



ANNALS OF B POD

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VOLUME IX ISSUE II
WINTER ISSUE 2015

SEEING

DOUBLE

P A G E 4

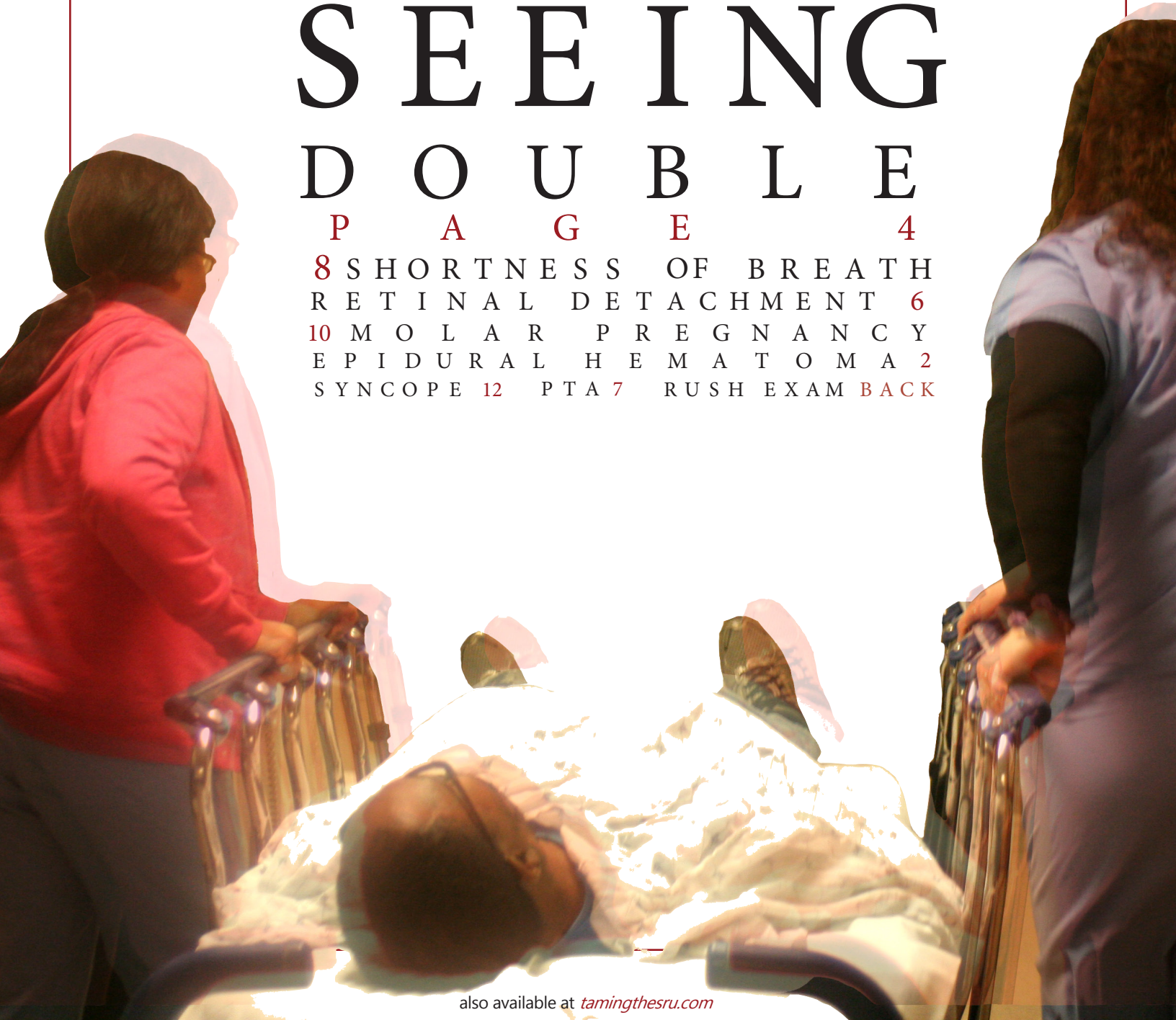
8 SHORTNESS OF BREATH

RETINAL DETACHMENT 6

10 MOLAR PREGNANCY

EPIDURAL HEMATOMA 2

SYNCOPE 12 PTA 7 RUSH EXAM BACK



Headache ...plus...

#sickornotsick

Julie Teuber MD
 University of Cincinnati R1

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It has been a busy winter

Flu season is upon us and every day we face the question of how far to take the clinical evaluation when we see generic complaints. The decision comes partly from objective data, partly clinical decision rules, and mostly from an unquantifiable summation that is clinical gestalt. This issue we feature some complex cases with simple chief complaints, emphasizing the point that the sickest patients evolve from the mundane. #sickornotsick

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History of Present Illness

The patient is a 24 year old male with no significant past medical history who was brought in to the ED by his father approximately 20 hours after falling out of a taxi while intoxicated. After he fell, the patient went home to "sleep it off." Although the patient reported a hangover the next day, the patient's father was worried because he continued to have vomiting intermittently throughout the day. Friends told the patient they thought he hit his head but he had no recollection of the event and there was no clear history of loss of consciousness. The patient's only complaints were headache, nausea and vomiting. He stated multiple times "Doc I'm fine, I'm just hungover."

Past Medical History

None

Social History

Social alcohol use, denies illicit drugs

Medications

None

Allergies

None

Physical Exam

General: well nourished; well developed; in no apparent distress

HEENT: Head atraumatic, pupils equal, round and reactive to light, extraocular movements intact, sclera clear, mucus membranes moist, oropharynx nonerythematous

Neck: No cervical spine tenderness

Chest: Lungs CTAB, RRR, normal S1, S2

Abdomen: Soft, nondistended, nontender

Extremities: Full range of motion, no tenderness or signs of trauma

Neuro: Alert and oriented X 4, cranial nerves II-XII intact. The patient has a normal gait without ataxia, speech intact. No dysmetria or dysarthria, GCS 15

Temp 99.6
 Heart Rate 96
 Resp Rate 18
 BP 98/74
 O2 Sat 100%

ED + Hospital Course

The patient received a CT scan (Image 1) right revealed a right epidural hematoma. Neurosurgery was consulted and the patient was admitted to the Neuro

ICU. Given that he was a GCS 15 without deficits, he was initially monitored in the ICU. However, early the next morning he was GCS 13 and developed right sided drift. He was taken to the OR for a right frontoparietal craniotomy for epidural evacuation. The patient did well post-operatively and was discharged home with no neurologic deficits.

discussion

The decision to perform a CT on this patient was not an easy one. Even knowing the clinically significant findings, one can see how this patient's work-up was not straight forward. The patient was young, hungover, and had symptoms



Image 1 - CT Head: Large right frontoparietal epidural hematoma with moderate mass effect and mild leftward midline shift. Nondisplaced right parietal and squamous temporal bone fracture.

consistent with a concussion. A diagnosis of concussion or mild traumatic brain injury (TBI) in this case seems reasonable given his presentation and the fact that these are much more common than an epidural hematoma. In this particular case, a combination of gestalt, clinical decision rules, and parental concern were used to make the decision to perform a head CT. Clinical decision rules are not perfect and physician gestalt plays an important role in coming to a decision about how to approach each patient. Typically, emergency providers use one of two decision rules to help identify which mild TBIs need head CTs, either the Canadian (Table 1) or New Orleans (Table 2) Head CT Rule. These rules were developed in an effort to decrease over-testing and avoid radiation while still catching those with clinically significant findings.

If these rules are applied to this patient, he would have been deemed high enough risk to warrant a CT scan with both rules. He met the criteria of alcohol ingestion,

amnesia to the event and vomiting (>2 times for the Canadian Head CT rule) and therefore received a head CT which produced a significant finding that required surgical intervention.

