University of Cincinnati Medical Center - Department of Emergency Medicine

Annals of B Pod

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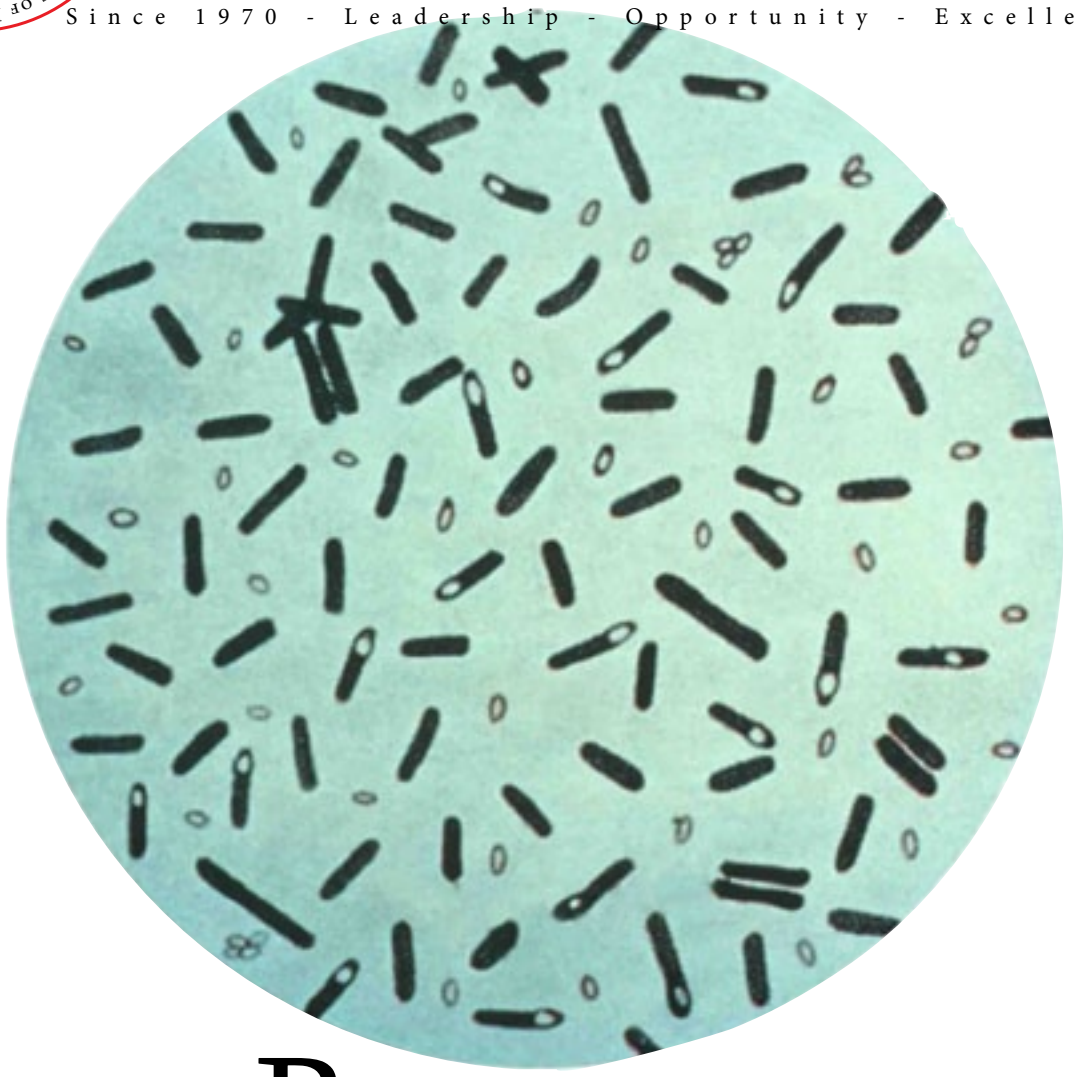

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also available at *tamingthesru.com*

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ANNALS OF B POD
FALL 2014

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Welcome to a new year of Annals of B Pod.

B Pod is a coveted area of our emergency department where first year residents primarily manage and are mentored by fourth year residents.

Annals of B Pod is a quarterly resident produced publication of the University of Cincinnati Department of Emergency Medicine - its articles do not necessarily represent the views or standards of care for the depeartment - remember to always use your clinical judgement.

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Cover photo of Clostridium Botulinum - http://www.earthzine.org/wp-content/uploads/2009/01/micro.jpg

Community Corner

Molly Bister, MD
University of Cincinnati 2014

Dr. Bister graduated in the Class of 2014, was a former editor of Annals of B Pod and currently serves as an attending physician at O'Bleness Hospital in Athens, OH

History of Present Illness

The patient is a pleasant female in her mid-50s with a history of COPD on home O2 (2L) who presents for shortness of breath and chest pain. While she is short of breath at baseline, she states her dyspnea began getting worse two weeks ago, and has been progressively more severe. Her sputum is thicker than usual and tastes different, but she has had no fevers or sick contacts. A few days ago, she began to have sharp chest pain that begins at her xiphoid process and radiates to her left shoulder and left axilla. She rates this pain 9/10 and it is constant. It is exacerbated by movement and relieved with nothing. It has not been specifically related to exertion. She has no personal or family history of DVT/PE and no risk factors aside from a moderately sedentary lifestyle. On review of systems, the patient notes that she feels like her face, neck, and hands have been swollen for the last two days. She states that she has not had any lower extremity swelling recently. The patient's PCP recently closed his practice, and she is out of her anxiolytic medication.

Past Medical History

COPD, anxiety, GERD, hepatitis C, OA, morbid obesity

Social History

Smokes 1 ppd, denies EtOH or illicit drugs

Medications

clonazepam, albuterol, omeprazole, ibuprofen, oxycodone/acetaminophen, "some other inhaler"

Allergies

morphine (itching)

Physical Exam

Temp 97.7
Heart Rate 110
Resp Rate 22
BP 149/101
O2 Sat (2L) 95%

General: middle-aged morbidly obese female in NAD
HEENT: mild facial plethora (if I had not been specifically looking for it, I probably would not have noted anything unusual). Large, short, full neck, full ROM, trachea midline, no LAD, unable to appreciate JVD.
Respiratory: Coarse breath sounds bilaterally; diminished in the bases R>L; no wheezes, rales, or rhonchi. Slightly prolonged expiratory phase.
Cardiac: Tachycardic, distant heart tones, no M/R/G.
Abdomen: Obese, soft, nondistended, nontender, +BSx4.
Extremities: 1+ edema of the bilateral upper extremities, some pitting in the hands. 2+ radial and DP pulses bilaterally. No edema of the lower extremities.
Neuro: Grossly normal.

Labs

12.3		
12.6	378	
39.3		

132	92	8
3.5	33	0.6

100

BNP 15 Troponin I <0.05

EKG: sinus tachycardia, no ischemic changes
Radiology: See Figure 1

Emergency Department Course

The patient had an IV established and was placed on cardiac and pulse ox monitoring. She received hydromorphone and lorazepam with much improvement in her comfort. On discussion with the

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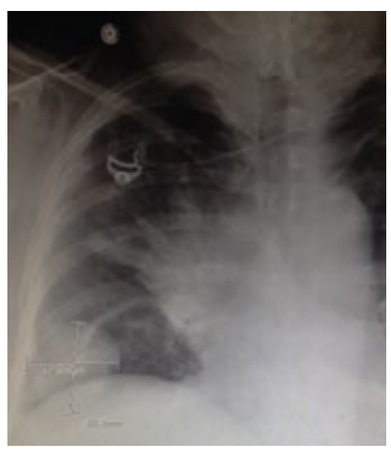

Acute Limb Ischemia

Kristopher Ford, MD
University of Cincinnati R4

case

History of Present Illness

The patient is a 88 year old female with a past medical history significant for ischemic stroke, atrial fibrillation and hypertension who presents to the ED with numbness in her right foot. She reports that she woke up at 4 am and her right foot felt numb. She states she "couldn't feel my foot." She called her home heath nurse and after assessment was sent to the ED. She denies any pain in the foot. She has residual weakness in her left lower extremity from a previous ischemic stroke and states that she feels like she can't move her foot "almost like I'm having another stroke." The patient is on warfarin for atrial fibrillation. She denies any recent falls or trauma. She denies any other symptoms.

