ANNALS OF B POD

Since 1970 - Leadership - Excellence - Opportunity

Celebrating 10 Years
2006 - 2016

2011
Critical care and community ED topics are highlighted

2006
Dr. Bernard publishes the first issue
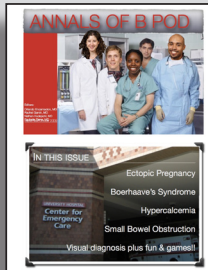
2010
The first pharmacy article is published

10th Anniversary

2013
AOBP is published online

2006 ——————————————————— 2016

2010
Led by Dr. Gensic, AOBP has its most productive year, publishing 6 issues

2008
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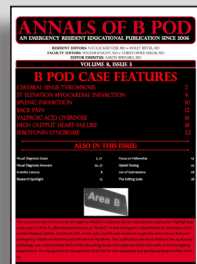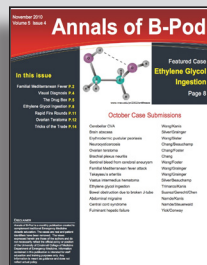
2015
AOBP teams up with PEM Blog for peds-EM topics
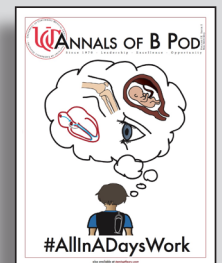
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UC EMERGENCY MEDICINE

ANNALS OF B POD
FALL 2016

10 years

This issue marks the 10 year anniversary of Annals of B Pod. After 10 years and over 300 articles, Annals of B Pod continues to fulfill its initial mission of sharing the interesting case presentations, unique pathology and diagnostic dilemmas that present to B Pod.

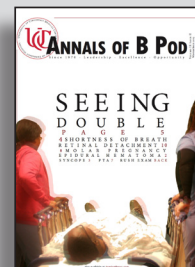The time and effort put into publishing each issue by resident and faculty editors highlights their dedication to continually producing high-quality work. Each issue feels special because we, as editors, know that the publication embodies a unique and treasured tradition at the University of Cincinnati Emergency Medicine Residency. This issue feels extra special because it marks the beginning of our year-long celebration of our 10th anniversary. Over the coming year, former editors will reflect on their time in B Pod, and how sharing these experiences through Annals of B Pod shaped their career.

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The BEAUTY of B Pod

Molly Bister, MD
University of Cincinnati
Class of 2014

At this point, I've been away from B Pod as long as I was in it. And yet, I often feel as if I've never left. My current ED has (on paper, anyway) 19 beds, and is set up in roughly the same circular configuration. It even has a little off-shoot, like B Pod did during my fourth year.

On the surface, the patients look different: rural instead of urban. Overwhelmingly white. Lots of kids. Their problems, though, are the same: spotty access to primary care. High unemployment. Low health literacy — low literacy of any kind, really. Polysubstance abuse. Uncontrolled chronic health problems. Untreated mental illness.

I have medical students and residents that rotate through as well. And it was working on Annals that really brought home that there is no such thing as a boring case. That there are teachable moments in even the most routine COPD exacerbation. And that sometimes there is a zebra lurking amongst the often endless-seeming stream of gastroenteritis, renal colic, CHF, and chronic pain issues.

B Pod is where I learned that every patient deserves at least a minute of honest thought (Dr. Stettler). That you must strive for consistency of thought and action (Dr. Baxter). And from everyone, but most prominently Dr. Carleton, that you always — always — do the best thing for the patient.

It's not as easy to do the best thing out here as it is in B Pod. Limited access to specialists. Hospitalists who can and often do refuse admissions. Patients who don't understand why they need to be transferred to a tertiary facility an hour and a half away and can't be admitted locally. Nursing staff with big hearts but little experience. No SRU to bump patients to when they start to crump. And, of course, the ever-present specter of Press-Ganey over it all.

But I don't think I could have been better prepared to fight those battles, because, as I said in the beginning, I never really left B Pod.
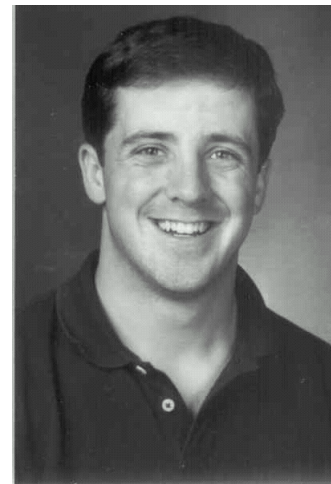
B Pod is not just a place. It's a state of mind. It's the foundation of the Cincinnati tradition and expectation of excellence. It's in our hearts. And for that, I will always be grateful.

Molly Bister

A Letter From...
Our Editor Emeritus

Aaron Bernard, MD
University of Cincinnati
Class of 2007

Dear Colleagues,

It's been 10 years since the first issue of Annals of B Pod, and the R4s and R1s who founded this journal are now well-established clinicians and academicians. I find myself reading the most recent issue of Annals of B Pod and staring at it with nostalgia for the past, awe of the present, and inspiration for the future.

Like many classes that graduated from the University of Cincinnati Department of Emergency Medicine, the Class of 2007 reveled in the Department's tradition, prided itself on excellent patient care, and hoped to make their mark as a class and as individuals in the specialty of Emergency Medicine. The journal arose out of a desire of R4s to teach and an interest of the R1s to learn. There was a sense that great learning was happening and excellent patient care was being provided, but the experiences were not being fully appreciated. We wanted to share the fascinating cases we had not just with those on duty, but rather with all of our colleagues at UC and potentially beyond.

The first edition was a Microsoft Word document with pirated graphics and lacked the sophistication and polish the journal now has. I was nervous to distribute it but was relieved to find it was met with an overwhelmingly positive response. Perhaps more than the medical knowledge, the first edition brought excitement. The journal provided a sense of community, a shared experience, and a pride in the patients that we care for and the job that we do. With these emotions as a backwind, and with the hard work and enthusiasm of subsequent editors and contributors, the journal continued on to this day. I could never imagine it would last this long nor that it would develop into such a high-quality publication.

As one of the most imperfect residents ever to graduate from the Department, I am honored that you have left my name on the journal as Editor Emeritus and feel humbled to think I could be even a small part of such a proud tradition that is UC Emergency Medicine. I'm excited to see where this journal goes in the future, and excited to think about how the current R4s and R1s will make their mark.

Leadership. Excellence. Opportunity.

Sincerely,

Aaron Bernard

Giant Cell ARTERITIS

Nicole Soria, MD
University of Cincinnati R2

History of Present Illness

The patient is a female in her 80s with a past medical history significant for elevated blood pressure without a formal diagnosis of hypertension who was sent to the Emergency Department from her primary care physician's office with a chief complaint of sinus congestion and vision changes. She reports that about a week ago she began having "sinus pressure" over her right forehead and cheek without associated cough, runny nose, or fevers.

She also notes a few days of double vision in her right eye with gradual change in her peripheral vision. Otherwise, she denies any photophobia or pain with eye movement. She also denies ocular discharge, erythema, or pruritus. She denies dizziness, seizures, or difficulty with speech. While she does endorse a mild posterior headache on presentation, she denies any other recent headaches. She does describe some jaw discomfort and pain with chewing recently. She has tried over the counter medications without improvement, but has taken aspirin with some relief.