Epidural hematomas due to head trauma have an incidence of approximately 2% of all head injuries presenting to the emergency department. The reported mortality ranges from 5-50% depending on initial GCS, pupil response, motor exam and associated brain injuries on CT. It occurs more frequently in young males. The classic presentation involves head trauma with loss of consciousness, a quick return to baseline consciousness (popularly known as the lucid interval), and an eventual decline in mentation. Epidural hematoma often represents a neurologic emergency that requires prompt surgical intervention to prevent herniation and death. Patients are often taken to the OR for a craniotomy and hematoma evacuation and monitored in ICU setting post-operatively. Any patient with a depressed GCS (<14), focal neurologic deficit attributable to the epidural hematoma, progressively declining mental status or hemorrhage volume > 30 mL are usually taken to the OR. There are however cases, such as our patient, in which the decision is made to treat non-operatively. Patients who have epidural hematoma volume < 30 ml with midline shift < 5mm, a GCS 14 or greater with no neurologic deficits are eligible for non-operative management. These patients

Avoid Secondary Injury

∅ hypoxia

∅ hypotension

treat signs of ↑ ICP
 head elevation 30°
 analgesia/sedation
 3% saline/mannitol

Table 3 - Avoidance of secondary injury in TBI patients. Remember: pupillary abnormality and posturing are indications for mild hyperventilation and there is no indication for empiric or chronic hyperventilation

are admitted to an ICU setting for repeat neurologic examinations and serial head CT imaging. In this case, the patient was observed in the neuro ICU and had a decline in his GCS as well as a focal neurologic deficit, thus the decision was made to take him to the OR.

In addition to neurosurgical intervention, measures should be taken in the ED to prevent secondary brain injury in TBI patients. (See table 3) Resuscitation should be aimed at preventing hypoxia and hypotension while specifically addressing sedation and analgesia as a way to prevent or treat increased ICP.

1- Maharah, I; Tosiello, L. The Canadian CT Head Rule for Patients with Minor Head Injury. *The Lancet*. 2001; 357 (9266): 1391-1396.

2- Stiell, I; Clement, C. Comparison of the Canadian CT Head Rule and the New Orleans Criteria in Patients With Minor Head Injury. *JAMA*. 2005;294(12):1511-1518. doi:10.1001/jama.294.12.1511

3- Miller, E; Derlet, R. Minor Head Trauma: Is Computed Tomography Always Necessary? *Annals of Emergency Medicine*. Volume 27, Issue 3, March 1996, Pages 290-294.

4 - Bouida W, Marghi S, Souissi S, Ksibi H, Methammem M, Haguiga H, Khedher S, Boubaker H, Beltaief K, Grissa MH, Trimech MN, Kerkeni W, Chebili N, Halila I, Rejeb I, Boukef R, Rekkik N, Bouhaja B, Letaief M, Nouira S. Prediction value of the Canadian CT head rule and the New Orleans criteria for positive head CT scan and acute neurosurgical procedures in minor head trauma: a multicenter external validation study. *Ann Emerg Med*. 2013 May;61(5):521-7. doi: 10.1016/j.annemerg-med.2012.07.016. Epub 2012 Aug 22.

New Orleans Head CT Rules CT IF ANY OF THE FOLLOWING ARE PRESENT

- Seizure
- Visible trauma above the clavicles
- Drug or EtOH ingestion
- Headache
- Vomiting
- Age >60
- Short term memory loss anterograde amnesia

SENSITIVITY - 82% SPECIFICITY 26%

Table 2 - Applicable for adults with a GCS of 15 and blunt head trauma within 24 hours that caused LOC, definite amnesia, or witnessed disorientation. In red are presenting symptoms in our patient. Sensitivity and specificity from external validation (Bouida et al 2013)⁴

Canadian Head CT Rules CT IF ANY OF THE BELOW ARE PRESENT

<p>High Risk Features</p> <ul style="list-style-type: none"> GCS<15 at 2 hours after injury Open/depressed skull fracture Basilar skull fracture signs hemotympanum, raccoon eyes, Battles sign CSF Oto/Rhinorrhea Vomiting >= 2 episodes Age >=65 	<p>Medium Risk Features</p> <ul style="list-style-type: none"> Amnesia >30 minutes before impact Dangerous mechanism pedestrian vs auto, ejections, fall from >3 feet or 5 stairs 	<p>Mild Head Injury</p> <ul style="list-style-type: none"> Witnessed LOC, definite amnesia, witnessed disorientation in patients with a GCS of 13-15
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SENSITIVITY - 100% SPECIFICITY 60%

Table 1 - Applicable for adults with a GCS of 15 and blunt head trauma within 24 hours that caused LOC, definite amnesia, or witnessed di In red are presenting symptoms in our patient. Sensitivity and specificity from external validation (Bouida et al 2013)⁴

Double Vision

from peripheral to central: localization is key

Megan Redmond, MD
University of Cincinnati R4

case originally seen by R4
Megan Redmond, MD and
MSIV Todd Vinsant

History of Present Illness

A 78 year old male with past medical history significant for coronary artery disease status post stenting, hypertension, hyperlipidemia, chronic kidney disease presented with complaints of double vision and feeling off balance. The patient stated he woke up that morning with double vision. He reported the sensation of double vision was worse when he looked side to side and completely resolved upon closing one of his eyes. He denied wearing glasses or contact lenses and denied any eye pain or trauma. He also reported feeling off balance, however he denied any focal numbness or weakness of his extremities. He noted an episode of slurred speech approximately 1 hour prior to arrival that has since resolved. He denied any other difficulties with word finding or language. Otherwise patient denied headache, head trauma, neck pain, chest pain, or shortness of breath.

Past Medical History

Coronary artery disease status post LAD stent x 1, Hypercholesterolemia, Hypertension, Chronic Renal Insufficiency, Anemia

Past Surgical History

Coronary stent placement to LAD- 2003
Appendectomy- 1986

Medications

Aspirin, Atorvastatin,
Labetalol

Allergies

No known

Social History

Social alcohol use, denies tobacco, illicit drugs

Temp
97.5
Heart Rate
94
Resp Rate
20
BP
146/76
O2 Sat
97%



CT Head: No evidence of acute intracranial hemorrhage, infarct or acute mass effect. Mild diffuse cerebral atrophy



MRI: Acute right pontine infarct

Physical Exam

General: Well developed, well nourished, non-toxic appearance
Eyes: PERRL, conjunctiva normal. Left eye with full extraocular movements in all directions. No ptosis, hyphema, hypopyon present.
HENT: Atraumatic, external ears normal, oropharynx moist. Normal range of motion, no tenderness, supple
Respiratory: No respiratory distress, normal breath sounds, no rales, no wheezing
Cardiovascular: Normal rate, normal rhythm, no murmurs, no gallops, no rubs,
GI: Soft, nondistended, normal bowel sounds, nontender
Musculoskeletal: No edema, no tenderness, no deformities. Back without tenderness
Integument: Well hydrated, no rash
Lymphatic: No lymphadenopathy noted
Neurologic: Alert & oriented x 3. **Right eye has full lateral abduction, however is unable to adduct his right eye medially past midline when looking left. Left eye with nystagmus with left-**

ward gaze. Accommodation intact. No ptosis, symmetric eyebrow raise, normal smile without facial droop. **Past-pointing with left upper extremity with finger to nose.** Intact 5/5 motor strength in upper and lower extremities, sensation intact upper and lower extremities. Gait unsteady with mild gait ataxia. NIHSS 2 for partial gaze palsy and limb ataxia.
Psychiatric: Speech and behavior appropriate

Hospital Course

The patient presented with acute onset of a partial gaze palsy consistent with a right internuclear ophthalmoplegia and limb ataxia, giving him a NIH stroke scale score of 2. He was not a tPA candidate due to a greater than 4.5 hour time since last seen normal. The patient subsequently admitted to neurology service for further evaluation and management of these symptoms which were concerning for posterior circulation infarct. Imaging studies obtained as an inpatient included an MRI and CTA.

CTA Head/Neck:

70% or greater stenosis proximal left vertebral artery, with 50% stenosis distal right vertebral artery beyond PICA, and 50% stenosis the basilar artery beyond AICA.