Review of Systems

otherwise negative

Past Medical History

hypertension, ischemic stroke with residual left side weakness, atrial fibrillation, hypothyroidism

Social History

denies tobacco, alcohol or illicit drug use

Temp 98.3
Heart Rate 108
Resp Rate 18
BP 186/67
O2 Sat 98%

Physical Exam

General: Well appearing elderly female in no acute distress
HEENT: Atraumatic, PERRL, no scleral icterus, mucous membranes moist with no erythema, uvula midline
Respiratory: Clear to auscultation bilaterally, no rales or wheezing
Cardiac: Irregularly irregular, tachycardic, no murmurs
Abdomen: soft, nontender, nondistended, normal bowel sounds
Extremities: no signs of injury, 2+ radial pulses bilaterally, 2+ left dorsalis pedis pulse, nonpalpable dorsalis pedis and posterior tibialis pulse on right, weak Doppler signal on right dorsalis pedis, right foot cool to touch with delayed capillary refill
Neurologic: Awake and alert, residual paralysis in left leg, able to wiggle toes on right, moves upper extremities with 5/5 strength, decreased sensation to right foot

The 6 Ps of the ischemic limb

Paresthesias
Pallor
Pain
Poikilothermia
Pulselessness
Paralysis

Emergency Department Course

Upon arrival to the ED the patient was hemodynamically stable. She had a cool right foot with no palpable dorsalis pedis or posterior tibialis pulse. A doppler signal was able to be obtained but it was much weaker when compared to the left foot. Vascular surgery was immediately consulted and she was given an unfractionated heparin bolus and started on an infusion. The patient's INR was 1.6. Vascular surgery took patient to the OR immediately where she received an aortogram with bilateral runoff, angioseal of left femoral artery, right femoral artery cut down with open thrombectomy of her right superficial femoral artery/profunda, and instillation of tPA into her right superficial femoral artery. She tolerated the surgery well without complications. She was continued on Coumadin and transferred to a skilled nursing facility on post operative day 3.

discussion

Acute limb ischemia carries a high mortality and morbidity. Prompt recognition and initiation of treatment in the emergency department is paramount. The incidence has been reported as 1.5 cases per 10,000 persons per year. Acute limb ischemia is defined as a sudden decrease in limb perfusion that threatens viability of the affected limb. In contrast to chronic limb ischemia, ischemia is considered acute if the clinical presentation is within 2 weeks of symptom onset. Symptoms include pain, paresthesias, weakness, numbness, skin discoloration. The pneumonic "Six P's" is often used to help identify the presentation of limb ischemia. The six P's include pain, paresthesia, pallor, pulselessness, poikilothermia, and paralysis (Figure 1). Although rarely will all of these findings be present, it underscores the importance of a thorough but focused physical exam as the diagnosis and treatment of acute limb ischemia should be made based on history and physical without the use of imaging. An adequate physical exam includes a careful examination of limbs looking for decreased temperature, pallor and mottled appearance. Sensory and motor exam as well as assessment of femoral, popliteal, posterior tibial and dorsalis pedis pulses should be performed. The most important exam adjunct is the performance of an arterial brachial index (ABI). The ABI allows for the measurement of perfusion pressure with an affected limb and if less than 50 mm Hg represents limb ischemia (Figure 2).

Acute limb ischemia is caused by acute arterial thrombus formation, embolism, dissection or trauma. Emboli usually originate from the heart and are more common in patients with atrial fibrillation, MI leading to left ventricular thrombus forma-

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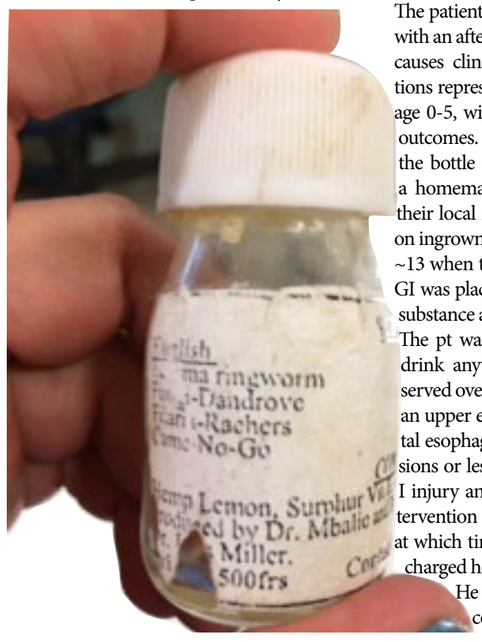
Peds Tox

Home Remedies
Gone Wrong

Riley Grosso, MD
University of Cincinnati R2

A previously healthy 19 month old male presents to CCHMC 3 hours after ingesting "aftershave". His parents think he drank a very small amount and they found him coughing with the open bottle. His mother tried to gag him immediately. He vomited once, it had a small amount of blood in it. He has not vomited since. He has been drooling, refusing to drink, and a little fussy although otherwise acting like himself. He has not had any shortness of breath, diarrhea, or noisy breathing. They went to an outside hospital where a CXR was performed, which was normal. The family was then transferred for evaluation. He has no significant past medical history, he was born full term and is up to date on all of his vaccinations. He takes no medications and has no allergies his parents are aware of.

He is afebrile with otherwise normal vitals for his age and is saturating 100% on room air. He is sitting in his mom's lap drooling in no acute distress. He does not have any trauma to his face or mouth. He is not swallowing his secretions and his soft palate is erythematous with a few erosions on its posterior aspect. Otherwise is oropharynx exam is normal. He is breathing comfortably on room air, without stridor or wheezing. His lungs are clear to auscultation bilaterally. He has a normal cardiac, abdominal, and extremity exam. He is neurologically intact and is acting appropriate for his age. His gait is normal.



Bottle of Cameroon home remedy with a pH of almost 13
Photo by Riley Grosso, MD

> "Oral lesions are not predictive of distal injury, any symptoms that raise suspicion for distal injury should trigger further work-up and endoscopy within 24h. The most sensitive of these symptoms are drooling and dysphagia..."

The patient's physical exam was not consistent with an aftershave ingestion, which usually just causes clinical intoxication. Cosmetic ingestions represent >13% of ingestions in children age 0-5, with almost all of these with benign outcomes. The parents were able to produce the bottle of "aftershave" which was actually a homemade liquid made in Cameroon by their local medicine man that his father used on ingrown hairs. The pH of the substance was ~13 when tested with pH paper. A consult to GI was placed given the alkaline nature of the substance and the child's symptoms.

The pt was admitted to GI after refusing to drink anything in the ED and he was observed overnight and then taken to the OR for an upper endoscopy in the morning. His distal esophagus showed erythema without erosions or lesions, which is considered a Grade I injury and generally there is no further intervention needed. He was observed for 48h at which time he was taking PO and was discharged home.

He has had no interval development of complications at 6wk f/u with GI.

Common alkaline substances ingested by children include drain cleaners, hair relaxers, household cleaners, and dishwasher detergents. Unlike their acidic counterparts, these liquids tend to be tasteless, so children do not cough and gag on them in the same way they do acidic substances. Although this does cause less aspiration and subsequent airway injury, they tend to ingest larger amounts of alkaline substances. The pattern of injury is multifactorial; it is dependent on the pH, with pH >10 causing more injury, the amount ingested, and the make-up of the substance ingested. While liquids tend to cause distal esophageal and gastric injury, as in our patient, granular substances such as dishwasher detergent tends to stick to the upper airway and is often inhaled.[2] Oral lesions are not predictive of distal injury, and any symptoms that raise suspicion for distal injury should trigger further work-up and endoscopy within 24h. The most sensitive of these symptoms are drooling and dysphagia, but providers should also be concerned if patients have vomiting, stridor, odynophagia, or abdominal pain. Do not attempt to neutralize the substance, induce vomiting, or dilute with water or milk. The NG should be only be place under endoscopy, as these patients are at high risk for perforation at sites of injury with blind NG placement.[3]

Alkaline ingestions cause liquefactive necrosis with saponification of fats and denaturation of proteins and even blood vessel thrombosis. The severity of injury on endoscopy is graded from 0, which is no evidence of injury at all, to IIIb, which is extensive necrosis and ultimately 100% of these patients have stricture formation. The patient above had Grade I injury, which is just edema or hyperemia of the mucosa, and subsequently had little chance of developing sequela. Once the injury severity is over Grade IIb, there is >70% chance of stricture formation.[2] Strictures are the major long-term GI complication from alkaline ingestion and eventually require dilation and stent placement, although not usually in the immediate post-ingestion period. The risk of developing an upper GI malignancy later in life after alkaline ingestion is approximately 1000x that of the non-ingestion population, although no screening is currently recommended for these patients.[2]

Management of the acute alkaline ingestion

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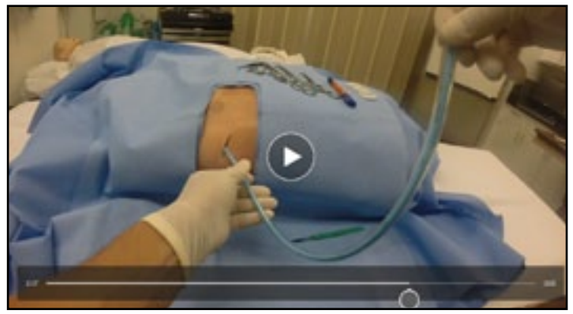
Quick Hit Procedures
Bougie-assisted Chest Tube

Ryan LaFollette, MD
University of Cincinnati R3



See the video online at tamingthesru.com

Problem:
Chest wall thickness / edema can make it difficult to maintain a subcutaneous tract to the pleural surface

Solution:
Using Seldinger technique, a bougie can maintain the track, encourage proper chest tube positioning, and minimize pleural violations making you look like a pro

1. Collect
Chest tube kit
Drape kit
Atrium
Chest tube (any greater than 24F will do)
Betadine
1% lidocaine w/epi
4x4 gauze
2 x large tegaderm
Sterile gloves
2-0 silk suture
Gum-elastic bougie
Marking pen

2. Prep
Drape and prep the patient

Inject lidocaine w/epi
make a superficial wheel, then deep into rib space (be sure to cover the periostium and consider blocking adjacent ribs as well)

Mark the anticipated depth on the bougie, clamp and cut chest tub and pre-load bougie

3. Cut
Make skin incision

Bluntly dissect to rib with kelly forceps

Pucture parietal pleura over rib

Ensure proper location by palpating pleura and lung

4. Bougie
With finger in pleural space, slide bougie along finger and direct couday tip cephalad

Stabilize bougie and slide chest tube over until adequate depth

Remove bougie

Attach atrium

Secure tube

B Pod Case

Botulism
and how to find it

Nicholas Ludmer, MD
University of Cincinnati R1
Phil Moschella, MD
University of Cincinnati R4

CC: Difficulty Swallowing

HPI: The patient is a 22 year old female with no significant past medical history who presents to the ED with a chief complaint of dysphagia. The patient first noticed difficulty swallowing solid foods 2 weeks ago. She states that she felt like food was getting caught in her throat. Initially she only had difficulty swallowing solid foods and was able to eat soft foods and liquids. However, she reports that over the course of two weeks her condition gradually worsened to the point where she could no longer tolerate fluids. She states that she has pain in the back of her throat when she attempts to swallow. She denies any fevers, chills, congestion, swollen lymph nodes or neck stiffness. She reports that she has been to 3 different emergency departments and urgent care centers in which she was treated empirically for strep throat. The patient states that she has felt more fatigued than usual. Upon inquiring about any possible sick contacts, her friend reports that the patient's roommate is in the ICU for "some autoimmune disorder," but did not know any specifics. She denies any past medical history, takes no medications, denies allergies, and denies any tobacco, alcohol, or illicit drug use.

