leader!

Join the governing force behind EMRA by running for a Board position. As an elected Board member, you'll steer the organization's future while representing your peers in emergency medicine.

2025 Elections

- **Candidacy deadline: July 25** *(Candidates can run from the floor of the meeting as well.)*
- **Election Day: Sept. 8** *(must be present)*

Positions to Be Filled

- **President-elect**
- **Vice Speaker**
- **Board Secretary/EM Resident Editor-in-Chief**
- **Director of Education**

What You Need to Know

TERMS & ELIGIBILITY

- **All positions require a 2-year commitment (3 years for President-elect).**
- **Any current resident or fellow can run and serve their full term—even if you graduate during your time in office.**

ROLES & RESPONSIBILITIES

- **Lead projects and represent emergency medicine at key events, plus actively participate in regular virtual meetings.**
- **Expect a significant travel commitment that complements your residency duties.** *(Travel is funded by EMRA.)*

CAMPAIGN ESSENTIALS

- **Discuss your plans with your program director, chief residents, and your family—support is crucial!**
- **Secure the necessary endorsements and prepare your application:**
  - A concise, 200-word platform statement
  - Your CV and a professional headshot
  - A letter of support from your program director
- **Familiarize yourself with campaign rules.**
- **Attend the Rep Council Fall Meeting, where you'll deliver a 3-minute speech, followed by a position-specific Q&A.**

CRAFT YOUR PLATFORM

- **Choose 2-3 key issues that resonate with EMRA's mission and strategically frame your campaign.**
- **Hone your speech with ample practice and mentor feedback to make a confident, compelling case for your candidacy.**

Are You Our Next National Leader?

Embracing a leadership role on the EMRA Board is a bold step toward shaping the future of emergency medicine. If you're ready to balance your residency with impactful service, now's the time to lead!

- **Talk to current officers to understand the ins and outs of the role firsthand.**
- **Scan the QR code to learn more about EMRA's mission, goals, and available Board roles.**

What's Important to You?

The EMRA Representative Council Fall Meeting takes place Sept. 8, with resolutions due July 25. This means now's the time to start thinking about what's important to you and how you want your professional organization to act on your behalf.

"[RepCo is] the single location in EMRA where ALL of the voices across the organization can be heard," said Speaker Jacob Altholz, MD. "No matter the geographic area or particular interest at hand, RepCo comes together and decides how we as an organization are going to see or advocate on certain issues, with all voices present."

You hear this multiple times per year: *Help set the direction of EMRA. Use your voice. Tell RepCo what you believe and how to advocate*.

EMRA Representative Council 2025 Fall Meeting

SEPT. 8, SALT LAKE CITY

- Each program sends 1 voting representative and 1 alternate councilor.
- All members are encouraged to show up and participate.

RESOLUTION DEADLINE: JULY 25

- **Visit www.emra.org/repco for a resolution formatting template.**
- **Virtual resolution review takes place Aug. 25.**

But does it actually work?

Consider these EMRA actions — all of which started with RepCo:

- **Full, active support of both 3-year and 4-year residency training;**
- **Support for better leave policies surrounding the birth or adoption of a child, or a death in the family;**
- **Advocating for better work schedules and working conditions for pregnant doctors;**
- **Opposition to the Standardized Video Interview (if you don't know what this is, Google it and then thank RepCo).**

"I also like to bring the AMA example: no smoking on airplanes started as a resolution from medical students," said Vice Speaker Ian Brodka, MD. "We can make a change in the world around us for the better."

Policymaking is not difficult, nor does it need to be boring. All it takes is an interest in the world around you.

"My dream 'slate' is a set of resolutions that are informed, cutting edge, and really seek to advocate for changes on issues affecting trainees directly," Dr. Altholz said. "Formatting can change, wording can change, but the identification of the issue and a possible solution is the single most difficult step in resolution writing. The rest can get hashed out."

And like emergency medicine overall, it's a team effort.

"We are happy to help, every step of the way if you reach out to us!" Dr. Brodka said. "Have thoughts about what you think your residency experience should be with rotations and procedures? Tell us with policy! Think EMRA should have more supporting stances on certain hot button topics and there's a gap in our current policies? Draft and submit what you want us to believe! Have opinions about AI use in the ED and think we should take the same stance? By all means, make policy for us to stand by!

"We always love to see involvement with our RepCo and to know what the membership of this organization wants to see from us."

SimWars: The Legend

Gather your team, get your flair (boring scrubs? nope), and come steal the championship at EMRA SimWars 2025.

- **Competition date: Sept. 8**

Fall Medical Student Forum

Get your questions answered by program leaders & faculty, through general sessions and breakouts tailored to your phase of training.