Given his stroke in the setting of severe intracranial stenosis, the patient was started on dual antiplatelets agents aspirin and clopidogrel. Symptoms of diplopia and ataxia improved. Interventional Neuroradiology was consulted for treatment of his severe left vertebral artery stenosis. They recommended aggressive medical therapy, with the possibility of intracranial vascular stenting should symptoms recur. The patient was discharged home in stable condition on hospital day 4 with home health and physical therapy resources.

discussion – internuclear ophthalmoplegia

Diplopia is defined as the perception of 2 images of a single object.¹ In the evaluation of diplopia, the first step is to determine if the

patient is demonstrating monocular or binocular diplopia. To test this, have the patient close one of their eyes. If diplopia persists with one eye closed, this is called monocular diplopia, and occurs due to a distortion of light transmission through the eye to the retina. Common causes of monocular diplopia include cataracts, corneal irregularities, and refractive errors such as astigmatism.² If diplopia is present with both eyes open but disappears when one eye is closed, this is termed binocular diplopia. It's key to differentiate between these two entities because the pathophysiology and diagnostic implications of each are different.

Monocular diplopia is secondary to local eye disease, and workup can typically be deferred to outpatient ophthalmology follow-up. Binocular diplopia on the other hand occurs secondary to disconjugate alignment of the eyes, and has a broad differential diagnosis. Cranial nerve palsies are the most common cause of binocular diplopia, typically involving cranial nerves 3, 4 or 6. Other differential considerations causing ocular misalignment include neuromuscular transmission dis-

Differential of CN palsy

Pons/Midbrain infarct

Compressive lesions

aneurysm, tumor

Inflammatory/Infectious

sinusitis, sinus thrombosis

diabetes

Mechanical Obstruction

fat infiltration (Graves)

traumatic fracture

orders, and those processes causing mechanical interference with ocular motion.¹ Suggestive findings on history and physical exam can help differentiate between these causes and help guide further workup and neuroimaging.

As in our patient, a finding of an internuclear ophthalmoplegia on physical exam can provide important clues to the etiology and localization of pathology causing binocular diplopia. Internuclear ophthalmoplegia (INO) is characterized by paresis of eye adduction in horizontal gaze, but not in convergence. In other words, on horizontal gaze testing there is diplopia, weak adduction of the affected eye (typically can't adduct past midline), and nystagmus of the contralateral eye.¹ This occurs secondary to a lesion in the medial longitudinal fasciculus (MLF) in the dorsomedial brainstem tegmentum of the pons or midbrain.³ The most common causes of an INO vary by patient demographics. In younger patients < 45 years, the most common cause of INO is multiple sclerosis, and these defects are typically bilateral, up to 73%. In older patients > 60, and especially those with cardiovascular risk factors, the most common cause of INO is an ischemic infarction of the pons or medulla, and the findings are typically unilateral.⁴ The pathophysiology of this infarct is typically small artery occlusion or lacunar disease involving the penetrating arteries originating from the basilar artery.³ Other causes of INO include infections, tumor, and trauma.



Figure 1: Photo illustration showing left eye limitation in medial gaze. In true INO there is maintenance of accommodation. Photo Illustration by Ryan LaFollette.

1 - Colby, K. Diplopia: Symptoms of Ophthalmologic Disorders. In Merck manual online: Professional Edition. (Accessed on November 30, 2014)

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2. Bienfang, DC. Overview of Diplopia. In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed on December 1, 2014.)

<http://www.uptodate.com/contents/overview-of-diplopia?source=machineLearning&search=diplopia&selectedTitle=1%7E150§ionRank=1&anchor=H14#H14>

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http://www.uptodate.com/contents/internuclear-ophthalmoparesis?source=search_result&search=ino&selectedTitle=1%7E19

5 - Keane JR. Internuclear ophthalmoplegia: unusual causes in 114 of 410 patients. Arch Neurol 2005; 62:714.

DIFFICULTY SEEING

Sudden onset

Kristopher Ford MD
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History of Present Illness

The patient is a 52 yo male with a history significant for hypertension and diabetes presented to the emergency department stating that he was losing vision in his left eye. He reported that 2 days ago he noticed that he couldn't see very well out of the left upper visual field in his left eye. He states that over the past two days that has slowly worsened to involve the left lower field of vision and now the right lower visual field from left eye. The patient stated that he thought it would get better but it has continued to get worse. He denied any pain in the eye. He denied any pain with eye movements. Initially he denied floaters but when further explained he did state that 4-5 days ago he did have episodes of "seeing black dots" in his left eye. The patient denied any trauma. He denied photophobia.

Review of Systems

Negative

Medications

Lisinopril, hydrochlorothiazide

Past Medical History

Hypertension, diabetes

Allergies

No known

Social History

Denies tobacco, alcohol or illicit drug use

ED + Hospital Course

After examination, dye was placed in the eye in preparation for slit lamp examination. While allowing the anesthetic to work, an ultrasound of the eye was performed (Figure 1). After this finding ophthalmology was consulted and came to bedside immediately. Ophthalmology performed a dilated exam and diagnosed the patient with a retinal detachment. The patient was placed in the left lateral decubitus position with affected visual field down for approximately 45 minutes. He was then sent to the ophthalmology clinic where he underwent a surgical procedure for correction.



Figure 1 - Representative picture of an ocular ultrasound can easily be performed with a tegaderm over a closed eye, lubricant gel and a high frequency linear ultrasound probe³

Physical Exam

Temp	Heart Rate	Resp Rate	BP	O2 Sat
98	85	16	160/100	98%

General: Well appearing, no distress

HEENT: atraumatic, no scleral injection, extraocular movements intact with no pain, pupils 5->3 bilaterally reactive to light, no photophobia, good light perception bilateral eyes, intra-ocular pressure readings 12, 13, 12 bilaterally, left temporal visual field cut, acuity right eye 20/20, bilateral eyes 20/40, left eye 20/100

Chest: clear, CTAB, RRR with no murmurs

Abdomen: soft, nontender

Extremities: no signs of trauma, strong distal pulses, full range of motion

Neurologic: Awake and alert, oriented x4, 5/5 strength upper and lower extremities bilaterally, sensation intact, no pronator drift, no ataxia, no dysmetria

discussion

Retinal detachment is an important diagnosis to make in the emergency department because catching it early may prevent permanent vision loss. Many patients present to either their primary care physician or the ED, so it is more important to recognize this disease state and ensure urgent ophthalmology referral. The lifetime incidence of retinal detachment is 1 in 10,000. There are many risk factors on history that should heighten the suspicion for retinal

detachment. Age is likely the number one risk factor, as the incidence exponentially increases once patients reach the 7th and 8th decade of life. Other common risk factors include myopia, previous cataract surgery, and trauma. Less common risk factors include congenital retinal disease, diabetic retinopathy, familial history of detachment.

There are three types of retinal detachments.

Rhegmatogenous retinal detachment is caused by breaks in the retina. Tractional detachment is caused by scarring of the retinal surface and vitreous cavity. Exudative detachment is caused by leakage of fluid into the subretinal space.

The main symptoms that patients present

Continued on page 13

Mastering Minor Care PTA

peri-tonsillar abscess



Above - Left PTA with minimal uvular displacement. Atomized and local lidocaine can be effective analgesia. Letting the patient control a laryngoscope and suction can decrease anxiety and increase exposure during the procedure.
Below - endocavitary probe US of PTA with visible carotid

CASE

The patient is a 35 year old caucasian male who presents with one week of sore throat that became primarily left sided the few days prior to presentation

DIFFERENTIAL

A spectrum of tonsillar disease from tonsillitis to peritonsillar cellulitis to peri-tonsillar abscess

INTERVENTION

Medical (cellulitis or small abscess)

IV vs PO antibiotics with anaerobic coverage (amp/sulbactam, amox/clav, penicillin and metronidazole)¹. Small studies show steroids decrease recovery time.

Surgical (empiric drainage or known abscess)

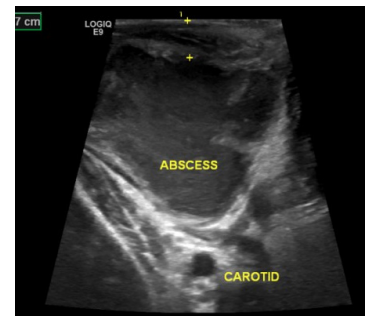
Needle aspiration or I&D. Both have about a 10% recurrence rate but I&D has shown to have faster improvement in symptoms.²

Intra-oral US for detecting PTA

SENSITIVITY - 89-95% SPECIFICITY 79-100%

1 - Powell J, Wilson J. An evidence-based review of peritonsillar abscess. Clin Oto. 2012;37:136-145.