> "The patient's roommate is in the ICU for "some autoimmune disorder," but did not know any specifics

Pertinent Physical Exam:
Vitals: T: 98.3F HR: 95 RR: 16 BP: 115/70 O2: 99% RA
HEENT: Normocephalic, Atraumatic. Patient had appreciable bilateral ptosis. Examination of the patient's eyes revealed pupils that were equal and reactive to light. Her extra ocular muscles were intact, though she had limited lateral abduction of her eyes bilaterally. Examination of the oropharynx revealed large tonsils without exudate or erythema. The palate was symmetrical and uvula midline. She exhibited no trismus. No cervical lymphadenopathy was appreciated.
Pulmonary: Clear to auscultation bilaterally, no wheezes, rhonchi, rhales
Abdomen: Normal bowel sounds, soft, non-tender, non-distended. No rebound or guarding
Neuro: AAOx4, On CN exam she has bilateral lateral gaze palsy and diplopia, otherwise CN intact. Sensation to light touch was in tact throughout. Strength was

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#corecontent

NSTEMI
more than just demand ischemia

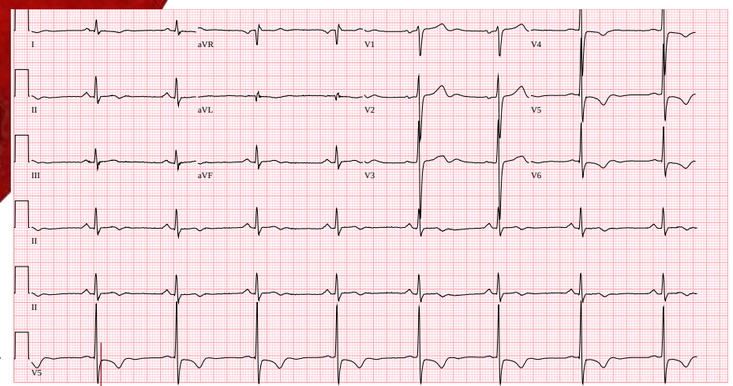
Riley Grosso, MD
University of Cincinnati R2

case

A 3 year old male with a medical history of hypertension and Type-II diabetes presents with 3 days of chest pain and shortness of breath. The patient reports that the pain began when he was laying in bed and describes it as a tightness around the left side of his chest. He denies any radiation of the pain. It improved from a 7/10 to a 5/10 with sublingual nitroglycerin given by EMS providers. He also has had shortness of breath and vomiting but denies fevers, cough, abdominal pain, numbness, or back pain. He is on medication for hypertension, diabetes, and takes an aspirin daily. His physical exam reveals an elderly gentleman in no acute distress who is afebrile, mildly bradycardic with a heart rate of 58, mildly hypertensive to 152/87, breathing 18 times per minute and saturating 97% on room air. He has somewhat diminished pulses in both legs but his exam is otherwise unremarkable. Notably he has no S3, his lungs are clear, and he has no leg edema.

The patient's EKG shows sinus bradycardia with a rate of 49, biphasic T waves in V1-V3, V5-V6, T wave inversions in V4-V6 without Q waves. His first troponin came back at 23.5, and the patient was placed on a heparin infusion with bolus with a planned admission to cardiology for NSTEMI. However, the interventional cardiologist was contacted given his markedly elevated troponin and ongoing chest pain. A bedside echo was performed which demonstrated severe lateral wall hypokinesis. A repeat troponin was >30 and the patient was continuing to have chest pain, so the decision was made to activate the cath lab.

The patient's angiogram revealed 99% stenosis of the left circumflex artery, for which PCI was performed. Interestingly, severe right illiofemoral arterial disease was also discovered and a stent was placed. The patient developed a large hematoma after catheterization, which required direct pressure and transfusion of 1 unit of pRBC. The patient otherwise did well post-catheterization and was discharged 4 days later in good condition.



right: initial patient EKG
left: graphic by Nickolas Raymon[1]

discussion

Chest pain is the second most common chief complaint in US emergency departments. Up to 15% of chest pain visits are attributable to acute coronary syndrome (ACS). Traditionally ACS incorporates three different diagnoses that ultimately require different treatment algorithms. The first and most urgent of these diagnoses is ST-elevation myocardial infarctions (STEMI). Patients with diagnosis of STEMI should be considered for reperfusion therapy immediately, most often accomplished by emergent consultation with interventional cardiologists and/or directly activating the cath lab if applicable. The

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Depakote Ingestion
check your sprinkles

Kari Gorder Shewakramani, MD *(author)*
University of Cincinnati R1
Kris Ford, MD
University of Cincinnati R4

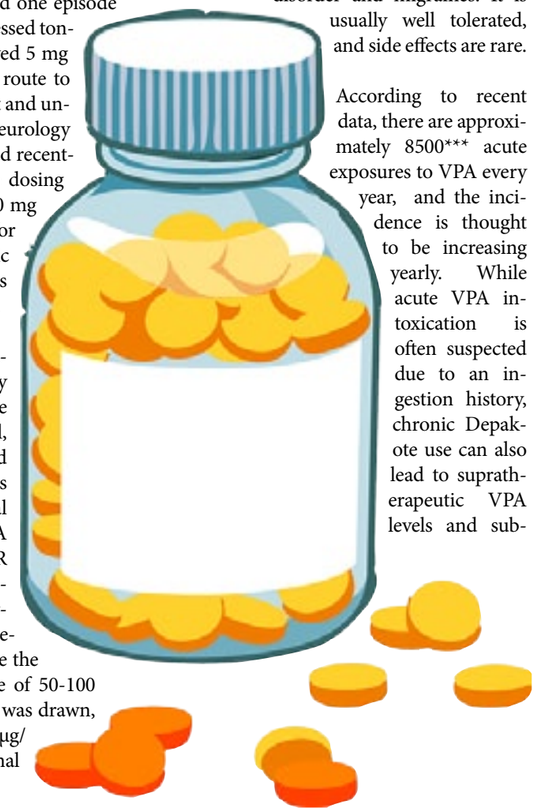
case

A 60-year-old male with a history of epilepsy on Depakote presented to B pod via EMS with reports of a seizure. Per EMS, the patient's wife reported that the patient had been not feeling well over the last 24 hours, with increased lethargy and one episode of vomiting prior to a witnessed tonic-clonic seizure. He received 5 mg of IM Versed via EMS en route to the ED, and was somnolent and unarousable upon arrival. Neurology records revealed that he had recently increased his Depakote dosing from 1000 mg BID to 1500 mg qAM and 1000 mg qHS for persistently subtherapeutic valproic acid (VPA) levels and breakthrough seizures.

On physical exam, the patient was hemodynamically stable and afebrile. While he initially appeared postictal, he remained somnolent and arousable only to vigorous physical stimuli for several hours while in the ED. A head CT was normal, CXR unrevealing and an electrolyte panel was within normal limits. His VPA level returned at 205 µg/mL, above the standard therapeutic range of 50-100 µg/mL. An ammonia level was drawn, and was found to be 112 µg/dL. LFTs were within normal limits.

The patient remained somnolent. After consultation with poison control, due elevated ammonia and supratherapeutic levels of VPA, the decision was made to start an infusion of L-carnitine. During his hospital stay, his mental status improved, and his ammonia level decreased to 37µg/dL. He was discharged in stable condition on hospital day 1 with neurology follow-up.

Valproic acid (VPA, also known by its trade name Depakote) is an anti-epileptic medication used to treat both generalized and partial seizure disorders, as well as bipolar disorder and migraines. It is usually well tolerated, and side effects are rare.



According to recent data, there are approximately 8500*** acute exposures to VPA every year, and the incidence is thought to be increasing yearly. While acute VPA intoxication is often suspected due to an ingestion history, chronic Depakote use can also lead to supratherapeutic VPA levels and subsequent side effects. The therapeutic dosing range of VPA is typically 50-100 µg/mL, and symptoms of intoxication are usually seen above 180 µg/mL. Common symptoms of VPA intoxication include CNS depression, ranging from mild sleepiness and lethargy to respiratory depression and coma. Seizure patients may also experience increased frequency of seizures. Patients may also experience hemodynamic instability, including tachycardia and hypotension, as well as GI side effects, such as nausea, vomiting and diarrhea. Unique to VPA is its metabolic effects on the liver, with hyperammonemia and transaminitis often being seen in patients with both therapeutic and supratherapeutic VPA levels; up to 50% of patients on VPA may have transient elevation of their ammonia levels, with or without abnormal LFTs. This is thought to be due to VPA's effects on an enzyme involved in the urea cycle. As a result, VPA-induced hyperammonemic encephalopathy (VHE) and VPA-induced hepatoxoicity (VHA) are rare but potentially fatal consequences of VPA intoxication.