Past Medical History
Elevated blood pressure

Social History
0.5 ppd, denies alcohol use

Allergies
None

Medications
None

Vitals
T 36.1 HR 90 BP 153/71 RR 16 SpO2 97% on RA

Physical Exam

The patient is a well-appearing elderly female who is in no acute distress. Her eye exam reveals pupils that are approximately 4mm and round with an afferent pupillary defect on the right. Retinal exam reveals no obvious optic disc edema or AV nicking. Visual acuity is 20/50 OD and 20/25 OS. Intraocular pressure is 13 mmHg OD and 14 mmHg OS. Her extraocular movements are intact. Her head shows no physical signs of trauma. She has no temporal tenderness bilaterally. Her neck has no lymphadenopathy, no JVD, and range of motion is normal.

Cardiovascular and pulmonary exams are unremarkable. Her abdominal exam is benign. She is alert and oriented to person, place, time, and situation and has no cranial nerve deficits or other lateralizing features and exhibits normal coordination.

Labs and Imaging

WBC: 13.5 ESR: 64 CRP 100.7
Non-contrast head CT: normal

Hospital Course

The patient's history, presentation, and workup were concerning for an ophthalmologic (*e.g.*, central retinal artery or vein occlusion), intracranial (*e.g.*, tumor, optic neuritis, MS lesion, stroke, bleed, abscess) or rheumatologic (*e.g.*, giant cell arteritis) etiology. Ophthalmology was consulted and agreed that her age, recent development of jaw claudication and right eye afferent pupillary defect in the setting of a negative head CT were all concerning for temporal arteritis. She was admitted as an inpatient to the medicine service to receive IV solumedrol for three days, and ENT was consulted for a temporal artery biopsy. She was started on a baby aspirin, and rheumatology was also consulted. Her biopsy confirmed the diagnosis of giant cell arteritis. The patient was discharged on high-dose prednisone and aspirin, and is scheduled for follow up with rheumatology, ophthalmology, and ENT.
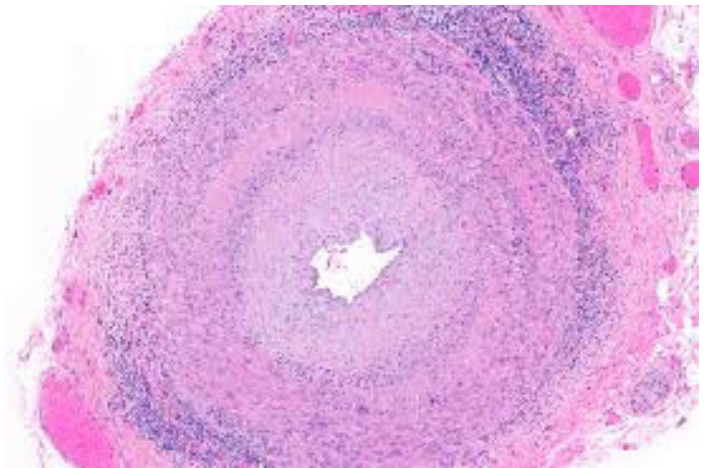
Figure 1: A representative temporal artery biopsy demonstrating the necrotizing vasculitis seen in giant cell arteritis. Image from Wikimedia Commons.

Discussion

Giant cell arteritis (GCA), also known as temporal arteritis or cranial arteritis, is a chronic granulomatous vasculitis of unknown etiology affecting the medium- and large-sized arteries. First described by Horton et al in 1937, it is the most common systemic vasculitis in Western countries with an overall incidence rate of 15-25 per 100,000 each year.[1,2] GCA is primarily seen in the elderly, and its incidence rises to 45 per 100,000 for those in their ninth decade.[3] It is a challenging diagnosis that has a myriad of presentations and high recurrence rate despite optimal treatment. Prompt diagnosis, however, can prevent long-term sequelae of the disease, making identification in the Emergency Department crucial.

The presenting symptoms in GCA are widespread and can be vague. Classically, it presents in an individual older than 55 years of age with a new onset headache accompanied by temporal tenderness and jaw pain or claudication. Other common symptoms can include scalp tenderness, temporal artery abnormalities on physical exam, visual symptoms, and associated polymyalgia rheumatica. While the temporal artery is the most well-known artery to be affected by GCA and is the culprit for the classic presenting symptoms described above, many other aortic branch arteries can be involved, including the subclavian, axillary, ophthalmic, ciliary, and vertebral arteries.[2]

With regards to visual symptoms, the pathophysiology stems from optic nerve ischemia due to ophthalmic artery inflammation. Classically, these patients will present with sudden, painless, monocular or bilateral visual loss. However, patients may also complain of diplopia or ocular pain. GCA should be considered in any elderly patient who complains of visual symptoms. On physical exam, the optic nerve will appear swollen and pale, often with flame shaped hemorrhages. As the swelling subsides, optic atrophy sets in, and vision can be significantly impaired. An afferent pupillary defect can also be seen.

In GCA, extra-cranial branches are affected 10-15% of the time.[6] Some patients may present with symptoms directly related to the artery involved, such as arm claudication in patients with subclavian artery involvement, or aortic dissection in patients with aortic involvement. However, patients with extra-cranial involvement often present with non-specific and constitutional symptoms such as weight loss, anorexia, asthenia, night sweats, or low grade fevers.

While the diagnosis of GCA may be straightforward in some patients, it is often much more challenging. In the ED, any elderly patient presenting with an acute or subacute headache with accompanying neurological or systemic symptoms should raise concern for GCA. However, broadening the differential to include intracranial hemorrhage, mass, ischemic stroke, arterial dissection, malignant hypertension or infection is critical, and workup for patients with these complaints should include lab work, imaging and a thorough neurologic exam.

The laboratory hallmark of GCA is an elevation in acute phase reactants, and >95% of the time both erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) will be abnormal.[4] ESR is elevated >100 mm/h up to 60% of the time, but may be normal in 7-20% of patients with GCA.[4,6] As such, a normal ESR does not rule out GCA. Additionally, the ESR level does not correlate reliably with the severity of disease. Furthermore, because normal values of ESR increase with age and are

> **Age-Adjusted ESR Values**
>
> Males: [Age] / 2
>
> Females: [Age + 10] / 2

Table 1 Age-adjusted upper limit of normal values for ESR in men and women. From Kale and Eggenberger, 2010.

higher in women, the upper limit of normal for ESR should be adequately age-adjusted (see Table 1).[3,7] Other laboratory abnormalities in patients with GCA include anemia, thrombocytopenia, and an elevated alkaline phosphatase.

When only the aorta or great arteries are involved, constitutional symptoms and elevated serologic inflammatory markers may be the only signs of GCA. Thus, the diagnosis should be considered for patients who present with fever of unknown origin, unexplained weight loss, or unexplained elevated ESR or CRP.[5]

While there are new diagnostic modalities being investigated, for GCA including ultrasonography, high-resolution magnetic resonance imaging and positron emission tomography, the gold standard for diagnosis remains temporal artery biopsy. This is typically done by general surgery, rheumatology or ENT. A large area (>2 cm) should be biopsied to avoid missing the skip lesions that can occur in GCA. Classically, a necrotizing vasculitis is seen (Figure 1).

In GCA, a prompt diagnosis is important, largely because of the morbidity associated with the complications, specifically with regards to ocular symptoms. Patients suspected of having GCA should be started on steroids

prior to definitive diagnosis to help prevent permanent vision loss. While the ideal steroid regimen is unknown, an initial dose of 40 to 60 mg of oral prednisone daily has been shown to be effective. However, if the patient already is exhibiting visual symptoms, there is some evidence that pulse-dose methylprednisolone is superior, which requires initial inpatient admission. Additionally, temporal artery biopsy should be performed within 48 hours of starting steroids, which may require inpatient admission from the ED for high-risk patients. Patients should also be started on low dose aspirin, as it has been shown to significantly decrease the rate of vision loss and stroke during the course of disease.[4]

Patients may require long-term steroid administration for GCA, and should be counseled on the side effects of short- and long-term steroid usage, including hyperglycemia, weight gain, and osteoporosis.