2 - Lamkin RH, Portt J. An outpatient medical treatment protocol for peritonsillar abscess. Ear, Nose & Throat J 2006;10:685.



Fetid breath, sloughing gingival mucosa and blunted interdental recesses are the hallmarks of the disease

Griffen A. Gingivitis and periodontitis in children and adolescents: An overview. Up to Date.

Wilder RS, Moretti RJ. Gingivitis and periodontitis in adults: Classification and dental treatment. Up to Date.

ANUG

acute necrotizing ulcerative gingivitis
Vincent's Angina - Trench Mouth

CASE

The patient is a 25 year old African American female with a history of HIV infection, not currently on treatment, who presented with 5 days of mouth and gum pain.

DISEASE

Painful, erosive and necrotic gingival disease of rapid onset, primarily affecting people in their 20s. Risk factors include immunosuppression, pre-existing gingivitis, poor oral care, malnutrition.

TREATMENT

Local dentistry debridement is standard of care with oral microbial wash (chlorhexidine rinse) and systemic antibiotics with anaerobic coverage (penicillin) if febrile or ill appearing.

Shortness of Breath

an evaluation of thyroid storm

Riley Grosso, MD
University of Cincinnati R2

case originally seen by Jack Palmer, MD and Elizabeth Beckman, MD

History of Present Illness

The patient is a 44 year old male with Grave's disease who presented with several weeks of shortness of breath. He denied taking any medications for his Grave's disease recently. He originally attributed his shortness of breath to paint exposure, however it continued to worsen to the point where he felt as if he was going to pass out. He reported the shortness of breath was worse during exertion. He also complained of worsening shortness of breath while lying flat. He denied cough, chest pain, palpitations, fevers, leg swelling, or nausea.

Review of Systems

Bloating, frequent stools, otherwise negative

Past Medical History

Hypertension, Grave's Disease, Asthma

Medications

Aspirin, Methimazole (off for 9 months)

Allergies

No known

Social History

Denies tobacco, alcohol or illicit drug use

Temp
97.2
Heart Rate
134
Resp Rate
19
BP
132/75
O2 Sat
98%

ED + Hospital Course

The patient was found to be in atrial fibrillation with rapid ventricular response likely secondary to thyrotoxicosis. He was given metoprolol IV three times without response and was then placed on an esmolol drip to help control his heart rate. He was also given methimazole and dexamethasone in the ED and then admitted to cardiac stepdown. The patient was diagnosed with thyrotoxicosis without evidence of thyroid storm secondary to his previously diagnosed Grave's Disease. The patient had positive antibodies to thyroid peroxidase and thyrotropin receptor previously documented, which are consistent

with the diagnosis of Grave's Disease.

The patient's heart rate proved difficult to control with esmolol, so he was started on digoxin for rate control along with metoprolol. Digoxin was chosen preferentially over diltiazem because he was found to have non-ischemic cardiomyopathy with an EF of 20%. Endocrine was consulted for management of his thyrotoxicosis and recommended continuing the methimazole. The patient was discharged on digoxin, metoprolol, and methimazole.

Physical Exam

General: well-developed, well-nourished AA male in NAD
HEENT: normocephalic, atraumatic, moist mucous membranes, no scleral icterus, no proptosis, no blots/hemorrhages/papilledema on retinal exam
Neck: **diffusely enlarged thyroid gland** without nodularity, no rigidity
Pulm: lungs clear and equal bilaterally without wheezes, rales, or rhonchi
Cardiac: tachycardic with an irregularly irregular rhythm, no murmurs, rubs, gallops, no S3 or S4 heard, pulses +2 in all extremities
Abdomen: +BS, soft, non-tender without rebound or guarding
Musculoskeletal: full ROM of joints, no edema in BLE
Skin: well hydrated without rashes or lesions
Neuro: AAO x 4. DTR +2/4 in BLE, CN II-XII intact, motor and sensory exam grossly normal
Psych: mildly anxious otherwise normal affect and mood

Labs

BMP, CBC, BNP, troponin within normal limits
TSH: <0.01, Free T4; 5.71(normal 0.61-1.76)
T3: 21.6 (normal 2.8-5.3)

Grave's Disease

Grave's disease is the leading cause of thyrotoxicosis and thyroid storm, causing 60-80% of presentations to the ED with thyrotoxicosis.⁴ Grave's is an autoimmune disease that presents during the 3rd-5th decades of life, predominately in females. The patient may present with a constellation of symptoms consistent with thyrotoxicosis, painless enlargement of the thyroid gland, decreased TSH, and increased T4/T3. A variety of antibodies to the receptor for thyroid stimulating hormone are produced, continuously activating this TSH receptor. Our patient had the thyrotropin binding- inhibiting immunoglobulins, which mimics TSH binding to its receptor. Our patient also had elevated thyroid-peroxidase autoantibodies which are more commonly found in Hashimotos thyroiditis but can also be found in Grave's disease.



lead II rhythm strip: HR 185, atrial fibrillation with a rapid ventricular response, LVH, no new ST changes

Thyrotoxicosis

Hyperthyroidism has an incidence in the US of 0.05%-1.3% with most of these patients presenting with very few, if any, symptoms of hyperthyroidism.¹ The term hyperthyroidism specifically refers to excess circulating thyroid hormone secondary to increased thyroid gland activity. Thyrotoxicosis refers to excess thyroid hormone secondary to any cause, including thyroid hormone overdose. Patients with thyrotoxicosis can present with a variety of symptoms, including weakness, heat intolerance, anxiety, psychosis, diplopia, dyspnea, palpitations, diarrhea, menorrhagia, or hair loss. Objectively patients with thyrotoxicosis can present with diaphoresis, hyperreflexia, lid lag, exophthalmos, pretibial myxedema, or

tachydysrhythmias.

A thyroid storm is rare, accounting for <1% of admissions for thyrotoxicosis, however its mortality can reach 30% if left untreated. The definition of thyroid storm is a life threatening condition caused by the exaggeration of the clinical manifestations of thyrotoxicosis. Though defined by four major features, it is uncommon for a single patient to have all four features of thyroid storm: fever, tachycardia, central nervous system dysfunction, and GI symptoms. Thyroid storm is most commonly observed in patients with Grave's disease, however toxic goiters and a variety of thyroid cancers can also be at fault.² The Burch & Wartofsky Score can be utilized to assess

likelihood of thyroid storm. See table 1 for details on the 6 symptomatic categories of scoring. A score >45 is highly suggestive of thyroid storm, a score from 25-44 is suggestive of impending storm, and a score <25 is unlikely to represent storm. Our patient had a Burch and Wartofsky Score of 30. Utilizing this classification system he was not in a thyroid storm. In a recent retrospective cohort study, the presence of fever, altered mental status, and an identifiable precipitating event (usually infection or trauma) significantly increased the chances of being diagnosed with a thyroid storm instead of thyrotoxicosis. Patients with thyroid storm have significantly longer hospital lengths of stay and end up in the ICU more often than those with thyrotoxicosis.³

Burch & Wartofsky Score <i>grading scale of thyroid storm severity</i>					
<25 - unlikely to represent storm		25-44 - suggests impending storm		>45 - highly suggestive of storm	
Temp		CNS		GI/Liver	
99-99.9	5 pts	Agitation	10 pts	N/V/D	10 pts
100-100.9	10 pts	Delirium	20 pts	Jaundice	20 pts
101-101.9	15 pts	Seizure	30 pts		
102-102.9	20 pts	/Coma			
103-103.9	25 pts				
>104.0	30 pts				
HR		Heart Failure		Precipitant	
99-109	5 pts	Pedal Edema	5 pts	Negative	0 pts
110-119	10 pts	Rales	10 pts	Positive	10 pts
120-129	15 pts	Atrial Fib	15 pts		
130-139	20 pts				
>140	25 pts				

Treatment of Thyrotoxicosis

The treatment of thyrotoxicosis and thyroid storm is aimed at: a) controlling the production of thyroid hormones b) blocking the action of circulating thyroid hormones c) supportive care and d) addressing the precipitating event if one can be identified. Controlling the production of thyroid hormones is accomplished by blocking the action of thyroid peroxidase, thereby stopping the thyroid gland from organifying iodine. Propylthiouracil (PTU) and methimazole are the most commonly used antithyroid drugs, with PTU being the drug of choice in thyroid storm because it also blocks the peripheral conversion of T4 to T3, which is the more active metabolite. Patients in thyroid storm will also receive inorganic iodine, as this decreased the rate of thyroid hormone synthesis within the thyroid gland. High dose glucocorticoids will both block release of thyroid hormone from the thyroid and decrease peripheral conversion from T4 to T3. Our patient received methimazole & dexamethasone. The methimazole was chosen over PTU likely because he had been

managed on that medication as an outpatient previously and did not need the extra benefit of blocking peripheral conversion.