discussion

In a patient with supratherapeutic Depakote levels who presents with CNS depression or other concerning symptoms, an ammonia level and LFTs should be checked. Should either of these values be significantly elevated, in addition to stopping Depakote, aggressive hydration and providing supportive care, L-carnitine infusion may be initiated in the ED.[1] As valproic acid is partially metabolized by mitochondrial beta-oxidation, the initial treatment for both VHE and VHA involves the use of L-carnitine, an amino acid derivative and co-factor in the mitochondrial long-chain fatty acid metabolism cycle found to be necessary for the proper breakdown of VPA. VPA use itself has been found to directly deplete serum levels of carnitine. Additionally, patients with dietary carnitine deficiency, inborn errors of metabolism, cirrhosis or other metabolic disorders are at increased risk for VPA intoxication due to lower intrinsic levels of carnitine and would benefit from treatment. Carnitine supplementation or infusion has been found to assist in the correction of hyperammonemia in these patients and aid in rapid neurologic improvement. While there is no agreed-upon VPA or ammonia level at which to start L-car-

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Pharm Consult

Pulmonary Embolism

Alteplase Dosing Guidelines

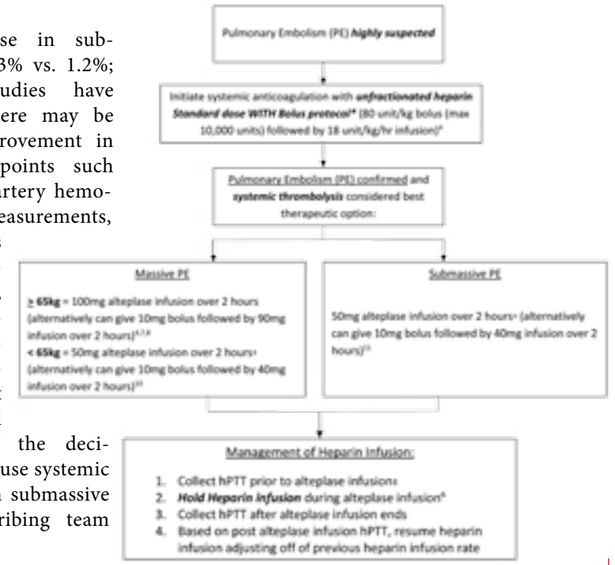
Alyssa Penick, PharmD; Jessica Winter, PharmD, BCPS; Nicole Harger, PharmD, BCPS
University of Cincinnati Critical Care Pharmacy

Pulmonary embolism (PE) is associated with acute and chronic risk including 30% mortality in untreated PE and development of right ventricular dysfunction in 60-70 per 100,000 new cases of PE per year.[1,2] Timely diagnosis and safe treatment is imperative upon presentation to the Emergency Department. Systemic anticoagulation is the mainstay of treatment. Adjunctive therapies such as systemic thrombolysis, catheter directed therapy, and embolectomy are also options. This article focuses on the dosing of systemic alteplase in massive and submassive PE.

Systemic thrombolysis in PE

Investigators have studied varying doses of different thrombolytics, including alteplase, tenecteplase, and streptokinase. The United States Federal Drug Association (USA FDA) currently has approved the use of alteplase for the indication of acute pulmonary embolism. Current guidelines recommend systemic thrombolytic therapy for patients with massive (high risk) PE in the absence of contraindications (Table 1). Thrombolytic treatment has been associated with significant reduction of PE-related mortality (OR: 0.29; 95%; CI: 0.14-0.60, P<0.001) in a 2014 meta-analysis including massive and submassive PE. However, there was also increased risk of major bleeding associated with thrombolytic therapy (OR: 2.91; 95% CI: 1.95-4.36, P<0.0001). In massive PE, where hemodynamic compromise is of greatest concern, systemic thrombolysis is endorsed by the American College of Chest Physicians (ACCP), American Heart Association (AHA), and the European Society of Cardiology (ESC), unless any major contraindications to thrombolysis exist.[4,7,8]

Thrombolytic use in submassive (intermediate risk) PE is only recommended on an individual risk-to-benefit analysis, as there has been no proven benefit on mortality with increased risk of hemorrhage in a prospective trial evaluating tenecteplase use in submassive PE (6.3% vs. 1.2%; p<0.001)9. Studies have shown that there may be associated improvement in morbidity endpoints such as pulmonary artery hemodynamic measurements, arteriovenous oxygen, pulmonary perfusion, and echocardiographic assessment (improved right ventricular wall movement).[7] If the decision is made to use systemic thrombolysis in submassive PE, the prescribing team must carefully weigh the risk of bleed with the potential benefits of systemic thrombolysis.



* In the case of heparin allergy or history of heparin induced thrombocytopenia (HIT) Recommend Argatroban Weight Based Protocol as alternative anticoagulant
† Can consider additional alteplase 50mg infused over 2 hours if clinical response not optimal to first dose (maximum 100mg alteplase to be given)
‡ Consider holding alteplase infusion if pre-thrombolytic hPTT> 130 and hemodynamically stable. If hemodynamically unstable/shock recommend NOT delaying thrombolytic for supratherapeutic hPTT. Collect hPTT per UC Health Lab Draw Policy

Dose of Systemic Alteplase in PE

USA FDA approved dosing for treatment of acute PE is **100mg infusion over 2 hours** (±10% bolus). However, recent studies have investigated the utility of smaller doses in patients with decreased weight as well as decreased clot burden (submassive PE). Wang, et al[11] studied 100mg vs 50mg alteplase in patients with masive PE. They found no difference in efficacy out-

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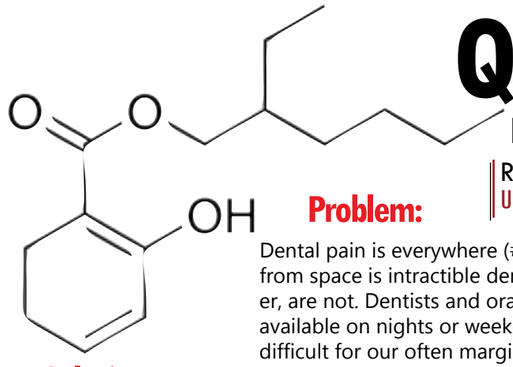
Contraindications	Warnings
Active internal bleeding	Age > 75 years
History of cerebrovascular accident	Current use of anticoagulation, e.g. vitamin K antagonists, direct thrombin inhibitors, Xa inhibitors
Recent intracranial or intraspinal surgery or trauma	Pregnancy
Known bleeding diathesis	Recent trauma
Severe uncontrolled hypertension	Traumatic or prolonged cardiopulmonary resuscitation
Intracranial neoplasm, arteriovenous malformation, or aneurysm	Recent internal bleeding (1 to 2 weeks)
	Recent gastrointestinal or genitourinary bleeding
	Chronic, severe, and poorly controlled hypertension (SBP > 175 mmHg or DBP > 110 mmHg)
	Recent major surgery, e.g. coronary artery bypass graft, obstetrical delivery, organ biopsy, previous puncture of noncompressible vessels
	Cerebrovascular disease
	High likelihood of left heart thrombosis, e.g. mitral stenosis with atrial fibrillation
	Acute pericarditis
	Subacute bacterial endocarditis
	Hemostatic defects including those secondary to severe hepatic or renal disease
	Septic thrombophlebitis or occluded AV cannula at seriously infected site

Table 1:[3] Alteplase Contraindications and Warnings in PE[6]



Quick Hit Procedures

Dermabond for Dental Fx

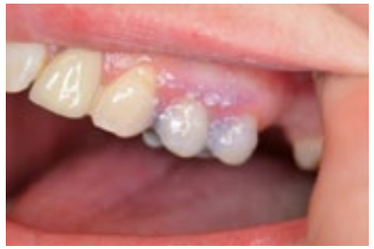
Ryan LaFollette, MD
University of Cincinnati R3

Problem: Dental pain is everywhere (#1 reason of need to return from space is intractible dental pain[1]). Dentists, however, are not. Dentists and oral surgeons are not readily available on nights or weekends, and access is more difficult for our often marginalized patient population.

Solution: 2-octyl cyanoacrylate (Dermabond)

case

32 year old otherwise healthy female with a history of a filling in teeth 14 and 15 (left maxillary first and second molars) presents with tooth pain which started that day after eating dinner. Since that time, she has had significant sensitivity to air, hot, and cold, and has not been able to work due to the pain. The earliest she could get an appointment with her dentist was in 48 hours. On exam she is hemodynamically stable, tearful, with an un-roofed filling visible on tooth 14 without surrounding erythema or evidence of abscess or fracture.



Uses
- Dental caries
- Acute dental fracture
- Displaced filling
- Superficial, hemostatic intra-oral laceration

discussion

What is 2-octyl cyanoacrylate?

A monomer which rapidly polymerizes when exposed to air and/or fluid and creates and hemostatic and bacteriostatic film. Increasingly popular as an alternative for laceration repair, it also has several uses in oral surgery and periodontics for temporary repair

How does it work?

Fractures, dental caries and displaced fillings all cause a 'toothache' by the common pathway of pulp exposure through erosion of enamel and dentin.
Pain is caused by nociceptor activation when either external stimuli (hot, cold, air) causing fluid movement through patent tubules (~2 microns wide) which travel from pulp to oral cavity.
In case of fracture, they are activated by direct exposure of the nerve to the oral environment

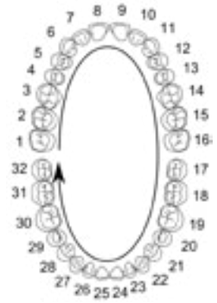


Diagram of adult dental numbering system[3]

Will it it melt gums?