Many patients have improvement in their visual symptoms after initiating treatment. However, for some, visual loss is permanent, especially those with a delayed diagnosis. A timely diagnosis of GCA by the ED physician is key to preventing long term damage, and requires a high index of suspicion for patients in the key demographic who present with headache, jaw pain, or systemic constitutional symptoms.

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Incidentaloma

Jessica Merriam, MD
University of Cincinnati R2

History of Present Illness

The patient is a Nepalese-speaking female in her 50s with no known medical history who presents to the Emergency Department with nausea, vomiting, and diarrhea for the past three days. She describes numerous episodes of non-bloody, non-bilious vomiting associated with persistent nausea throughout this time. She also endorses countless episodes of loose, watery stools which are also non-bloody. She does note some mild, crampy abdominal pain which is greatest in the epigastric region and primarily occurs just prior to her episodes of diarrhea.

On the day of presentation she became diaphoretic with her symptoms, so her family encouraged her to come to the ED for evaluation. She denies any fevers, chest pain, shortness of breath, dysuria, or sick contacts. She has occasionally had similar episodes of vomiting and diarrhea over the past several months. She also endorses occasional diffuse headaches over the past few months that usually respond to Tylenol. She also denies weight loss, fevers or other constitutional symptoms.

Of note, the patient states that she is from Nepal and just moved to the United States two weeks prior to her ED visit. She has spent the past 20 years of her life in a refugee camp in Nepal and has not visited a doctor during this time. She has not yet established care with a primary physician in the U.S.

Vitals

T 36.5   HR: 82   BP: 138/103   RR: 15   SpO2 98% on RA

Physical Exam

Physical exam reveals a thin female who appears uncomfortable. HEENT exam is unremarkable, and mucous membranes are moist. The patient's pulmonary and cardiovascular exams are normal. Abdominal exam reveals a soft, non-distended abdomen with active bowel sounds, mild periumbilical tenderness but no rebound, guarding or peritoneal signs. She is mildly diaphoretic, but the remainder of her musculoskeletal and skin exams are normal. She is awake, alert and oriented with a nonfocal neurologic exam.

Imaging

CT Abdomen/Pelvis: 5.2 cm left adrenal neoplasm is incompletely characterized. Central area of low attenuation could represent necrosis or fat. Recommend enhanced abdominal MRI for further evaluation. Diffuse osteopenia.

CXR: Large right lung apex opacity, likely a combination of right upper lobe atelectasis and neoplasm. Findings likely reflect an endobronchial obstructing right upper lobe mass.

Hospital Course

This patient presented with nausea, vomiting and abdominal pain and was found to have both a lung mass and an adrenal mass. In a woman who had recently immigrated from a refugee camp in Nepal, there was significant concern for tuberculosis or malignancy. She was admitted to the hospital for several days, and was seen by both the pulmonary and infectious disease services. Ultimately, it was felt that her lung findings were residual architectural changes from a previous tuberculosis exposure, and active tuberculosis was ruled out. She was discharged with plans for outpatient work-up of what was felt to be an incidentally found adrenal mass.

However, she returned to the hospital several weeks later for hypovolemic shock presumed to be due to her persistent, profound diarrhea. She was admitted to the MICU and underwent an extensive work-up for an infectious cause of her symptoms, including intestinal biopsies to evaluate for GI tuberculosis, all of which were unremarkable.

The patient then began developing progressive weight loss and sweating, and was eventually seen by endocrinology who sent urine metanephrine levels given her known adrenal mass. These resulted with values greater than ten times the upper limit of normal and she was ultimately diagnosed with a pheochromocytoma. She was started on alpha-blocking medications and six weeks later underwent uncomplicated surgical removal of the mass. She has been doing well since the procedure with near complete resolution of her symptoms and follows regularly with her primary care physician.

Discussion

Incidental findings are not uncommon in the Emergency Department given the frequency of advanced imaging performed as part of diagnostic workups. Adrenal incidentalomas are defined as adrenal masses that are incidentally discovered on abdominal imaging performed for reasons other than suspected adrenal dysfunction. The prevalence of such adrenal lesions ranges between 1.4-2.9% on autopsy with an increase in frequency with advanced age.[1] Nearly half of these masses are benign adenomas, and they often play no role in the reason for the Emergency Department visit. Nonetheless, these lesions can be malignant or secretory lesions, as in the above patient, which may actually contribute to the ultimate diagnosis. In this category of more ominous adrenal lesions, pheochromocytomas are still exceedingly rare, comprising only 8% of all adrenal masses.[2]

During embryogenesis, neural crest cells migrate to the sympathetic chain and the adrenal medulla, resulting in chromaffin cells which are responsible for secreting the catecholamines epinephrine, norepinephrine, and

Simple Sugar:
Diabetic Ketoacidosis AND
Hyperosmolar Hyperglycemic Syndrome

...What's the Difference?

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University of Cincinnati

Edited by: Nicole Harger, PharmD, BCPS

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic syndrome (HHS) are both considered hyperglycemic crises. However, they have different treatment algorithms, making it imperative that providers are able to differentiate between the two disease processes (Table 1), as failure to initiate appropriate treatment can lead to poor patient outcomes.

	DKA			HHS
	Mild	Moderate	Severe	
Glucose	>250	>250	>250	>600
pH	7.25 - 7.30	7.00 - 7.24	<7.00	>7.30
Bicarbonate	15 - 18	10 - 14	<10	>18
Urine/Serum Ketones	+	++	+++	Small Amount
Serum Osmolality*	Variable	Variable	Variable	>320mOsm/kg
Anion Gap**	>12	>12	>12	Variable

Table 1: Criteria for the diagnosis of DKA and HHS.
*Serum Osmolality = ((2 x [Na]) + (BUN/2.8) + (glucose/18))
**Anion Gap = ([Na] – ([Cl]+[CO2])

In DKA, there is a relative or absolute insulin deficiency and an increase in counter-regulatory hormones (*e.g.* glucagon, corticosteroids). DKA typically occurs in type I diabetics, as they are unable to produce any insulin. Utilization of glucose, a primary macronutrient, requires insulin as a cofactor. Therefore, when glucose cannot be utilized by patients due to the lack of insulin, the body will consume free fatty acids for energy. The breakdown of fatty acids results in the production of ketones. The anion gap metabolic acidosis in DKA comes from the accumulation of these ketones (mainly beta-hydroxybutyrate and acetoacetate), which are acids. Patients with DKA typically present with nausea, vomiting, abdominal pain, and rapid respiratory rate due to the metabolic acidosis caused by these ketones. Serum bicarbonate is decreased in DKA due to metabolic compensation of the acidosis.

The primary treatment for DKA is the provision of exogenous insulin to decrease free fatty acid metabolism and ketone production. Insulin therapy should be continued until the anion gap is normal, and patients may require dextrose in their IV fluids to maintain normal glucose values if the anion gap remains greater than 12 despite a normal serum glucose.

Conversely, patients with HHS are typically type II diabetics and generally still produce some insulin at baseline. Since only minimal levels of insulin are required to suppress lipolysis, patients with HHS typically have sufficient insulin to prevent the formation of ketones. As such, patients with HHS typically do not have a significant anion gap metabolic acidosis, and serum bicarbonate will often be normal. Patients with HHS typically present with profound hyperglycemia (glucose >600 mg/dL). This causes an elevated serum osmolality, which can lead to fluid shifts between the body and the central nervous system, resulting in severe symptoms including altered mental status, coma, and possibly death. More commonly, the profound hyperglycemia causes an osmotic diuresis, causing patients to present with symptoms consistent with severe dehydration. Treatment of HHS involves fluid resuscitation, and these patients may not require any additional insulin at all.