Current symptoms are treated by inhibiting the action of circulating thyroid hormone. This can be done by giving B-blockers. It is important to note that treatment with B-blockers is not reserved for treatment of atrial fibrillation caused by thyrotoxicosis but is beneficial in the treatment of fever, diaphoresis, agitation and GI symptoms.² Patients who present with thyrotoxicosis or thyroid storm who are not in atrial fibrillation should still receive B-blockers, as they inhibit the adrenergic effects of the circulating thyroid hormone.

Supportive care is often administered in the form of antipyretics, fluid replacement, and management of thyroid storm induced heart failure symptoms. Our patient had persistent atrial fibrillation with rapid ventricular response even after B-blockade was initiated, so additional antiarrhythmic medications

were initiated in the form of digoxin. It is important to note that the patient continued to receive B-blockers in the form of oral metoprolol. Reversing the precipitating event, if one exists, should be the major focus of the treating physician, focusing on rapidly identifying and treating any infections, medication errors or metabolic abnormalities that may have acted as precipitant.¹

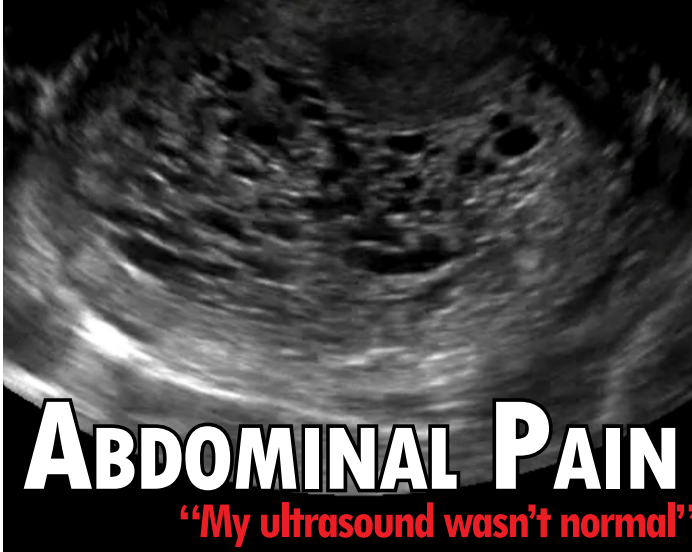
1 - Idrose A. Chapter 224. Thyroid Disorders: Hyperthyroidism and Thyroid Storm. In: Tintinalli JE, Stapczynski J, Ma O, Cline DM, Cydulka RK, Meckler GD, T.eds. Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 7e. New York, NY: McGraw-Hill; 2011. <http://accessmedicine.mhmedical.com/content.aspx?bookid=3488&Sectionid=40381707>. Accessed November 26, 2014.

2 - Sarlis, Nicholas J (05/2003). "Thyroid emergencies" . Reviews in endocrine & metabolic disorders (1389-9155), 4 (2), p. 129.

3 - Angell, Trevor E (10/24/2014). "Clinical Features and Hospital Outcomes in Thyroid Storm: A Retrospective Cohort Study" . The journal of clinical endocrinology and metabolism (0021-972X), p. jc20142850

4 - Jameson J, Weetman AP. Chapter 341. Disorders of the Thyroid Gland. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson J, Loscalzo J. eds. Harrison's Principles of Internal Medicine, 18e. New York, NY: McGraw-Hill; 2012. <http://accessmedicine.mhmedical.com/content.aspx?bookid=331&Sectionid=40727146>. Accessed November 26, 2014.

#sickornotsick



ABDOMINAL PAIN

“My ultrasound wasn’t normal”

Kristopher Ford MD
University of Cincinnati R4

case originally seen by R1 Mike Miller, MD and R4 Dina Gozman, MD

History of Present Illness

The patient is a 18 year old female G1P0 at 7 weeks and 5 days by last menstrual period who presented to the ED with vomiting. The patient had a positive pregnancy test 3 weeks ago as well as a transvaginal ultrasound a week later at a clinic. She was told that the ultrasound was “not normal” (Image 1) and she should follow up with a gynecologist. The patient reported that over past few days she has had mild abdominal cramping and a few episodes of vomiting. She stated the pain was 4/10 in intensity and was located across lower abdomen with no radiation. She denied any dysuria, hematuria, constipation, diarrhea, hematochezia, vaginal bleeding or discharge.

Past Medical History	Medications	Social History
None	None	Social alcohol use, denies tobacco or illicit drug use
Past Surgical History	Allergies	
None	No known	

Temp	Heart Rate	Resp Rate	BP	O2 Sat
98.3	72	16	119/68	98%

Physical Exam

General: Well appearing female in no distress
 HEENT: Normocephalic, atraumatic, ocular movements intact, no scleral icterus, oropharynx clear with normal sized tonsils and no erythema, uvula midline
 Chest: Lungs clear to auscultation with no wheezing
 Heart: Regular rate, rhythm, no murmurs
 Abdomen: Soft, nondistended, nontender, no signs of peritonitis
 Extremities: Full range of motion , no edema, no tenderness, no signs of trauma
 Skin: No rashes
 Neuro: Awake and alert, fully oriented, moves all extremities spontaneously

Labs
 β-hCG - 135,930

Image 1 - Representative transvaginal ultrasound - demonstrated an irregular gestational sac with heterogeneous uterine contents with no free fluid, normal ovaries and uterus suspicious for complete molar pregnancy or other gestational trophoblastic disease³

ED + Hospital Course

Gynecology was consulted after the abnormal findings on ultrasound and markedly elevated β-hCG. They made the diagnosis of molar pregnancy given the ultrasound findings, beta hcg level and clinical presentation. The patient was admitted to gynecology for dilatation and curettage and did well post-operatively.

discussion

The term molar pregnancy is comprised of two separate entities complete hydatidiform moles and partial hydatidiform moles (see table 1 for comparison). These diseases are part a the larger spectrum of related conditions known as gestational trophoblastic disease. From the standpoint of the emergency provider, the former is more relevant to clinical practice as these can present in early pregnancy. In fact, a significant portion of confirmed hydatidiform moles are diagnosed initially as missed abortion and confirmed as moles after the specimen is sent to pathology after dilatation and curettage. The American College of Obstetricians and Gynecologists (ACOG) estimate that moles are observed in 1 in 600 therapeutic abortions and 1 in 1500 pregnancies. The diagnosis is important not only for the short term goal of terminating the pregnancy, but also because there is increased risk of developing malignant sequelae after molar pregnancy often require chemotherapy after evacuation of the mole.

Historically, the diagnosis of complete moles was made in the second trimester. The clinical presentation included excessive uterine size, anemia, toxemia, hyperemesis, hyperthyroidism, and respiratory failure. With the current availability of ultrasound and more accurate and sensitive detection of hCG levels, the diagnosis is now usually made in the first trimester before these symptoms have a chance to develop. Alternatively, partial moles typically present with signs and symptoms of a missed or incomplete abortion, including vaginal bleeding or pelvic cramping. Markedly elevated hCG levels, usually greater than 100,000 mIU per mL, as in our patient, are highly suggestive of complete moles. Unfortunately since partial moles typically have less prominent trophoblastic hyperplasia they infrequently have markedly elevated hCG levels. The main ultrasonographic finding of a complete mole is a vesicular pattern, or “snowstorm” pattern as it is classically known. This is due to the characteristic marked swelling of chorionic villi in complete moles. Partial moles have ultrasound findings that may be less obvious to non-radiologists or non gynecologists including focal cystic changes in placenta and a ratio of transverse to anteriorposterior dimension of the gestational sac greater than 1.5. While the combination of the hcG levels and ultrasound findings aid in the diagnosis, the final diagnosis can only be confirmed after the specimen from dilatation and curettage is sent to pathology.