No evidence of adverse outcomes (root necrosis, lack of follow-up) in the literature, although large databases are lacking. In tagged rat models it appears to be excreted in urine and stool and not retained in tissue.[2]

1 - Integrated Medical Model. Dental Conditions. Johnson Space Center. Accessed 2014 Sept.
2 - Leggat PA, Kedjarune U, Smith DR. Toxicity of cyanoacrylate adhesives and their occupational impacts for dental staff. Industrial Health.
3 - "Universal Numbering System" by Kaligula - Own work (based on Human dental arches.svg)

Depakote Tox

Continued from page 7

nitine administration, the Central Ohio Poison Control center recommends administration of L-carnitine with ammonia levels greater than 100 µg/dL and exam findings consistent with CNS depression. The recommended dosing of L-carnitine is an initial bolus of 100 mg/kg IV, followed by q4 hour maintenance dosing at 15 mg/kg IV until ammonia levels return to the normal range and neurologic status improves. VPA levels should be checked every 2-4 hours until they approach the therapeutic range.

In summary, acute or chronic supratherapeutic levels of VPA may cause a spectrum of symptoms that present in the ED, including severe CNS depression. In addition to supportive care, L-carnitine is a safe and likely helpful medication to begin in the ED for patients with findings of encephalopathy or hyperammonemia.

1 - Spiller HA, Krenzelok EP, Klein-Schwartz W, et al. Multicenter case series of valproic acid ingestion: serum concentrations and toxicity. J Toxicol Clin Toxicol. 2000;38(7):755-60.
2 - Perrott J, Murphy NG, Zed PJ. L-carnitine for acute valproic acid overdose: a systematic review of published cases. Ann Pharmacother. 2010; 44(7-8); 1287-93.
3 - Coulter DL, Allen RJ. Secondary hyperammonemia: a possible mechanism for valproate encephalopathy. Lancet. 1980; 1:1310–1311
4 - Lheureux, PE, Penaloza, A and Zahir, S. Science review: Carnitine in the treatment of valproic-acid induced toxicity—what is the evidence? Crit Care. 2005; 9 (5); 431-440
5 - Lheureux PE, Hantson P. Carnitine in the treatment of valproic acid-induced toxicity. Clin Toxicol. 2009; 47 (2); 101-111

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Visual Dx

Continued from back cover

Sialolith

Sialoliths most often affect the Submandibular glands (80-90%) with the bulk of the remaining affecting the parotid glands. As in this patient, submandibular stones often migrate to and obstruct Wharton's duct. While the exact cause of stone formation is not known, it is believed that stagnation of calcium rich saliva causes development of stones. This patient was prescribed sialogogues (Lemon Drops), NSAIDs, and salivary massages. ENT recommended surgical removal although lithotripsy is an option in stones less than 7mm.

Acute Limb Ischemia

Continued from page 3

tion, or patients with prosthetic valves who are not on anti-coagulation. Acute thrombus formation of a lower extremity artery usually originates at the site of an atherosclerotic plaque. Thrombosis can also occur in arterial aneurysms (most commonly popliteal), and in bypass grafts. Thus patients with atrial fibrillation, recent acute MI, known atherosclerosis, previous lower extremity bypass grafts are at increased risk for acute limb ischemia.

The classification of acute limb ischemia is based on diagnostic physical exam findings and prognostic limb viability (Table 1). Stage 1 has no sensory loss or muscle weakness, audible venous and arterial doppler, and has no immediate threat to limb viability. Stage 2a has minimal to no sensory loss, no muscle weakness, often inaudible arterial Doppler, audible venous Doppler, and represents a marginally threatened limb which is usually salvageable if treated promptly. Stage 2b involves sensory loss usually involved with rest pain, mild to moderate muscle weakness, usually inaudible arterial doppler and audible venous doppler, and represents an immediately threatened limb which may be saved with immediate revascularization. Stage 3 involves profound sensory loss, paralysis, inaudible arterial and venous doppler, and represents an irreversibly damaged limb with inevitable permanent damage.

Grading Ischemic Limbs

stage	category	sensory	strength	doppler
1	Viable	intact	intact	arterial and venous
2a	Marginally Threatened	minimal to intact	intact	arterial
2b	Immediately Threatened	lost	weak	venous
3	Irreversibly Damaged	lost	lost	none

Table 1 - Grading system for ischemic limbs by signs and symptoms

The management of acute limb ischemia is directly related to the stage of ischemia. Imaging modalities including duplex ultrasonography, computed tomographic angiography and magnetic resonance angiography can be considered for stage 1 and stage 2a. In all cases, once acute limb ischemia is diagnosed based on history and exam, vascular surgery should be consulted and a heparin bolus and infusion should be initiated. These are recommendations from the 2012 American College of Chest Physicians guideline on antithrombotic therapy for peripheral artery disease and the 2007 Inter-Society Consensus for the Management of Peripheral Artery Disease. Imaging should not delay the initiation of heparin. Subsequent treatment depends on stage of the acute ischemia and the preference of the vascular surgeons. This involves endovascular or open surgical revascularization. Endovascular revascularization involves catheter directed thrombolysis with plasminogen activators such as alteplase. Surgery involves thromboembolectomy with balloon catheters, bypass surgery, and other adjunct techniques including angioplasty, intraoperative thrombolysis and endarterectomy. A meta-analysis of randomized trials comparing endovascular vs surgical revascularization found similar limb savage rates but slightly more complications with

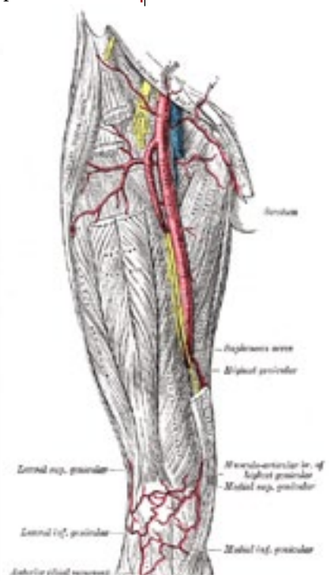
Continued on page 11

How to perform an ankle brachial index measurement

$$ABI = \frac{\text{Highest pressure in ankle of ipsilateral limb}}{\text{Highest pressure of both arms}}$$

ABI is the systolic pressure of the ankle divided by the systolic pressure of the arm. Place an appropriately sized BP cuff on the arm as if taking a normal blood pressure. Place a doppler probe in the antecubital fossa with ultrasound gel. Inflate the BP cuff to at least 20 mm hg higher than expected systolic pressure until you no longer hear a doppler signal. Slowly deflate the cuff by 1 mm Hg at a time until you hear a doppler signal. That represents the brachial artery systolic pressure. Repeat with the other arm. Take an appropriately sized BP cuff and place immediately proximal to the ipsilateral malleoli. Find the Doppler signal with ultrasound gel of the dorsalis pedis artery. Inflate the BP cuff until you can no longer hear the signal. Just as with the arms, deflate by 1 mm Hg until you can hear the signal again. Repeat this process for the posterior tibialis artery. The highest value of the two represents your ankle systolic pressure.

Figure 2



Great vessels of the right leg[5]

Quick Hit Visual Dx

J'mir Cousar, MD
University of Cincinnati R4

A 59 year old gentleman presenting with one day of sharp right-sided chest pain that radiates toward his back. There is no associated shortness of breath or nausea. However he does report the appearance of a rash.

Dermatologic findings included a vesicular rash located along the T3-4 dermatomes on the right posterior and anterior chest wall.

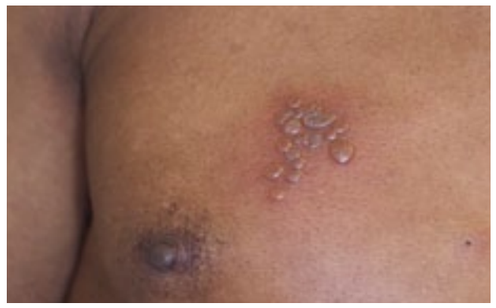
Course: The patient was diagnosed with herpes zoster. He was discharged with acyclovir, prednisone, and oxycodone-apap.

discussion

Varicella-zoster virus (VZV) is the causative agent for herpes zoster (shingles) as well as varicella infection. Herpes zoster appears when the immune response against VZV weakens usually secondary to advanced age. Increased risk for infection occurs among individuals with iatrogenic immune suppression, HIV, and organ transplantation. Lifetime incidence is estimated to be 10-20% with over 95% of adults having serologic evidence of VZV infection.

Classic clinical features include a prodrome of headache, malaise, and photophobia. This is followed by puritis, paraesthesia and dermatome associated pain before appearance of a rash that does not cross the midline. Differential diagnosis includes small pox, cellulitis, contact dermatitis, and measles.

Diagnosis is made clinically. In atypical or severe presentations of disease, viral culture, antigen testing, or PCR testing of vesical fluid can be confirmatory. To reduce the severity of postherpectic neuralgia and reduce the risk of severe disseminated disease in immunocompromised patients, antiviral medication should be started within 72 hours of rash onset. Consider hospital admission in patients with disseminated disease, CNS involvement, and severe immunocompromise. Patients are considered contagious until all lesions have crusted over.



Anterior chest showing vesicular lesions classic for varicella
Photos by J' mir Cousar

Takhar, SS, Moran, JJ. Ch 148 Disseminated Viral infection in Tintinalli' s Emergency Medicine, 7th ed, McGraw-Hill, USA, 2011.

endovascular techniques. However, consensus does not exist in this regard as subsequent studies have not reproduced the higher complication rate of endovascular therapy in patients with acute ischemia. Based on the evidence available, endovascular revascularization is usually preferred for stage 1 and 2a with surgical revascularization being reserved for stage 2b. Stage 3 often requires surgical amputation.