Due to the considerations above, it is important for clinicians to determine how much free water loss a patient has had before choosing a fluid for resuscitation. To do so, a corrected sodium should be calculated, as hyperglycemia produces a falsely low serum sodium level due to dilution. If a patient presents with normal or elevated sodium, it can be assumed the patient will have a high serum osmolality, as they will be hypernatremic once their sodium is corrected. The formula for corrected serum sodium level is:

$$Na + 1.6 \times ([glucose] - 100/100)$$

After a corrected sodium concentration is ascertained, a free water deficit should be calculated. A free water deficit is calculated using the following equation:

$$0.6 \times patient\ weight\ in\ kg \times \{([corrected\ Na]/[desired\ Na])-1\}$$

Booze & Blood:

From B Pod to the OR

Jimmy Summers, MD
University of Cincinnati R2

History of Present Illness

The patient is a male appearing to be in his 40s presenting by EMS with what is reported as alcohol intoxication. EMS was called for a patient found down near a bar and reported to "smell of alcohol." EMS reports that the patient was making crude gestures en route to the hospital. On presentation to the Emergency Department, the patient opens his eyes to voice, but does not follow commands and is unable to provide any information as to what happened. No past medical or social history is able to be obtained due to the patient's altered mental status.

Vitals

T 36.5   HR 58   BP 148/90   RR 9   SpO2 93% on RA

Physical Exam

The patient is a well-developed male lying in bed in mild distress. He does not smell of alcohol. The patient has evidence of an abrasion to his right ankle, left knee, and right forearm without any other signs of trauma or deformities.

On neurologic exam, the patient opens his eyes to voice but is noted to have a dysconjugate gaze. Pupils are reactive (4mm to 3mm) and equal. He does localize briskly across midline to painful stimuli with all four extremities but does not follow commands. He is non-verbal and does not respond to questions with any vocalization. His strength and coordination cannot be assessed due to mental status. The patient's GCS is 9 (E3V1M5). The remainder of the exam is unremarkable.

Labs and Imaging

Glucose: 130
WBC: 17.5   H/H: 17.5/50.2
Ethanol level: negative
Salicylate, acetaminophen levels: negative

CT Head without contrast:
There is a large left subdural hematoma with a 9mm left-to-right midline shift with uncal herniation. Scattered areas of subarachnoid hemorrhage.

Hospital Course

The patient presented with concern for intoxication. On examination, he had a dysconjugate gaze, some minor abrasions on his lower extremities, and no other signs of trauma. However, his mental status and physical exam did not appear congruent with the EMS report. Based on this, there was concern for a medical cause for his altered mental status, so a broader workup was initi-

ated, including blood work and a CT scan of the head. The CT scan showed evidence of a large subdural hematoma with midline shift.

Both trauma and neurosurgery were immediately consulted. Additional cross-sectional imaging was negative for any additional traumatic injuries. The patient was then taken directly to the OR for emergent decompressive hemicraniotomy.

He was transferred to the Neuroscience Intensive Care Unit for further management. On ICU day 1, the patient self-extubated and was weaned to room air. However, on ICU day 4, the patient had an acute decline in mental status and became hypoxic and tachycardic.

Figure 1: CT scan of the head showing a large left-sided subdural hemorrhage.

He was reintubated and transported to the OR where he was found to have reaccumulated a subdural hemorrhage and developed a new epidural hemorrhage. Despite reevacuation, the patient continued to have a low GCS postoperatively. Due to his very poor prognosis, the patient's family decided to withdraw support on ICU day 11 and the patient subsequently expired.

Discussion

Alcohol use is implicated in up to 12% of all ED visits yearly, with uncomplicated alcohol intoxication comprising more than 600,000 visits each year.[1] EMS frequently transports intoxicated, altered patients to the ED, and determining an appropriate workup can be difficult. During a busy shift, it can be tempting to attribute a diminished mental status to simple alcohol intoxication. Many of these patients cannot provide a history and collateral information may be poor, making it important to consider a broad differential, including toxidromic, traumatic, metabolic, and infectious etiologies. In the above case, the patient's presentation was not completely consistent with alcohol intoxication, and the patient's ethanol level was ultimately negative. His large subdural hematoma (Figure 1) was the cause of his diminished mental status, and required emergent operative intervention.

Traumatic brain injury (TBI) is one of the leading causes of death and disability in the United States. It covers a wide clinical spectrum, from a mild concussion to more severe presentations, such as the patient discussed above. TBIs include subdural hematomas, epidural hematomas, traumatic subarachnoid hemorrhages, cerebral contusions, diffuse axonal injury, and penetrating injuries, such as from gun shot wounds. In the United States alone, over one million Emergency Department visits each year can be attributed to TBIs, making their management of significant importance for the ED physician.[2]

Initial management of patients with a known or suspected traumatic brain injury (TBI) should follow the ABCs by ensuring adequate oxygenation, ventilation, and circulation. Rapid sequence intubation is safe in patients with a TBI, although the act of intubation itself can cause complications specific to these patients and thus should be thoughtfully performed based on the clinical scenario of the individual patient. If a patient requires intubation, it is important to obtain an expedited neurologic exam before sedation. However, this should not delay intubation if the patient is hypoxic or failing to protect their airway. Additionally, hypoxemia ($PaO_2 < 60mmHg$) is a known cause of secondary brain injury, and independently increases morbidity and mortality for patients presenting to the ED with a TBI.[3] As such, if a patient is being in-

tubated for a depressed mental status or their expected clinical course alone, care should be taken to avoid any iatrogenic hypoxemia during the intubation.

The act of laryngoscopy and subsequent tracheal manipulation can cause adrenergic stimulation and a resultant reflex tachycardia and hypertension. For the patient with a TBI, this can cause an increase in intracranial pressure (ICP), leading to morbidity and mortality. Pretreatment with lidocaine to attenuate this rise in ICP prior to intubation is somewhat controversial and is not routinely recommended.[4]

Post-intubation sedation medications, such as fentanyl and propofol, are routinely recommended for patients with a TBI. By treating pain, analgesic medications reduce the expected hypertensive and tachycardic response, thereby blunting any rise in ICP that could be attributed to pain or agitation.[5] Additionally, in patients with signs of increased ICP, the head of the bed should be raised to 30 degrees immediately post-intubation.

Post intubation ventilatory management is critical for patients with TBI. Historically, it was taught that patients with increased ICP should undergo prophylactic hyperventilation. As a patient's $PaCO_2$ decreases, cerebral chemoregulators cause a reflex decrease in

cerebral blood flow. This can cause a decrease in ICP by up to 25%. However, if a patient is persistently hyperventilated, the lack of cerebral blood flow actually increases morbidity and mortality. Thus, routine hyperventilation of TBI patients is not recommended. Conversely, hypoventilation and subsequent hypercapnea can lead to cerebral vasodilation and subsequent cerebral edema. Ultimately, a normal $PaCO_2$ of 35 - 45 mmHg should be targeted in ventilated patients with a TBI,[3] making end-tidal CO2 monitoring in the ED important in these patients.