The management of molar pregnancies involves evacuation by suction curettage. Close monitoring and follow up is required due to the risk of malignancy. Patients who do not wish to preserve childbearing ability may have a hysterectomy to reduce the change of malignancy, although there still remains a small risk.

ACOG recommends that serial hCG levels should be monitored for all complete or partial molar pregnancies in an effort to detect malignancy early. These hCG levels should be performed weekly until levels are undetectable for 3 weeks, then monthly until undetectable for 6 months. Since a new gestation would interfere with this testing, patients are strongly encouraged to use reliable contraception for at least 6 months.

- 1 - Soper J, et al. Diagnosis and treatment of gestational trophoblastic disease: ACOG Practice Bulletin No. 53. Gynecologic Oncology, 2004.
- 2 - Berkowitz, R, et al. Molar Pregnancy. The New England Journal of Medicine, 2009.
- 3 - Molar pregnancy. http://sonocloud.org/molar-pregnancy_v1259

Complete

vs

Partial Molar Pregnancies

Karyotype
Fetus/embryonic tissue
Swelling of chorionic villi
Trophoblastic hyperplasia
Uterine size
Postmolar malignancy

COMPLETE MOLAR

Diploid (usually 46, XX)
Absent
Diffuse
Diffuse
Large for gestational age
6-32%

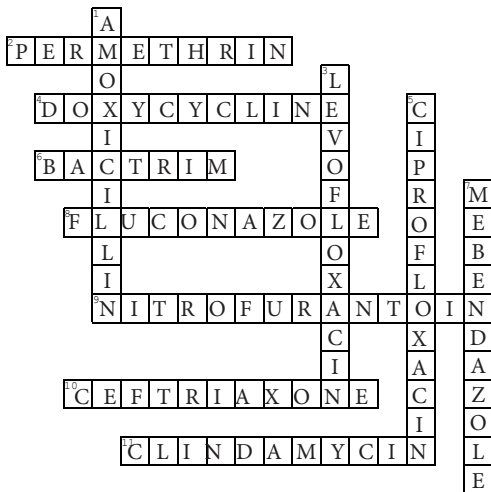
PARTIAL MOLAR

Triploid
Present
Focal, variable
Focal
Small for gestational age
< 5%

Figure 1 - Important distinctions both in defining characteristics and prognosis of complete vs partial molar pregnancies

B-Pod Bugs and Drugs Crossword

Name the first line antimicrobial therapy for the given patient



List of Submitted B Pod Cases

	R4	R1
New Onset HIV –swab/+blood	Redmond	Palmer
Posterior Cerebellar Infarct	Ford	
Prolapsed Colostomy	Ford	Teuber
Isopropol alcohol overdose	Redmond	May
Pneumomediastinum	Redmond	Bartholomew
Syphilis & HIV rash	Bell	Titone
Syncope with 2nd degree AV block	Redmond	Teuber
Hydrops gallbladder	Redmond	MS4
Epidural Hematoma w/ SAH	Verzwyvelt	Teuber
Latent TB & INH toxicity	Walsh	Bartholomew
Molar Pregnancy	Gozman	Miller

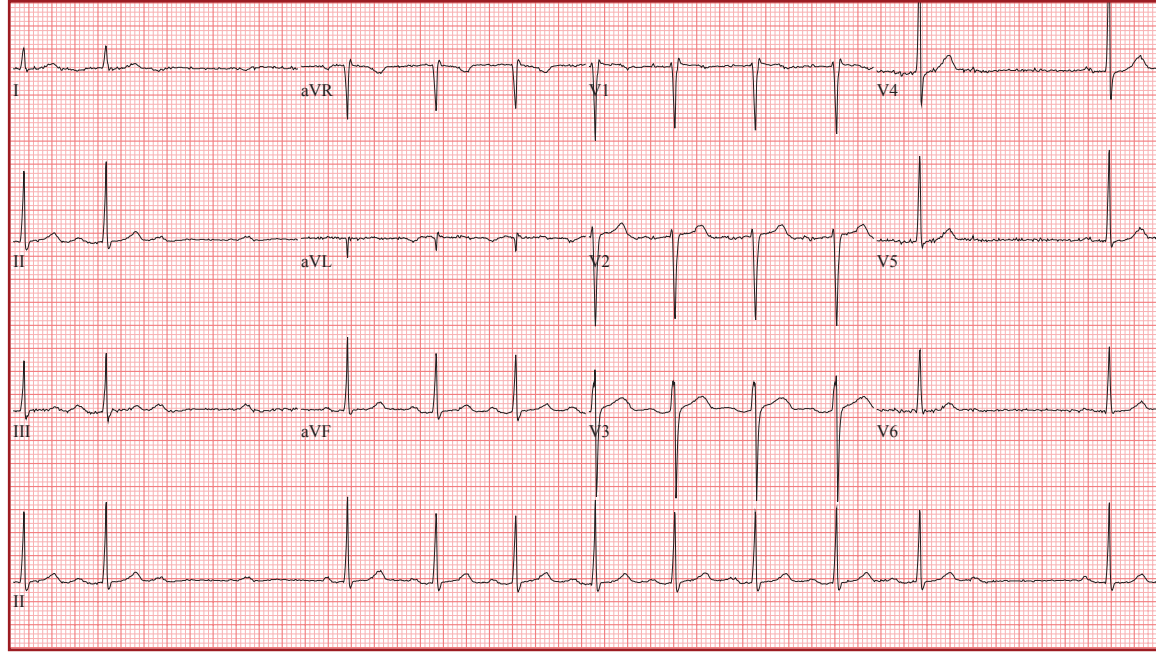
Annals of B Pod is looking for YOU to submit your interesting cases of B Pod - There is a composition book at the R4 desk - please make sure to include the R1/R4 involved in the case, a brief synopsis and a patient sticker
annalseditors@gmail.com

SYNCOPE

A first presentation of heart block

Riley Grosso MD
University of Cincinnati R2

case originally seen by
R1 Julie Teuber, MD and
R4 Kristopher Ford, MD



HR 64 Rhythm: sinus rhythm with a high grade AV block with multiple dropped beats.

History of Present Illness

The patient is a 26 year-old male with no known medical problems who presented to the emergency department after passing out. He reported that after arguing with his girlfriend he stood up from the couch and then fell to the ground. The girlfriend did not notice any shaking or loss of bowel or bladder control during this episode. He reported that this is the fifth time this has happened to him since he was a teenager and it always seems to happen when he is angry. He stated that he also experiences intermittent episodes of gripping chest pain and shortness of breath during these episodes but they also happen

when he is angry and doesn't pass out. He denies nausea, vomiting, decreased intake, or fevers.

Review of Systems

Negative

Past Medical History

None

Medications

None

Allergies

No known

Social History

Denies tobacco, alcohol or illicit drug use

Temp
97.8

Heart Rate
58

Resp Rate
16

BP
123/92

O2 Sat
100%

Physical Exam

General: well developed, African-American male in no acute distress, converses in full sentences

HEENT: normocephalic, atraumatic, pupils equal round and reactive to light bilaterally, extra-ocular movements intact, oropharynx benign

Neck: supple, no lymphadenopathy, trachea midline, no masses

Pulmonary: clear to auscultation bilaterally, good air movement

Cardiac: regular rate and rhythm, no murmurs, rubs, or gallops

Abdomen: soft, non-tender, non-distended, no rebounding or guarding

Musculoskeletal: full range of motion in all joints, no edema or clubbing, +2 pulses in all extremities

Neuro: alert and oriented x4, moves all 4 extremities with equal strength, CN II-XII intact

Psych: mood and affect are appropriate

ED + Hospital Course

The patient was found to have a 2nd degree AV block on his EKG (see above) and was originally admitted to the cardiology service, however the patient refused to stay in the hospital and after multiple discussions with the team he signed out against medical advice. He presented back to the Emergency Department three days later with similar complaints. His EKG at that time showed bradycardia with a 1st degree AV block.