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2 - Norgren, L et al., Inter-Society Consensus for the Management of Peripheral Arterial Disease, Journal of Vascular Surgery, 2007.
3 - Hirsch, AT et al., ACC/AHA 2005 Practice Guidelines for the Management of Patients with peripheral arterial disease: a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and theACC/AHA Task Force for Practice Guidelines. Circulation, 2006.
4 - Uptodate.com/acute limb ischemia
5 - "Gray550" . Licensed under Public domain via Wikimedia Commons - http://commons.wikimedia.org/wiki/File:Gray550.png#mediaviewer/File:Gray550.png

NSTEMI

Continued from page 6

next two diagnoses that make up ACS presentations are differentiated by the presence or absence of biomarker elevation accompanying the patient's presentation. Non-ST Elevation myocardial infarctions (NSTEMIs) are episodes of cardiac ischemia that do not result in ST-elevation on EKG but are severe enough to cause release of detectable quantities of markers of myocardial injury. The most frequently used biomarkers are troponin and CK-MB. Unstable angina (UA) is an episode of cardiac ischemia that is not severe enough to result in either ST-elevation on EKG or detectable troponins or CK-MB.
Our patient's presentation falls firmly into the NSTEMI category of ACS, and initially our patient was being managed under what the American College of Cardiology refers to as the conservative therapy guidelines. These include administration of both anti-platelet and anti-coagulation therapies in the emergency department and admission for monitoring for high-risk events including arrhythmias and ongoing chest pain. These high-risk events would trigger diagnostic angiography and interventions as indicated by the results of this test, just as a positive result of provocative testing does.[3] There is evidence that early invasive therapy, which would include early angiography along a similar timeline to patients presenting with STEMIs, results in a 25% decrease in 2 year mortality and a 17% decrease in non-fatal MIs at 2 years. Given the level of our patient's elevated troponin, the option of early invasive therapy was raised. There are no accepted troponin cutoffs in the literature for the initiation of invasive therapy in NSTEMI, however our patient's bedside ECHO showing localized wall motion abnormalities which is considered a high-risk feature of NSTEMI and therefore an accepted reason to initiate early invasive therapy.[3] Consider consulting interventional cardiology for patients with NSTEMIs that exhibit high risk features, including arrhythmias or new-onset heart failure symptoms, as a new EF <40% is also an indication for early invasive therapy.[3]

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2 - Hollander JE, Diercks DB. Chapatienter 53. Acute Coronary Syndromes: Acute Myocardial Infarction and Unstable Angina. In: Tintinalli JE, Stapczynski J, Ma O, Cline DM, Cydulka RK, Meckler GD, T. eds. Tintinalli' s Emergency Medicine: A Comprehensive Study Guide, 7e. New York, NY: McGraw-Hill; 2011.
3 - R. Scott Wright, Jeffrey L. Anderson, Cynthia D. Adams, Charles R. Bridges, Donald E. Casey Jr, Steven M. Ettinger, Francis M. Fesmire, Theodore G. Ganiats, Hani Jneid, A. Michael Lincoff, Eric D. Peterson, George J. Philippides, Pierre Theroux, Nanette K. Wenger, James Patrick Zidar, 2011 ACCF/AHA Focused Update Incorporated Into the ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, Journal of the American College of Cardiology, Volume 57, Issue 19, 10 May 2011, Pages e215-e367, ISSN 0735-1097
4 - Anthony A. Bavry, Dharam J. Kumbhani, Andrew N. Rassi, Deepak L. Bhatt, Arman T. Askari, Benefit of Early Invasive Therapy in Acute Coronary Syndromes: A Meta-Analysis of Contemporary Randomized Clinical Trials, Journal of the American College of Cardiology, Volume 48, Issue 7, 3 October 2006, Pages 1319-1325, ISSN 0735-1097

Wilderness Medicine

The Intersection of Climate Change and Human Health

Renee Salas, MD, MS
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Renee graduated from University of Cincinnati Emergency Medicine Residency in 2013. She is in her second year of wilderness medicine fellowship and completing an MPH in Environmental Health at the Harvard School of Public Health. She will then be staying on as faculty in the Department of Emergency Medicine / Division of Wilderness Medicine at Massachusetts General Hospital / Harvard Medical School to continue her pursuits in academic wilderness medicine and work in the intersection of climate change and human health.

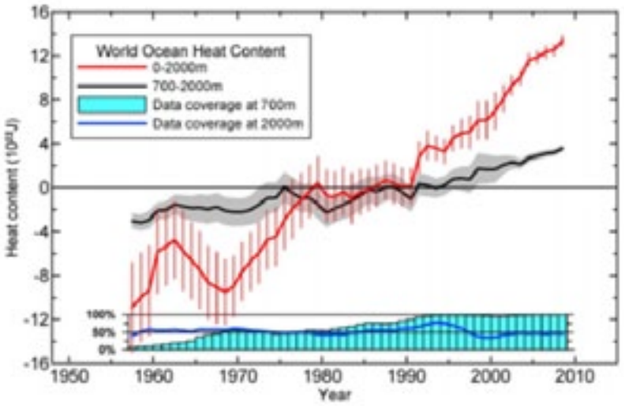

Figure 1: Time series for world ocean heat content.

What does the Lancet call the "biggest global health threat of the 21st century"? You may be surprised to learn that it is a topic which very few physicians currently learn about during their training – climate change.[1] While there are still the ever shrinking collection of skeptics who state that climate change is not occurring, there is no lack of consensus among scientists. In fact, the Intergovernmental Panel of Climate Change (IPCC), which first released its reports in 1988, currently have thousands of authors and is possibly the "largest scientific assessment exercise in human history."[2,3] The recent press that global warming has slowed below previously proposed predictions is due to the fact that the oceans are currently acting as a heat sink with potentially catastrophic warming of the world's oceans as a result (Figure 1).[4] Thus, climate change is a reality which has, and will continue to, change the environment in which we live.

The effects of climate change on human health are numerous. The World Health Organization estimates that between 2030 and 2050, nearly 250,000 additional deaths per year will occur due to just four of the effects of climate change on human health – malnutrition, malaria, diarrhea, and heat stress.[5] Aaron Bernstein, MD, MPH at the Harvard School of Public Health has created two diagrams which further exhibit some of the key effects on human health (Figure 2). While individuals may be able to predict some of the health effects, others are likely surprising. For example, increased ambient CO2 decreases the nutrients of crops such as wheat, rice, and maize which can lead to malnutrition.

Our efforts should be most directed at those populations which will be most vulnerable. There is a the stark contrast between the countries which have most contributed to CO2 emissions and those which will suffer the worst of climate change health consequences. Thus, it is imperative that physicians in all countries, but especially those with abundant resources, begin to understand their possible roles in this constantly evolving issue.

Currently, the climate change/human health intersection is not well established within the house of medicine. Some place it under wilderness medicine not only because these practitioners have an innate love for the environment but also have the technical skills to reach and practice in the fragile ecosystems that are the front line. In addition, the clinical skill set is well suited to manage the climate change related health effects. Wilderness medicine inherently overlaps with the international and disaster sub-specialties which also play key roles in responding to the health effects of climate change.

With history as an indicator, it was physicians who organized and created the International Physicians for the Prevention of Nuclear War (IPPNW) and were subsequently awarded the Nobel Peace Prize for their key influence in prevention of a nuclear holocaust. They felt it was a physician's duty. The current role of a physician in the arena of climate change is nebulous. Two key leadership roles, as I currently see it, are for physicians to provide the synthesis / advocacy or perform primary research. The goal of synthesis and advocacy is to re-frame climate change as a public health issue. Multidisciplinary efforts can be aimed at physicians through incorporation of this topic in educational venues and the creation of advocacy organizations similar to the IPPNW. In addition, I feel that every physician should take it upon themselves to learn the basics of the greatest global health threat of the 21st century. This is the nuclear holocaust of our generation, and the time to take action is now.

1 - Costello, A et al. Managing the health effects of climate change. Lancet. 2009; 373:1693-733.
2 - World Health Organization. 2014. Retrieved from http://www.who.int/globalchange/environment/climatechange-2014-report/en/
3 - IPCC. Fifth Assessment Report. 2014. Retrieved from http://www.ipcc.ch/.
4 - Levitus et al. World ocean heat content and thermosteric sea level change (0-2000 m), 1955-2010. Geophys. Res. Lett. 2012:39, L10603.
5 - World Health Organization. 2014. Retrieved from http://www.who.int/mediacentre/factsheets/fs266/en/

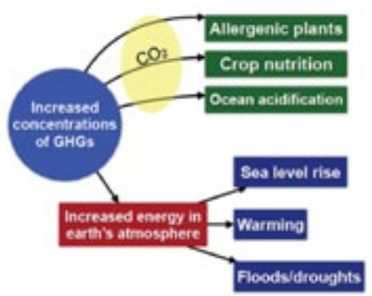

Figure 2: Effects of climate change on human health (courtesy of Aaron Bernstein, MD, MPH)

Botulism
Continued from page 5

grossly symmetric in bilateral upper and lower extremities 5/5 strength. Gait was normal.

Emergency Department Course:

Given the patient's multiple visits for "sore throat" and her neurologic findings, there was heightened suspicion for a more serious process. Basic laboratory tests were ordered, including CBC, BMP, ESR, CRP. She had an elevated white count of 15.4, an elevated CRP of 23, and mild electrolyte disturbance with Na+ of 153 and K+ of 3.2. Given concern for myethenia gravis ice pack test was performed which did not produce fatigue. Bedside nasopharyngoscopy was performed to assess for any structural etiologies. This only revealed pooled secretions with otherwise a normal examination. A NIF and FVC were completed at bedside to assess the patient's respiratory function, which revealed a NIF of -27 (normal <-25), and an FVC of 1.3 liters (normal 3-5L)

Given the patient's dysphagia and neurologic findings neurology was consulted. They were also impressed with the patient's neurologic findings and recommended admission to their service. At this time myasthenia gravis and guillan barre syndrome were the most significant differential diagnoses being considered. However, the neurology resident looked into the friend's information about the patient's roommate and discovered that she was a patient in the neuro ICU unit who was currently intubated for respiratory failure. Given the presentation of the patient and her roommate, the team did not feel this was a coincidence and botulism was elevated to the top of the differential. The patient was subsequently admitted to the NSICU for further management. The patient was thus admitted to the neuro ICU for close monitoring.