However, if a patient is showing signs of active herniation or rapid change in exam, hyperventilation can be an effective yet temporary strategy to decrease ICP. In a similar vein, current guidelines support the use of mannitol for TBI patients with signs of increased ICP, and while likely equally effective, more evidence is needed to clarify the precise role of hypertonic saline in patients with traumatic brain injuries.[3,5]

Hemodynamic monitoring and blood pressure control is also critical for patients with a traumatic brain injury in the Emergency Department. A patient's cerebral perfusion pressure (CPP) is dependent upon mean arterial pressure (MAP) and ICP, and is defined as: CPP = MAP - ICP. Thus, hypotension causes a decrease in cerebral blood flow, which can worsen brain ischemia. For TBI patients, hypotension is defined as a single blood pressure reading < 90 mmHg systolic, and is independently linked to increased mortality and poor neurologic outcomes.[3]

In the Emergency Department, a patient's ICP and CPP are not immediately known, as this requires the placement of invasive monitoring devices. As such, the ED physician should avoid hypotension with aggressive resuscitation based on assessments of the patient's volume status and potential etiologies for hypovolemia. Patients with poly-trauma, for example, may require blood products for adequate resuscitation. Ultimately, pressor support may be required. This is especially important during and after intubation, where sedation and other medications can lead to a period of iatrogenic hypotension.[3,5]

Patients with known TBI should receive laboratory

Emergency Department Management of Patients with TBI

- Avoid hypoxemia

- Target eucapnia (35-40 mmHg)

- Avoid hypotension

- Treat signs of increased intracranial pressure

- Reverse coagulopathy

Table 1: Key recommendations for Emergency Department management of patients with severe TBI.

Quick Hit: Fungal Sinusitis

Jeremy Liebman, MD
University of Cincinnati R2

History of Present Illness

The patient is a middle-aged male with past medical history significant for HIV on antiretrovirals (last CD4 count in the 600s) and poorly controlled diabetes mellitus who presented with rhinorrhea and facial pain.

The patient's symptoms started four days ago. He describes intermittent, thick, yellow rhinorrhea from the left nare over this time period associated with headaches, anterior facial pain and tenderness to palpation of his face on the left. He reports subjective fevers, but denies chills, sore throat, cough, shortness of breath or chest pain. No recent travel. He reports compliance with his antiretroviral medications but admits to difficulty controlling his blood sugars at home.

Vitals

T 37.5   HR 87   BP 148/90   RR 16   SpO2 98% on RA

Physical Exam

The patient is an alert, well-developed male in no acute distress. HEENT exam is notable for scant yellow, mucopurulent discharge from the left nare. Auricular examination is unremarkable. He does have tenderness with palpation of the maxillary and frontal sinuses on the left. There is no proptosis. Cardiac and pulmonary exams are unremarkable. Neurologic exam is unremarkable.

Labs

Glucose: 170
WBC: 16   CRP: 59.2   Lactate 2.3
Hemoglobin A1C: 14.4

Imaging

Head CT: Opacification of the left maxillary, frontal, and sphenoid sinuses and ethmoid air cells with heterogenous and hyperdense material suspicious for fungal sinusitis.

Hospital Course

ENT was consulted given the concern for fungal sinusitis. Bedside flexible laryngoscopy was performed in the Emergency Department and revealed thick mucoid secretions in the left nasal cavity. The patient was admitted to the hospital, and it was determined that the patient would undergo functional endoscopic sinus surgery (FESS) with ENT.

The patient underwent FESS and was noted to have a large burden of left-sided sinusitis. A left maxillary antrostomy and left sphenoidotomy with removal of tissue from both sinuses was performed, as well as left total ethmoidectomy and left frontal sinusotomy. There was low clinical suspicion for mucormycosis given a lack of obvious necrosis. Nevertheless, the patient was started on amphotericin B empirically. Surgical pathology revealed mucosa with chronic inflammation, moderate eosinophilia, and fibrosis.

Ultimately, cultures grew out Alternaria species. Amphotericin was discontinued, and the patient was started on a six to 12 month course of itraconazole. The patient was discharged to a SNF and subsequently home with home health care with continued antifungal coverage. He continues to have difficulty managing his diabetes medication and maintaining adequate control of his blood sugar.
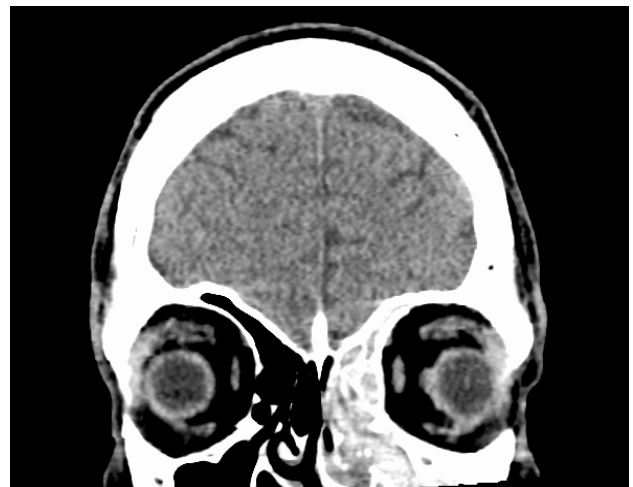

Figure 1: Coronal slice of a head CT showing fibrous debris in the left sinus, consistent with fungal sinusitis.

Discussion

Fungal sinusitis is commonly divided into five categories: acute necrotizing (fulminant), chronic invasive, chronic granulomatous invasive, fungal ball, and allergic. The former three of these are considered invasive, whereas the latter two are considered non-invasive.[1] Invasive fungal sinusitis implies the presence of fungal hyphae in the mucosa, submucosa, blood vessels, or bone of the paranasal sinuses, whereas noninvasive sinusitis lacks the presence of fungal hyphae in the mucosa or tissues.[2]

Acute necrotizing fungal sinusitis generally occurs in immune-compromised patients, including diabetic patients, patients with HIV/AIDS, and those patients receiving cytotoxic or immune-suppressing drugs. The incidence of this disease is difficult to ascertain, given
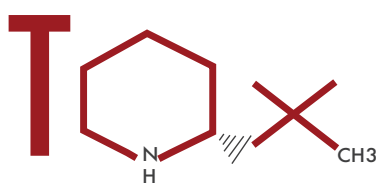
Tox Talks:

Salicylate Toxicity

Matt Riddle, MD
University of Cincinnati R4

History of Present Illness

The patient is a middle-aged male with a history of hypertension and gastroesophageal reflux disease who presented to the Emergency Department for acute worsening of his chronic tinnitus over the past day. He complains of constant ringing in his ears that has become much worse than baseline over the past 24 hours. He bit his tongue three days ago and has been using aspirin for the pain, taking about four to six tabs of 325 milligram aspirin around three times daily. He also notes some nausea without vomiting and light-headedness associated with his tinnitus. He denies visual changes, shortness of breath, chest pain, trauma, loss of consciousness, suicidal ideation, or confusion. He denies taking any other medications.

Vitals

T 37.1   HR 90   BP 156/98   RR 18   SpO2 98% on RA

Physical Exam

The patient is an alert, well-nourished male in no apparent distress. HEENT exam is unremarkable. There is no nystagmus. Extraocular movements are intact. Tympanic membranes are normal in appearance bilaterally. There is a small mucosal erosion of the right lateral tongue without active bleeding. Cardiopulmonary exam is normal. His abdomen is soft and nontender. He is alert and oriented with a nonfocal neurologic exam. He has a normal mood and affect and denies suicidal or homicidal ideation.

Labs

Venous Blood Gas: pH 7.46/pCO2 35/BE 1.2
BMP: 141/3.6/110/23/19/1.06/122
Salicylate Level: 52 mg/dL

Hospital Course

The patient was diagnosed with chronic salicylate toxicity and started on a sodium bicarbonate drip. He was admitted to the medicine stepdown unit for further management. He had resolution of his nausea and tinnitus. Serial salicylate levels were trended until levels fell within the therapeutic range, and repeat venous blood gases demonstrated resolution of his respiratory alkalosis. He was discharged the day after admission and suffered no long-term sequelae.