He was admitted to cardiology, who felt the patient had EKG concern-

ing for Mobitz Type I with a high degree AV block. His ECHO was normal and he had no more syncopal events or arrhythmias while in the hospital. He had a cardiac MRI to evaluate for myocarditis or structural abnormality as the cause for his arrhythmia. This MRI showed a possibility of an anomalous right coronary artery and recommended a cardiac CT, which showed normal coronary arteries. The patient was offered a pacemaker and refused, saying he had only had a few syncopal episodes in his life and because he had Type I not Type II Mobitz. The patient was allowed to leave with an event monitor and cardiology follow-up.

discussion

Syncope is the chief complaint in 1-2% of ED visits and 6% of hospital admissions per year in the US.² More importantly, patients deemed to be high risk have a mortality as high as 30% within 1 year of a syncopal event.¹ Patients with a cardiac etiology of syncope make up 10% of all

patients who present to the ED with syncope.² The ability to determine which of your patients are at risk for a cardiac etiology of syncope is important, as symptomatology of cardiac syncope overlaps significantly with that of vasovagal syncope. In patients with prior cardiac dis-

ease, a history of syncope while supine or syncope with exertion both increase the likelihood that they have experienced cardiogenic syncope.³ In patients without cardiac disease, a patient who complains of palpitations is more likely to have cardiogenic syncope. These cardiac-related

syncopal episodes are divided into structural causes, i.e. aortic stenosis, and dysrhythmias, usually tachyarrhythmias.² The pt in this case had a bradydysrhythmia, which is less likely to be the cause of a syncopal episode.²

There are a few decision rules that help decide who is at risk for serious outcomes. The most well-known rule is the San Francisco Syncope Rule (See Table 1). An older study found that the odds ratio of mortality at one year associated with abnormal EKG was 3.5.⁵ In this study abnormal was defined by a rhythm abnormality or conduction block, as in our patient.

A conduction abnormality at the AV node was identified as our patient's cause of syncope. These AV abnormalities are divided into first degree, which is a lengthening of the PR interval, second degree which consists of intermittent AV conduction, and the third degree which is complete interruption of the AV node. Our patient had a second degree block, which was read in the emergency department as a Mobitz Type II. This type of AV node conduction delay results in the same PR interval before and after the non-conducted atrial beats.⁶ This type of block is generally coming from an infranodal conducting system delay and therefore implies structural damage to the infranodal conducting system that can progress to a complete heart block suddenly. The inpatient team inter-

preted the patient's EKG's as a Mobitz I, which is also referred to as a Wenckebach block. In this type of AV node conduction delay, there is a progressive prolongation of AV conduction until an atrial impulse does not get conducted. This happens because each depolarization prolongs the refractory period of the AV node until an atrial impulse hits the AV node during a refractory period. Unlike a Mobitz II, this type of second degree AV block is considered transient and is usually associated with infection, drug overdoses, or cardiac ischemia.⁶

The patient underwent an extensive work-up of his 2nd degree AV block, including serial troponins and serial EKGs to rule out cardiac ischemia as the cause of his syncope. He also had an echocardiogram and cardiac MRI to rule out evidence of endocarditis or valvular disease. The MRI showed a possible anomalous right coronary vessel, so the patient got a cardiac CT, which showed normal coronary vessels. Given the patient's 2nd degree AV block of undetermined etiology was not at risk of degenerating into a complete heart block, the patient was given the option of a pacemaker but de-

ferred at the time of admission. He was discharged on an event monitor and instructed to follow up with cardiology.

San Francisco Syncope Rules	SYNCOPE HIGH RISK WITH ANY OF BELOW FEATURES
	Abnormal EKG ANYTHING OTHER THAN SINUS OR CHANGE FROM BASELINE Shortness of Breath Hematocrit < 30% Systolic BP < 90 mmHg History of CHF
SENSITIVITY - 98% SPECIFICITY 56%	

Table 1 - San Francisco Syncope Rules⁴

- 1 - Blanc JJ, L' Her C, Touiza A, et al: Prospective evaluation and outcome of patients admitted for syncope over a 1 year period. *Eur Heart J* 23: 815, 2002.
- 2 - Soteriades ES, Evans JC, Larson MG, et al: Incidence and prognosis of syncope. *N Engl J Med* 347(12): 878, 2002.
- 3 - Stern SC, Cifu AS, Altkorn D. Chapter 26. I Have a Patient with Syncope. How Do I Determine the Cause?. In: Stern SC, Cifu AS, Altkorn D. eds. *Symptom to Diagnosis: An Evidence-Based Guide*, 2e. New York, NY: McGraw-Hill; 2010
- 4 - Quinn, James V (02/2004). "Derivation of the San Francisco Syncope Rule to predict patients with short-term serious outcomes" . *Annals of emergency medicine* (0196-0644), 43 (2), p. 224.
- 5 - Martin, T P (04/1997). "Risk stratification of patients with syncope" . *Annals of emergency medicine* (0196-0644), 29 (4), p. 459.
- 6 - Piktetl JS. Chapter 22. Cardiac Rhythm Disturbances. 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.

Retinal Detachment Continued from page 6

with are photopsia, floaters and/or vision loss. Photopsia is described as flashes of light and floaters are often described as "black dots," "cobwebs," or "cloudy haze." Vision loss occurs if the macula becomes detached. The description of vision loss is usually peripheral in most cases. Progression to central vision loss varies and can take hours to days to even weeks. Once central vision loss occurs normal vision is unlikely even with repair. This underscores the importance of urgent ophthalmology evaluation even if patients are just presenting with acute floaters or photopsia and/or peripheral vision loss.

Evaluation involves assessing visual acuity, visual field testing and pupillary response. A direct ophthalmoscope may identify a retinal detachment, however it cannot exclude as its field of view is too narrow. Slit lamp exam or indirect ophthalmoscopy allows the best visualization of a retinal detachment. Dilating the pupil, although usually only done by ophthalmology, enhances the field of view. Additionally, ultrasound may be used to aid in the diagnosis. Emergency medicine physicians have developed an increased comfort with bedside ultrasonography such that the classic visual representation of retinal detachment on ultrasound allows this diagnosis to be readily made. Unfortunately, similar to the use of a direct ophthalmoscope, an ultrasound cannot

exclude retinal detachment because it does not image the periphery well.

Once retinal detachment is suspected, urgent ophthalmology consultation or referral is necessary. The treatment involves surgical correction either in the OR or with a noninvasive procedure in the office.

- 1 - Hyong Kwon Kang et al. Management of retinal detachment: a guide for non-ophthalmologists, *British Medical Journal*, 2008.
- 2 - D' Amico, D. Primary Retinal Detachment, *The New England Journal of Medicine*, 2008; 359:2346-54.
- 3 - <http://sonocloud.org/item/photos/48/104YBDYN5771/Retinal-detachment>



Dr. Carleton is a nationally renowned leader in airway management and shares his insightful tips through email and publication IC Cordes

IC Cordes:

Welcome to flight

Steven Carleton MD PhD Professor of Emergency Medicine

It is a dark and stormy night...but with just enough ceiling and visibility to permit a quick flight should the need arise. Lo and Behold!...the need arises. You are called to the scene of a motorcycle crash for a 46 year-old, unhelmeted rider who drove his blue Harley-Davidson Electroglide into a guardrail.

You arrive to find a largish, hairy, leather-clad, multi-tattooed biker in full spinal immobilization. He has open fractures of both legs, he grunts when you push on his belly, he has instability of his left clavicle, and his GCS is 10 (M-5, V-3, E-2). There is evidence that he has been drinking, in that there is a puddle of regurgitated Southern Comfort on the floor of the squad where the patient currently resides.

Vital Signs

Pulse - 118. Respirations 24 and unlabored. Blood pressure 106/68. O2 saturation 96% on 4L NC.