Hospital Course:

Overnight in the neuro ICU, the patient underwent elective intubation for slightly but progressively worsening NIF and FVC values. Further history from friends and family gathered by the inpatient team revealed that on the night prior to the onset of their symptoms both the patient and her roommate had shared a dinner consisting of chicken with home canned pesto sauce. The patient's mom brought in the pesto sauce, and it was sent to the State Health Department for analysis which eventually revealed presence of botulinum toxin. The patient was administered botulism antitoxin attained from the CDC.

On her 4th day in the ICU, the patient showed improvement in her respiratory parameters and was successfully extubated. She continued to have some difficulty controlling secretions and thus tube feedings were continued. She was transferred days later to a rehabilitation center for continued speech therapy and management of her tube feeds.

discussion

Botulism is an acute illness causing paralysis that is mediated by a neurotoxin produced by Clostridium botulinum. C. botulinum is a sporulating, anaerobic, gram-positive bacillus found in many soil and aquatic sediments. The toxin, botulinum neurotoxin (BoNT), can be of several types, differentiated by varying antigenicity, but types A, B, E and rarely F are most often associated with human disease.

BoNT, is an extremely potent neurotoxin that attacks the presynaptic terminal at the neuromuscular junction. It is estimated that as little as 1 g of aerosolized BoNT could cause the death of ~ 1.5 million people (McNally 1994). The neurotoxin, once absorbed into the body, acts as an active protease in the presynaptic terminal that prevents the release of neurotransmitters (namely acetylcholine etc.) into the synaptic cleft and thus prevents signal transmission.

The four major forms of Botulism in humans are: infant, food-borne, wound, and adult intestinal toxemia botulism. In addition, there are very rare reports of disease caused by either inhalation or iatrogenic therapeutic injection of the toxin. Across the United States it is estimated that annually there may be as many as 250 cases of infant Botulism (Cox 2002), versus roughly 24 cases of food-borne disease (McLauchlin 2006) whereas only a few cases of adult intestinal toxemia have been reported (Shapiro 1998).

The rapidity of progression of the clinical course is determined by whether preformed toxin or spores that germinate in the intestinal tract are ingested. However the clinical presentation remains consistent as the toxin attacks cranial nerves producing symptoms including blurred vision, diplopia, ptosis, dysarthria, dysphonia and dysphagia (Demebek 2007). There are varying degrees of descending muscle paralysis beginning with the neck and progressing to include respiratory muscles often requiring mechanical ventilation.[1] Death from botulism occurs secondary to respiratory arrest due to respiratory muscle paralysis/weakness and ultimate diaphragmatic failure.[8]

Left unrecognized or untreated, mortality from botulism may be as high as 40%.[3] However with treatment foodborne botulism has an overall mortality of 5-10%, wound botulism 15-17% and infant botulism less than 1%. Diagnosis is based on clinical presentation and history, and toxin identification from serum, stool, gastric aspirate, or vomitus or from growth of C. botulinum in culture. Treatment for botulism includes admission and aggressive supportive care. Spirometry, pulse oximetry, vital capacity should be followed sequentially as respiratory collapse can occur rapidly. Mechanical ventilation should be undertaken when vital capacity or negative inspiratory force is less than 30% of predicted. Medication treatment includes either an equine-derived antitoxin or a newly FDA approved (2013) human derived immune globulin against all 7 known serotypes of the nerve toxin. The human derived antitoxin has far less reported incidence of anaphylaxis as compared to the equine version.[2]

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Peds Tox
Continued from page 4

should start with ABCs, just like any patient who comes into the ED. Management after this should focus on predicting severity of injury and getting GI involved early. NG tubes should not be placed blindly and attempts to neutralize or dilute the substance or induce vomiting should be avoided. These patients are at risk for development of strictures and malignancy of the esophagus.

Mowery, Spyker, et al; 2012 Annual Report of the American Association of Poison Control Centers ' National Poison Data System (NPDS): 30th Annual Report
Lupa, M; Magne, J; Amedee, R; Update on the Diagnosis and Treatment of Caustic Ingestion; Ochsner Journal; 2009 Summer
Salzman, Matthew (05/01/2007). Updates on the Evaluation and Management of Caustic Exposures. Emergency medicine clinics of North America (0733-8627), 25 (2), p. 459. DOI: 10.1016/j.emc.2007.02.007 vv

patient after reviewing her chest film, she had been told about the findings from the CXR performed in 5/2014 but had not followed up secondary to some personal issues. Given her lack of primary care follow-up, her exam and radiography consistent with superior vena cava syndrome and malignancy, and the lack of pulmonology and vascular surgery availability at my facility, the patient was transferred to a tertiary care facility for further evaluation and management.

Records of the patient's stay at the tertiary care facility are not available to me; however, the patient did present to our ED one month later. She was hospitalized for 11 days, during which time she underwent bronchoscopy and biopsy, SVC stenting, and removal of a nodule from her vocal cord, subsequent to which she required mechanical ventilation for three days. Her facial, neck, and upper extremity swelling has completely resolved and her dyspnea is improved. She has started chemotherapy and radiation treatment.

discussion

Superior vena cava (SVC) syndrome is rare, with an incidence of approximately 15,000 cases per year in the US. The leading causes of SVC syndrome were once infectious (syphilitic thoracic aortic aneurysms, fibrosing mediastinitis), but in the modern era 60-85% of cases are related to malignancy, with lung cancer and non-Hodgkin lymphoma being the most common. The remainder of cases most often result from complications of indwelling central venous catheters.

Presenting symptoms of SVC syndrome include dyspnea, swelling of the face, neck, and less commonly arms, cough, chest pain, and dysphagia. Laryngeal edema can cause hoarseness and stridor. In severe cases, cerebral edema can result, causing headaches and confusion.

> “Most cases of SVC syndrome do not require emergent therapy because collateralization occurs”

A grading scale has been proposed by Yu et al. (Table 1)

In the past, the initial treatment of SVC syndrome was emergent radiation therapy. This had the drawback of interfering with subsequent histologic evaluation if the etiology was not already established. Currently, endovascular stenting is more commonly used and results in more rapid symptomatic improvement, although no specific treatment guidelines have been established. Most cases of SVC syndrome do not require emergent therapy because collateralization occurs quickly, although the development of severe laryngeal or cerebral edema necessitates immediate intervention.

Learning Points:

- The patient's exam findings were to me not terribly impressive initially – she was a very large lady and her face, although reddened, was no redder than mine or Dr. Carleton's on an average shift. I was shocked by how different she looked the second time I saw her. If I had asked to look at her driver's license or other previous photo, I might have realized the full extent of her symptoms on her initial visit. It wouldn't have changed my management in this case, but it is good to remember that an old photo can be as valuable as an old EKG.
- The CXR that the patient had done in May was performed as part of a routine pre-op evaluation, and the findings were incidental. It appears that they were appropriately communicated to the patient, and my sense from the patient is that she did not follow up because she was afraid of confirming that she had cancer. This case does speak to the importance of communicating those findings and documenting that you have done so.

Yu JB, Wilson LD, Detterbeck FC. Superior vena cava syndrome – a proposed classification system and algorithm for management. J Thorac Oncol. 2008 Aug;3(8):811-4
Up-to-date. Superior Vena Cava syndrome.

Chest X-Ray

Figure 1

1. There has been interval increase in the size of a large mass in the posterior aspect of the right lower lobe and probable increase in the size of a mass or lymphadenopathy in the right hilum with adjacent partial atelectasis of the right upper lobe since the prior study of 5/8/2014. These findings are most compatible with lung cancer until proven otherwise. A chest CT with contrast is recommended for a more definitive evaluation of these findings.
2. Development of mild elevation of the right hemidiaphragm

Chest CT

1. The patient has a large right hilar and mediastinal invasive malignancy which is infiltrated throughout the mediastinum and extends along the right paramediastinal region towards the anterior chest.
2. There is tumor encasement of the right main pulmonary artery with marked narrowing. There is also moderate encasement of the right mainstem bronchus. Most notably the patient is developing at least radiographically a superior vena cava syndrome as there is severe compression of the vena cava with centrally near occlusion. There is also occlusion of the azygos vessels from a surrounding tumor. Collateralization is developing across the chest wall and neck.
3. Multiple metastatic lesions are seen throughout the lung fields, the largest in the right lower lobe measuring 4.5 cm. There is also a large metastatic lesion to the spleen and to the right adrenal gland.
4. Patient is a developing a postobstructive pneumonitis predominantly in the right upper lobe and portions of the right middle lobe secondary to the marked mass involvement in the mediastinal and hilar regions.