Discussion

Salicylates are among the oldest therapeutics still in clinical use. Both acute and chronic salicylate poisoning remain common disease processes, making salicylate toxicity a must-know disease process for emergency providers.[1]

Sources for salicylate toxicity can include aspirin (acetylsalicylic acid), oil of wintergreen (methyl salicylate), salicylic acid (used as topical keratolytic and wart remover), Pepto-Bismol (bismuth subsalicylate), and topical preparations (*e.g.* Aspercreme), as well as combination medications that contain aspirin, such as Excedrin. Early signs and symptoms of salicylate toxicity include tinnitus, vertigo, nausea, vomiting, and diarrhea. Later signs that indicate more severe toxicity include altered mental status, noncardiogenic pulmonary edema and seizures.[2]

How To Make a Bicarbonate Drip

Add 3 amps of sodium bicarbonate to 1 L of D5W. Run this at 150-200 mL/h.

Salicylates have multiple mechanisms of toxicity. From an acid-base perspective, they directly stimulate the respiratory center of the medulla, causing a primary respiratory alkalosis. They also interfere with cellular respiration via multiple pathways, which leads to metabolic acidosis. Salicylates may cause alterations in mental status through direct toxicity, by decreasing cerebral glucose levels or by inducing cerebral edema. Acute lung injury and noncardiogenic pulmonary edema occur more often in elderly patients and patients with chronic toxicity. Finally, inhibition of cyclooxygenase can cause gastric mucosal injury and platelet dysfunction.[3,4]

There is significant overlap in the signs and symptoms of acute and chronic salicylate toxicity, including tinnitus, altered mental status, and acid-base disturbances, although there are some key differences (Table 1). An accurate history and physical exam can suggest salicylate poisoning, and can be supported by the presence of acid-base disturbances. Serum salicylate levels above 40 mg/dL typically correlate with toxicity. It is important to remember that levels may not begin to rise in acute toxicity until 5-6 hours after ingestion secondary to bezoar formation, enteric-coated tablets, and pylorospasm.

As with all poisoned patients, the treatment of aspirin toxicity begins with the ABCs. For patients with salicylate toxic-

The Two Midnight Rule

Daniel Axelson, MD
University of Cincinnati R4

You've heard of it. You've heard complaints about it. Heck, you've probably even certified a patient as "two-midnights" yourself. But what is the Two-Midnight Rule, and what does it mean?

Announced in 2013, the Two Midnight Rule was created by the Centers for Medicare and Medicaid Services (CMS) to clarify billing issues for Medicare patients. CMS pays for inpatient hospital stays through Medicare Part A and outpatient visits through Medicare Part B, making them very different entities with different reimbursement schedules.

A patient admitted to the hospital as an inpatient is charged based on something called a Medicare Severity Diagnosis-Related Groups (MS-DRG). This is a bundled lump sum payment, and is determined by CMS to be the amount that anyone with a similar diagnosis, such as a patient with chest pain, should cost to take care of (See? Those admitting diagnoses really do matter!). This is a set rate, and hospitals do not get paid more even if they provide additional services to a patient.

In contrast, a patient who presents for outpatient-level care, such as same-day surgery or an Emergency Department visit, is paid for via a Ambulatory Payment Classification (APC). Health systems still receive lump-sum payments based on the expected cost of taking care of a particular APC, but on the outpatient side, health systems can apply for multiple APCs on a patient and itemize more costs than on the inpatient side. Observation unit stays are billed under this system. However, Medicare typically pays more for inpatient admissions than for outpatient admissions, making this designation of financial importance to CMS.

Historically, hospitals had extensive discretion in classifying patients as inpatient or outpatient. In an effort to ensure proper payments, CMS enacted the "two-midnight benchmark," which for the first time mandated specific guidelines regarding what CMS viewed as appropriate conditions for inpatient admission to the hospital. Stays anticipated to span longer than two midnights were deemed appropriate for admission, and stays less than this were deemed inappropriate admissions and would not be reimbursed through Medicare Part A.

Shocker statement of the column: hospitals didn't love this. The timeframe is felt to be arbitrary, and is thought to not fully capture the complexity of the decision to admit a patient to the hospital. There is also criticism that the Two Midnight Rule offers a disincentive to hospitals for innovations that may increase the efficiency and reduce the length of time needed to offer inpatient-based care.

Medicare Recovery Audit Centers are responsible for monitoring inpatient admissions, and can still reject inpatient reimbursement claims if they deem the admission inappropriate. Interestingly, their funding comes from a portion of the recovered claims from rejected inpatient reimbursement stays. Conflict of interest? Certainly a questionable incentive. Meanwhile, observation unit stays continue to increase as hospitals set about the increasingly burdensome task of optimizing both care of patients and recovery of reimbursements.

While the Two Midnight Rule is currently undergoing review, as it stands now, every physician admitting a patient to the hospital must certify that the patient's anticipated length of stay is greater than two midnights.

https://www.healthaffairs.org/healthpolicybriefs/brief.php?brief_id=133

https://www.cms.gov/Newsroom/MediaReleaseDatabase/Fact-sheets/2015-Fact-sheets-items/2015-07-01-2.html

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declining autopsy rates, as well as empiric treatment of patients without histological or microbiological proof of infection.[3] Interestingly, of immunecompromised patients, HIV patients are less commonly infected, although they still remain at risk.[4] Mucor and Rhizopus species are commonly implicated. Patients may present with cough, fever, purulent nasal discharge, epistaxis and headache. Tissue samples will reveal hyphae and often reveal significant tissue necrosis. Speciation requires successful culture of the organism.

Treatment includes surgical debridement, intravenous Amphotericin B, and, to the extent possible, resolution of the underlying immunodeficiency.[5] Diagnosis is critical, as untreated invasive fungal sinusitis has a reported mortality rate of between 50 and 80%.[6] Our patient's culture was ultimately positive for Alternaria, a fungal species commonly found both in soil as well as indoor environments. The findings of chronic inflammation and eosinophilia suggest an allergic versus chronic invasive nature to the patient's sinusitis, rather than acute necrotizing sinusitis. Although HIV+, our patient had a CD4 count greater than 600. As such, the patient's poorly controlled diabetes (HbA1C > 14) was the likely causative agent underlying the patient's infection.

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This equation will give you the free water deficit in liters. Assessing free water deficit in patients with HHS is crucial, as correcting hypernatremia too quickly can lead to cerebral edema. No more than half the free water deficit should be corrected within the first 24 hours, and serum sodium should be checked as frequently as every 4 hours if the free water deficit is significant. The free water content of common intravenous fluids is listed in Table 2.

With appropriate replacement of free water, patients with HHS may correct their hyperglycemia without any exogenous insulin therapy or require less insulin than a DKA patient. However, depending on the sever-

A typical [desired Na] would be 145mEq/L.

Fluid	Amount of Free Water in 1 Liter
0.9% Saline	0mL
0.45% Saline	500mL
0.225% Saline	750mL
Dextrose or Sterile Water	1000mL

Table 2: Free water content of commonly used fluids. Patients with HHS should be initially resuscitated with normal saline and should not be switched to 0.45% saline too quickly to avoid risk of cerebral edema.

ity of insulin resistance, some patients may still require exogenous insulin to maintain appropriate serum concentrations. Often restarting the patient's home insulin regimen is adequate treatment for these patients.

In sum, DKA and HHS are two clinically unique presentations of hyperglycemia and are treated based on their underlying pathology. Rapid recognition of these disease processes in the Emergency Department is a critical first step in the treatment of both of these disorders.