- **Free event**
- **Date: Sept. 6**

MedWAR 2025

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EMRA ECG Challenge

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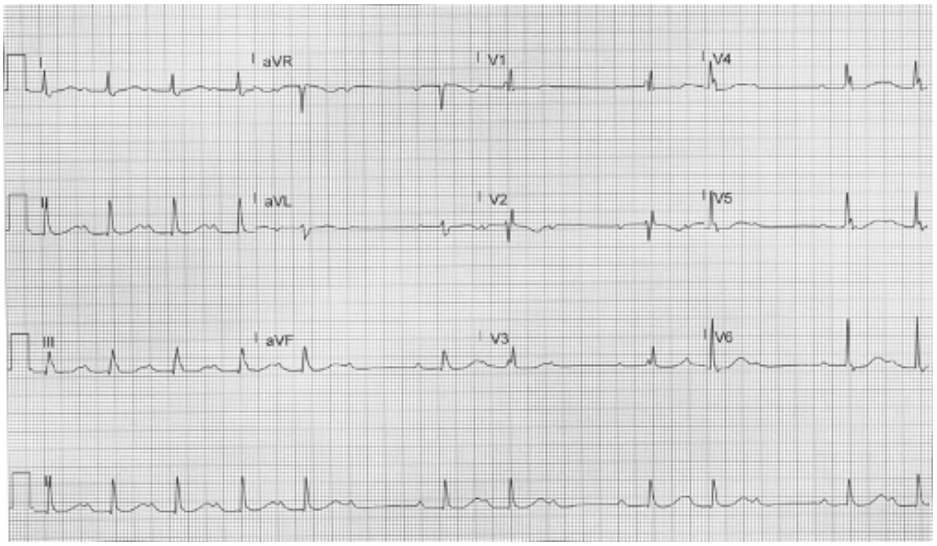
CASE

A 73-year-old female with a past medical history of bovine aortic valve replacement presents due to generalized malaise and fevers.

WHAT IS YOUR INTERPRETATION OF HER ECG?

ANSWER ON PAGE 54



References available online.

ECG Challenge

ANSWER:

This ECG shows a Mobitz type II 2nd degree AV block with an average ventricular rate of 61 bpm, normal axis, prolonged PR interval, and prolonged QRS complex duration with a RBBB.

Mobitz type II AV blocks are due to intermittent failure of conduction between the atria and ventricle, leading to what is often described as a dropped QRS. The conduction block is typically infranodal, but it can occur more proximally in the AV node. The distinguishing feature of a Mobitz type II AV block is a constant PR interval (which may or may not be prolonged), followed by a non-conducted P-wave. The PP and RR intervals will be constant, and the P-waves will march out.

It is important to differentiate Mobitz type I conduction from Mobitz type II conduction, as a Mobitz I is usually transient with a more benign course, while a Mobitz II is associated with worse outcomes if not treated (e.g., hemodynamic compromise, progression to a 3rd degree AV block). The QRS complex duration is typically not useful in differentiating between a Mobitz I and a Mobitz II AV, as a Mobitz I can have a prolonged QRS complex duration if there is aberrant conduction (e.g., an intrinsic bundle branch block) and a Mobitz type II can have a narrow QRS complex (i.e., duration < 110 msec) if the block is in the AV node. The key to distinguishing between the two is to compare the RR interval that includes the non-conducted P-wave to the PP interval. In a Mobitz type I AV block, the RR interval that includes the non-conducted P-wave will be less than twice the PP interval. In a Mobitz type II AV block, the RR interval that includes the non-conducted P-wave will equal exactly twice the PP interval.

This ECG also shows a RBBB. The characteristic findings in a RBBB include:

- **QRS complex duration ≥ 120 msec**
- **rsr', rsR', or rSR' pattern in lead V1 +/- V2**
  - Variations 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)**
- **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**

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. Otherwise, the presence of a RBBB does not confound the ECG evaluation of ACS as does a LBBB.

CASE CONCLUSION

This patient was admitted to the cardiology service and underwent placement of a permanent pacemaker due to intermittent 3rd degree AVB. Blood cultures drawn in the ED returned positive for Gram-positive cocci. A subsequent TEE was negative, but a PET scan noted increased activity at the aortic valve as well as the aortic graft concerning for infection, so the patient was treated with a 6-week course of antibiotics.

MOBITZ TYPE II AV BLOCK LEARNING POINTS

- **Constant PR interval in conducted beats**
  - Described as ratio of P-waves to QRS complexes
  - Typically an infranodal block resulting in a prolonged QRS complex duration
  - RR interval that includes non-conducted P-wave = twice the PP interval
- **Never a normal variant and frequently produces hemodynamic compromise**
  - High risk of progressing to a 3rd degree AV block
  - Atropine is unlikely to lead to clinical improvement and may lead to a high-grade AV block

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 second 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.**

References available online.

Board Review Questions

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1. A 61-year-old woman presents with vertigo that starts every time she moves her head. She has no hearing changes, tinnitus, or significant medical history. Her blood pressure is 178/85; there are no neurologic deficits. Bedside positional testing demonstrates nystagmus with a long latency and transient duration; it is suppressible with repeated testing. What is the most likely diagnosis?

A. Benign paroxysmal positional vertigo
B. Ménière disease
C. Transient ischemic attack
D. Vertebral basilar artery insufficiency

2. A 58-year-old man with known severe coronary artery disease presents via ambulance after a witnessed cardiac arrest. He has been intubated by EMS, and CPR is in progress. His end-tidal carbon dioxide (EtCO2) drops from 20 mm Hg to 9 mm Hg during the course of 10 minutes; the waveform maintains a normal morphology. Bedside echocardiogram shows ventricular fibrillation. What is the most likely cause of the drop in EtCO2?

A. Cardiac tamponade
B. Displacement of endotracheal tube
C. Inadequate chest compressions
D. Internal hemorrhage

3. What is the most significant risk factor for hospital-acquired pneumonia?

A. Chronic lung disease
B. Dialysis
C. Immunocompromise
D. Mechanical ventilation

4. Which aspect of the physical examination can best distinguish toxicity from an anticholinergic agent from that of a sympathomimetic agent?

A. Heart rate
B. Pupils
C. Skin
D. Temperature

5. What factor is associated with a diagnosis of SCIWORA (spinal cord injury without a radiographic abnormality)?

A. Axial loading mechanism
B. CT findings
C. Lumbar spine tenderness
D. Presentation in younger children versus adults

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

References available online.


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