Grading SVC Syndrome

grade	category	incidence (%)	definition
0	Asymptomatic	10	Radiographic superior vena cava obstruction in the absence of symptoms
1	Mild	25	Edema in head or neck (vascular distention), cyanosis, plethora
2	Moderate	50	Edema in head or neck with functional impairment (mild dysphagia, cough, mild or moderate impairment of head, jaw or eyelid movements, visual disturbances caused by ocular edema)
3	Severe	10	Mild or moderate cerebral edema (headache, dizziness) or mild/moderate laryngeal edema or diminished cardiac reserve (syncope after bending)
4	Life-threatening	5	Significant cerebral edema (confusion, obtundation) or significant laryngeal edema (stridor) or significant hemodynamic compromise (syncope without precipitating factors, hypotension, renal insufficiency)
5	Fatal	<1	Death

Table 1: Proposed grading scheme of SVC syndrome (Yu et al)

List of Submitted B Pod Cases

Eckart/Ford	Perinephric hematoma
Eckart/Ford	Acute Limb Ischemia
Dang/Moschella	Acetaminophen OD
Connell/Moschella	AKI/Hypotension
Plash/Moschella	Wellen's
Eckart/Bohanske	Pulmonary Embolus
Fichtenbaum/Cousar	Shingles
Connell/Ford	NSTEMI
Titone/Cousar	Secondary Syphilis
Connell/Gozman	PCA Stroke
Connell/Moschella	Psychogenic Polydipsia
Dang/Moschella	Idiopathic Intracranial Hypertension
Polsinelli/Yamin	Septic Emboli
Holmes/Gozman	Malaria
Polsinelli/Gozman	Endocarditis
Lagasse/Stull	Guillen-Barre
Lagasse/Stull	Acetaminophen + Ethylene Glycol OD
Polsinelli/Yamin	Acute Hepatitis C
Dang/Cousar	Posterior STEMI

Annals of B Pod is looking for YOU to submit your interesting cases of B Pod - There is a composition book at the R4 desk - please ensure to include the R1/R4 involved in the case, a brief synopsis and a patient sticker

Any ideas for features, guest columns, or other comments can be forwarded to the editors at annalseditors@gmail.com

comes, defined by right ventricular improvement, perfusion defects on V/Q scan, or pulmonary artery obstruction on CTPA at 24 hours or 14 days. However, they did find an increased incidence of bleeding complications in the 100mg infusion (32% vs 17%; p=0.054). This finding was emphasized in a subgroup analysis that evaluated dose effect on patients who are ≤ 65kg compared to those > 65kg. When the patients where stratified by weight, the study showed that patients ≤ 65kg and BMI < 25 kg/m2 where at highest risk of bleed with 100mg infusion (14.8% vs 41.2%; p=0.049; 8.7% vs 42.9%; p=0.014). The Moderate Pulmonary Embolism Treated with Thrombolysis (MOPPET) trial[10] looked at alteplase 0.5 mg/kg (max dose of 50mg) infusion vs anticoagulation with heparin alone in patients with submassive PE. Patients who received the alteplase had less pulmonary hypertension and recurrent PE at 28 days (16% vs 63%; p<0.001). These studies support exploring the use of lower doses (<100mg) alteplase in low weight patients and submassive PE. UCMC's dosing policy mirrors the findings of these studies.

Unfractionated Heparin during Systemic Thrombolysis

Systemic anticoagulation with unfractionated heparin (UFH) (intravenous or subcutaneous), low molecular weight heparin, and fondaparinux are currently recommended for treatment of acute PE. The advantage of using intravenous unfractionated heparin in acute PE and systemic thrombolysis is the short half-life and ability to reverse with protamine in the case of emergent bleed. Most studies investigating systemic thrombolysis in acute PE are European and infuse alteplase with heparin infusions concurrently. However upon approval for alteplase use in PE, the USA FDA formally recommends the suspension of IV UFH during systemic thrombolysis and checking an hPTT after completion of infusion.[6] On a recent survey of approximately ten American academic teaching institutions, 100% hold heparin infusions during systemic thrombolysis. There is still a great amount of ambiguity related to when to restart the heparin infusion and at what hPTT is considered safe post alteplase infusion. Alteplase package insert recommends restarting the heparin infusion when the hPTT returns to two times baseline. American College of Chest Physicians recommend waiting until the hPTT is ≤ 80 (approximately two times baseline).[4] UCMC's dosing guidelines recommend to check an hPTT after alteplase infusion is finished and to resume the heparin infusion per our standard heparin with bolus protocol. This would allow the patient to maintain or get to therapeutic hPTT range (90-130 seconds) as quickly as possible.

In conclusion, UCMC's altplase dosing guideline supports the use of lower doses in low weight patients with massive PE and submassive PE based on recent research in these patient populations. UCMC also recognizes that most studies with systemic thrombolytics concurrently administer IV UFH during alteplase infusion, however does not recommend going against USA FDA's recommendation to suspend the infusion.

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8 - Vahanian, et al. Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC).European Heart Journal (2008) 29, 2276–2315.
9 - Meyer G, et al. Fibrinolysis for Patients with intermediate Risk Pulmonary Embolism (PEITHO Investigators). N Engl J Med 2014;370:1402-11
10 - Sharifi M, Bay C, Skrocki L, Rahimi F, Mehdipour M. Moderate Pulmonary Embolism Treated With Thrombolysis (MOPETT). Am J Cardiol 2013;111:273e277.
11 - Wang C, Zhai Z, Yang Y, Wu Q, et al. Efficacy and Safety of Low Dose Recombinant Tissue-Type Plasminogen Activator for the Treatment of Acute Pulmonary Thromboembolism: A Randomized, Multicenter, Controlled Trial. Chest 2010;137;254-262.
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The Levy by the Numbers

UCMC's indigent care funding returns to the ballot

Dan Axelson, MD, MPH
University of Cincinnati R2

This November's election in Hamilton County will bring 40% of UC's charity medical budget up for a vote. The Hamilton County Tax Levy ("the Levy"), responsible in large part for reimbursing UC's charity care, is up for renewal. Heading in to election day, much uncertainty surrounding the Levy remains. This directly affects the emergency department, and we must be informed.

First passed in 1966, the Levy raised funds for improvements to Cincinnati's General Hospital. Cincinnati Children's was added to the Levy in 1976. It has been renewed every time it reached the ballot box since. Over the years, the Levy has morphed in to a principle funding stream allowing UC to continue providing medical care to those who otherwise could not afford it. Of the more than $50 million of free medical care UC gives out annually, the Levy reimburses $20.1 million. An additional $5 million goes to CCHMC's charity program. The remainder of the Levy funds go toward prison system medical care, and a smattering of other state initiatives.

Enter the Affordable Care Act (ACA) of 2010. Under President Obama's law, the state of Ohio has expanded eligibility for Medicaid, its medical coverage program for the poor. Theoretically, 70,000 previously uninsured residents of Butler, Clermont, Hamilton and Warren counties now qualify for Medicaid coverage, which calls into question the need for a separate, county funding stream for these patients' medical care. Opponents of the Levy cite that if the ACA achieves this coverage aim, the Levy would serve as a double tax on Cincinnati residents for indigent medical coverage with Medicaid and the Levy footing the same bill. However, continued federal delays in the ACA's implementation, as well as persistent legal uncertainties surrounding the law, make doing away with the Levy altogether a questionable proposition. The number of uninsured remains high despite a decrease (2013: 19%, 2014: 11%)[1]. In addition, the ACA does not ensure a patient's access to primary care. This remains a barrier to care that the Levy now covers, the loss of which would leave an already vulnerable patient population with even fewer options.

The Hamilton County Board of County Commissioners (BOCC) recently put forth seven recommendations regarding the Levy. The infographic on the right of this article explains three of these that are directly pertinent to us in the ED. The issue will be voted on November 4th, 1014 as Issue 7 on the ballot - remember to get out and vote.

1 - Institute for Policy Research of the University of Cincinnati. "Ohio Health Issues Poll (OHIP)" May 2014. https://www.interactforhealth.org/upl/OHIP_Uninsured_FINAL_082514.pdf

Current Hamilton County Board of County Commissioners (BOCC) Recommendations

11/2014

The Levy should be placed on the November, 2014 ballot at the current millage.

A millage is a tax based on the value of one' s property. Currently, the Levy' s millage costs the owner of a $100,000 home in the area $45.87 a year. Given lingering uncertainty in the healthcare landscape, the BOCC recommends freezing rates of the millage in the short term, but keeping the Levy on the ballot for potential renewal.

$13.5 MIL

UCMC should be funded in the Levy at $13.5 million a year.

This is a recommended decrease from prior annual funding, but notably not a recommendation for withdrawal of funds altogether. The decrease stems from a predicted positive financial impact on UCMC by the ACA. The continuation stems from UCMC' s good stewardship of funds to date, and their continued push to emphasize primary care over ED-based indigent care.

3 YEARS

The Levy term should be three years.

Traditionally renewed every five years, it' s first short-term, three year renewal was in 2011. The hope was that by 2014 the ACA would have clarified the healthcare landscape. With remaining uncertainty, the BOCC formally recommends another 3-year term.

Quick Hit
Visual Dx

Tyler Winders, MD
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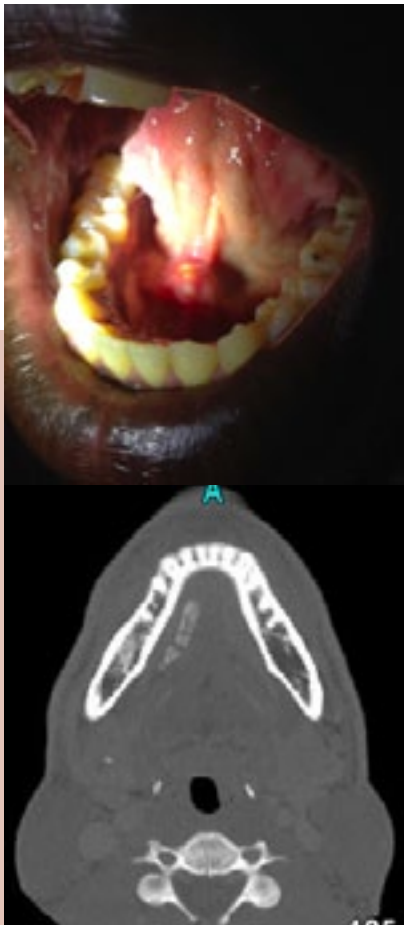
31 year old male, chief complaint...

"My jaw hurts and swells when I eat"

What's the diagnosis?

Answer on page 10



CT Exam