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ity, intubation should be reserved for true respiratory failure. The brief period of apnea during intubation and decreased minute ventilation following intubation can lead to a respiratory acidosis, promoting a shift of salicylates into the CNS, increasing toxicity. From a cardiovascular perspective, aspirin-poisoned patients are often volume-depleted. Supplemental fluids are indicated unless the patient has known pulmonary or cerebral edema. Hypotension not responsive to fluids can be treated with a vasopressor, such as phenylephrine or norepinephrine.

Activated charcoal actively absorbs aspirin, and at least one single dose should be given to awake and cooperative patients (as well as intubated patients) if presenting within 2 hours of ingestion. Multiple-dose activated charcoal decreases serum salicylate levels and should be considered for the same patient population.[5]

The mainstay treatment for salicylate toxicity is alkalinization with sodium bicarbonate. This decreases diffusion of salicylate into the CNS and increases renal elimination. A sodium bicarbonate drip should be used and titrated to a goal urinary pH of 7.5-8.0. Salicylic acid exists in two forms: an uncharged protonated form and a negatively charged anion. The deprotonated anion is not reabsorbed in the renal tubule. By increasing the urinary pH, the proportion of charged molecules is increased. This "traps" the ion in the urine and enhances urinary

Acute vs Chronic Salicylate Toxicity

	Acute	Chronic
Age	Younger	Older
Etiology	Often intentional overdose	Accidental, iatrogenic
Concomitant Disease	None	Often underlying chronic pain disorders
Suicidal Ideation	Typical	Usually absent
Serum Concentration	Marked elevation	Intermediate elevation
Mortality	Uncommon	More common due to missed diagnoses

Table 1: Signs and symptoms of acute versus chronic salicylate toxicity.

elimination of aspirin and clearance from the body.[6] Effective alkalinization relies on the kidney exchanging potassium for hydrogen, so hypokalemia must be treated aggressively. Salicylates are also readily removed by dialysis, with intermittent hemodialysis being the method of choice.[7] Indications for dialysis include altered mental status, cerebral edema, salicylate level > 100 mg/dL, acute or chronic kidney injury (serum creatinine > 2.0 in adults or 1.5 in elderly patients or those with low muscle mass), respiratory distress requiring supplemental oxygen, severe acidemia (pH ≤ 7.20), or failure of standard supportive therapy.

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small amounts of dopamine. Pheochromocytomas are catecholamine-secreting neoplastic versions of these cells.

The clinical manifestations of such tumors are highly variable as they have varying degrees of catecholamine secretion and receptor response, yet the overall mortality from these tumors is about 15%.[2] Many patients may be completely asymptomatic for years prior to a formal diagnosis, but some will experience the classic paroxysms of headaches, diaphoresis, and palpitations in the setting of a significantly elevated blood pressure. Weight loss and intermittent episodes of significant anxiety are also commonly reported.

There are case reports of pheochromocytomas secreting vasoactive intestinal peptide (VIP) in addition to catecholamines. These patients often present with profound watery diarrhea in addition to the typical symptoms, which may explain the initial clinical presentation of the above patient. If catecholamine secretion is severe, the patient's presentation may be life-threatening, and the need to rapidly identify the correct diagnosis even more crucial. Acute cardiomyopathy, hypertensive emergency, significant hyperthermia, and shock are just some of the various clinical presentations of patients with these lesions.

This diagnosis should be considered in the Emergency Department whenever a patient with a known adrenal incidentaloma presents with the characteristic symptoms of a pheochromocytoma. The triad of headache, diaphoresis, and palpitations has a positive likelihood ratio of 6.3, regardless of the presence of hypertension, and may be a useful clue in making the diagnosis.[3] Patients with severe hypertension or hemodynamic instability with no apparent etiology or hypertension that worsens with administration of beta-blockers should also prompt consideration of the diagnosis. Those with

familial syndromes such as neurofibromatosis and multiple endocrine neoplasia are particularly susceptible to developing these as well.

Laboratory data may reveal hyperglycemia, hypercalcemia, and erythrocytosis. While urine metanephrines are not a routine Emergency Department test, an abdominal CT often is. The literature suggests that in unstable patients in whom pheochromocytoma is even suspected, an abdominal CT
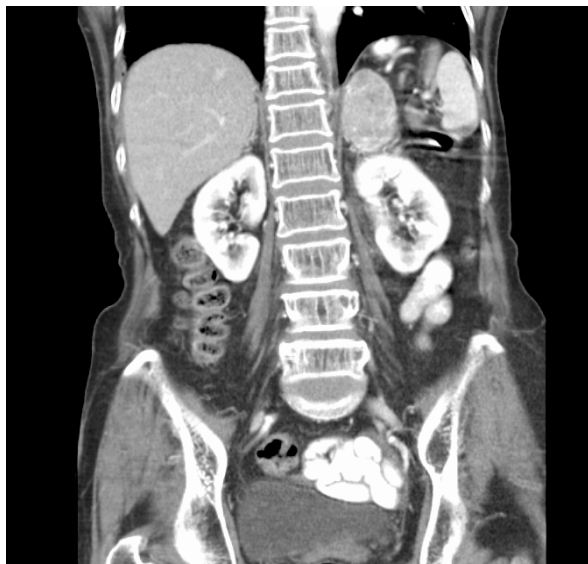

Figure 1: CT scan of the abdomen showing a left-sided adrenal mass.

is an acceptable diagnostic tool, as patients with neoplasms secreting enough catecholamines to cause hemodynamic instability will have radiographic evidence of the lesion. While it is not a diagnosis that is typically made in the Emergency Department, early workup may help expedite discovery of the diagnosis if there is a high enough index of suspicion.

Hemodynamically stable patients with suspected pheochromocytoma are appropriate for outpatient work-up, while more unstable patients will benefit from inpatient diagnostic testing and management of their symptoms. These patients should be admitted to a medicine service who can manage the various consulting teams that will be necessary, from endocrinology for

symptom control to urology for tumor removal. The treatment of these lesions primarily involves managing the severe hypertension until surgical removal of the mass can be performed. Alpha blockade with agents such as phentolamine or terazosin is the primary treatment.[4] Beta blockers are contraindicated for these patients because they may worsen hypertension due to the unopposed alpha effects of the circulating catecholamines.

There is no specific blood pressure target for these patients, and pheochromocytoma-mediated hypertension should be managed in the ED similarly to other patients with hypertensive emergency. There is very little data on initiation of alpha blockade for treatment of pheochromocytoma in the Emergency Department. However, the US Endocrine Society guidelines indicate that patients with known pheochromocytomas should receive initiation of alpha blockade as soon as possible, regardless of the presenting blood pressure.[5] This is to help mitigate the unpredictable possibility of developing hypertensive crisis in these patients.

Therefore, it would be appropriate to start a low-dose alpha blocker in the ED when this diagnosis is made. Given the chronic vasoconstriction due to catecholamine excess, fluid resuscitation is also important, as many of these patients are hypovolemic. Optimization of hemodynamics with these interventions should be undertaken prior to surgical intervention, as it leads to improved post-surgical morbidity and mortality.[2]

Ultimately, this is a rare diagnosis which can lead to any number of presenting complaints in the Emergency Department. It has an overall mortality of 15%, although this can be improved by earlier diagnosis. As such, the next time an adrenal incidentaloma is buried in the radiology read of an abdominal CT scan, consider adding pheochromocytoma to your differential diagnosis.

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testing to evaluate their coagulation status in order to stratify the risk of hemorrhagic progression. A typical order set would include a CBC, platelet function assays, and coagulation factor assays. Platelets should also be quantitatively measured with a CBC to assess for any thrombocytopenia that may contribute to worsening of their bleed.

In general, patients with severe thrombocytopenia (platelets <50k) with intracranial hemorrhage should receive a platelet transfusion.[6] Platelets should also be qualitatively evaluated with platelet function assays, such as a TEG, an aspirin function test, P2Y12 function test, or a bleeding time, especially in patients taking antiplatelet medications. However, there is some debate on the utility of platelet transfusions in patients with nonfunctioning platelets due to little evidence to suggest improvement in clinically significant outcomes.[7]

Given the common use of oral anticoagulants in certain patient populations, patients with a TBI should also receive a PT/INR and aPTT in order to further assess their coagulation cascade and need for potential reversal. Depending on the agent or etiology implicated in a patient's coagulopathy, many options exist for the reversal of coagulopathy, including vitamin K, protamine, fresh frozen plasma (FFP), and prothrombin complex concentrates (PCCs), as well as medication-specific reversal agents.

In general, surgical management of TBI is dictated by the type of lesion (Table 2).[8,9] Our patient had a subdural hemorrhage with 9 mm midline shift and was therefore a candidate for emergent surgical decompression and evacuation. However, not all traumatic brain injuries will be managed with aggressive interventions. Neurosurgical consultants will often recommend serial neurologic exams and a repeat head CT at six hours for patients diagnosed with new onset intracranial hemorrhage who do not meet criteria for emergent surgical or medical intervention.

In sum, patients who present with altered mental status may have a variety of medical issues, ranging from simple alcohol intoxication to a neurosurgical emergency. Emergency physicians must maintain a high degree of clinical suspicion so as not to miss reversible causes of altered mental status, such as an acute subdural hematoma. For patients with traumatic brain injuries, along with neurosurgical consultation, the management of the patient's airway, blood pressure and coagulation status are key acute interventions initiated in the Emergency Department.

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Indications for Surgical Intervention in Traumatic Brain Injury

Epidural hematoma	Subdural hematoma	Traumatic parenchymal lesion	Posterior fossa mass lesion	Depressed skull fracture
Volume of bleed >30 cc

>15 mm thickness

>5 mm midline shift

GCS < 9

Anisocoria | >10 mm thickness

>5 mm midline shift

GCS < 9

Neurologic deterioration | Volume of bleed >20 cc

>5 mm midline shift

Cisternal compression

Neurologic deterioration

Refractory intracranial hypertension | Distortion of the 4th ventricle

Effacement of basilar cisterns

Obstructive hydrocephalus | >1 cm depression with open fracture

Dural penetration

Intraparenchymal hematoma

Frontal sinus involvement

Pneumocephalus |

Table 2: Indications for surgical intervention in patients with TBIs. Adopted from Summary of Guidelines for the Surgical Management of Traumatic Brain Injury, 2006.
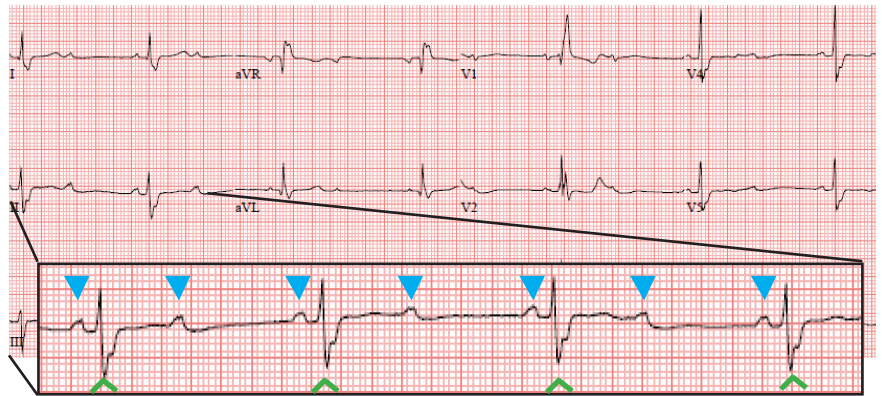
Complete Heart Block

E*KG focus

Grace Lagasse, MD
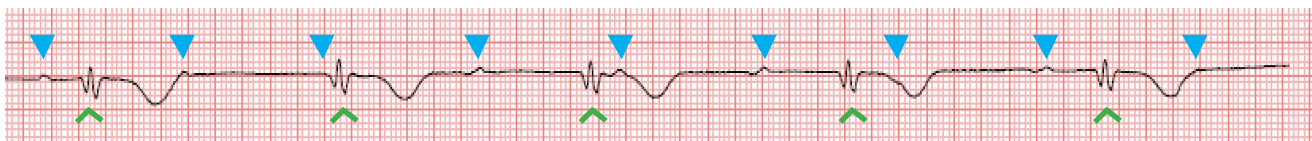**University of Cincinnati R3**

EKG 1. Note the regularity yet lack of relationship between the atrial contractions (blue closed arrow) and ventricular contractions (green open arrow). This patient also exhibits a 2:1 complete heart block. There are two atrial contractions for every ventricular contraction.

History of Present Illnesses

In EKG 1, a male in his 60s presented complaining of exertional chest pain. In EKG 2, a female in her 60s presented complaining of syncope and weakness. She has a history of pacemaker placement for heart block but was unsure when the battery was last changed.

Complete Atrioventricular Conduction Block

Complete atrioventricular conduction block, also known as complete heart block or third degree heart block, occurs when there is complete failure of conduction between the atria and ventricles. This results in independent contractions of the atria and ventricles. P waves and QRS complexes occur without any relationship to each other.

EKG 2. Note the regularity yet lack of relationship between the atrial contractions (blue closed arrow) and ventricular contractions (green open arrow). Due to the dissociation between the atrial and ventricular contractions, some of the P waves are buried within the strip.

Treatment

Stable patients may respond to atropine. Unstable patients, those who are hypotensive, or those with altered mental status should be treated with transcutaneous pacing. If transcutaneous pacing is unsuccessful, a transvenous pacer should be placed. All patients with complete AV block should be admitted for permanent pacemaker placement. If the etiology of complete heart block is due to acute MI then the patient should be immediately taken to the cath lab.

EKG and Case referred by

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Common Etiologies of Complete Atrioventricular Conduction Block

- Coronary artery disease — acute coronary syndrome or acute MI
- Degeneration of the conduction system
- Infectious etiologies — myocarditis, rheumatic fever, Lyme disease
- Connective tissue diseases — rheumatoid arthritis, scleroderma
- Infiltrative disorders — amyloidosis, sarcoidosis, Hodgkin's
- Traumatic — septal ablation, cardiac contusion, surgical trauma
- Congenital

TO WATCH HOW TO PLACE A TRANSVENOUS PACER VISIT:
HTTP://WWW.TAMINGTHESRU.COM/TRANSVENOUS-PACEMAKER/

1. Abedin, Zainul. Essential Cardiac Electrophysiology : The Self-Assessment Approach (2). Somerset, GB: Wiley-Blackwell, 2013. ProQuest ebrary. Web. 23 August 2016.
2. Tintinalli, Judith E, Gabor D. Kelen, and J S. Stapczynski. Emergency Medicine: A Comprehensive Study Guide. New York: McGraw-Hill, Medical Pub. Division, 2004. Print.

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 make sure to include the R1/R4 involved in the case, a brief synopsis and a patient sticker
annalseditors@gmail.com

List of Submitted B Pod Cases

Case	Case Physicians
Flexor tenosynovitis	Loftus/Whitford
AIDP	Loftus/Bell
New TB diagnosis	Kircher/Zimmerman
Fungal sinusitis	Loftus/Liebman
Multinodular goiter	Campbell/Burbage
Wellens	Lafollette/O'Neal
"ETOH"/Subdural hemorrhage	Selvam/Summers