You are 27 miles (13 minutes) from home.

What now? Well, as in every patient, every time, without exception (EPETWE [not to be outdone by Hinckley's DASH-1A]), you contemplate whether the patient requires intubation. Is he failing to oxygenate? No. Failing to ventilate? Unknown, but likely not. Failing to protect or maintain his airway? He protected it during his episode of emesis, but what about in 10 minutes? Unknown. Will his subsequent clinical course be improved by intubation? Certainly! He is going to end

up in the OR, for those femurs if for nothing else. However, there is a price to be paid for intubating in the field, and the currencies in which that price is paid are TIME and INTUBATING CONDITIONS. He doesn't look like a guy who normally has a pressure of 106/68, and he is tachycardic with several major sources of potential hemorrhage, and maybe a few occult sources, as well. The field time if we intubate will likely be increased by 10 to 20 minutes, and he may shift from stable to a hemodynamic disaster in that time. Further, you will have to perform your intubation in the back of a cramped squad with a balky suction and an ambulance crew that likely have never seen an intubation before to aid. Further, when you apply the Universal Airway Algorithm (EPETWE!!!), you note that he is not a CRASH, but is potentially DIFFICULT (obese, immobilized, short neck, bushy beard, bloody face, etc.).

You consider your choices for the difficult airway. Rapid sequence intubation (RSI) with direct laryngoscopy (DL), RSI with iGel. Your confidence in the former is blunted by the difficulty mnemonics. Your confidence in the second is blunted by inexperience with the devices. You are assailed by doubts, and find yourself wishing for an alternative. You decide to transport the relatively short distance without a definitive airway. In doing so, you are calling in all of your karmic chips. If he makes it O.K., you will be congratulated for your judgment. But if he loses his airway en route, his life and your reputation are at risk. If there was just an alternative!

You put the patient on a high-flow NRB and

fly! Your sphincter pressure is such that any unbound carbon atoms lurking in the terminal portion of your GI tract are compressed into tiny diamonds. Alas, that they cannot be recovered for gain! Eight miles out, his pulse ox begins a precipitous fall. Suction...no change! Bag...ineffective! DL...CL-IV view! Bougie...goosed! iGel...successfully inserted, but with a big gas leak and suboptimal ventilation. You arrive, hot offload, and bring the man to the SRU. His edematous airway is intubated after reluctant RSI with the Berci-Kaplan, using the video view, as the direct view was still a CL-IV. You cannot help but wonder whether the period of hypoxia will result in an anoxic brain injury.

This is a tough case, but not a far-fetched one. It is also true. If there was just an alternative! Well, THERE IS!!!! I LOVE TECHNOLOGY!!!

The King Vision video laryngoscope. We have one for the Sim Lab, and one for each aircraft. This device consists of a video screen that runs off of three AAA-batteries, and a disposable blade that contains the LED light source and CMOS camera. The video head slides into the blade and seals to a purple gasket that has a bulge which mates with a corresponding notch in the video head. This assures that the head can only be inserted one way. Two iterations of blade are available: 1) Channel blade. This blade has a channel on its right-hand side that accommodates and guides an endotracheal tube. In this way, it is functionally similar to the AirTraq, a capable but unloved device. 2) Non-channel blade. This blade is used just like that of the C-MAC, where a stylet-

ted tube is passed freehand into the video field and glottis. We elected to purchase the channel blades. Why? It eliminates the need to buy special stylets, and it solves the problem that I am confident that most of your encountered in Grand Rounds, intubating the non-standard positioned mannikin tomahawk-style (having to coordinate insertion of the tube off-handed and backwards). Having a device that permits intubation when positioning is compromised is a very helpful thing.

When you examine any blade, recognize whether it looks more like a Mac or a Miller. If it looks like a Mac, USE IT LIKE A MAC!!! The King Vision blade looks like a Mac – blunt, squared, slightly bulbous tip. Thus, it is a vallecula device. The King Vision suffers

from the same idiosyncrasy that afflicts all video laryngoscopes. The best glottic view DOES NOT produce the best intubating conditions. The blade should be inserted under visual guidance into the vallecula, engage the hyoepiglottic ligament, and then be lifted up and out and tilted back to obtain a glottic view. The “cheap seats view” is what you want – a view that shows the epiglottis and entire hypopharynx. Then advance the pre-loaded tube through the channel into the glottis. Passage will be facilitated by maintaining the blade in the mid-line, and by resisting the temptation for deep insertion. You will be absolutely convinced of the validity of this counter-intuitive method after a single training session with the device.

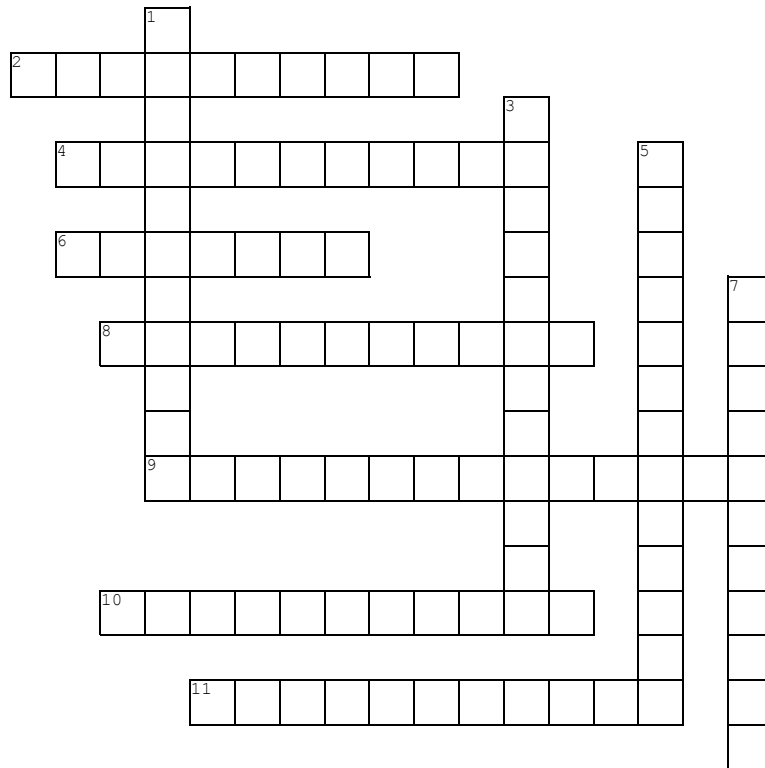
How might this device have changed this

case? It would likely have allowed successful, early intubation when the patient decompensated in the aircraft despite the suboptimal relative positioning of the patient and the doc. It might also have increased your confidence for intubating in the back of the squad pre-flight, and relieved you of all of the suffering that ensued because of this uncertainty.

You will not optimize the use of this modality unless you are trained, and you must PRACTICE. It is relatively easy to gain the muscle memory required for deft use of video laryngoscopy, but only if you do it regularly. Hmmmm....might more widespread use of the Berci-Kaplan or C-MAC facilitate this? Might practice with the available resources in the Sim Lab also help?

B-Pod Bugs and Drugs Crossword

Name the first line antimicrobial therapy for the given patient



Riley Grosso MD
University of Cincinnati R2

Across

2. 17yo female with scabies
4. 19yo female with PID
6. 57yo male HIV+ with PCP
8. 12yo male with tinea versicolor
9. 24yo pregnant female with UTI
10. 47yo female cirrhotic with SBP
11. 45yo female with a felon

Down

1. 23yo pregnant female with Lyme disease
3. 35yo healthy male with PNA
5. 67yo male with exposure to anthrax
7. 4yo male with pinworm

Answers on page 11

RUSH exam
Continued from back cover

The RUSH exam showed a free moving clot at the IVC/right atrial junction concerning for a clot in transit. The patient's creatinine was 2.0 and could not receive a CTPA. Cardiology was consulted and heparinization was recommended, however given the recent neurosurgical intervention, the patient received an angio-vac clot removal in the angio suite. The patient did well

and was eventually discharged from the hospital.

1: Rose JS, Bair AE, Mandavia D, et al. The UHP ultrasound protocol: A novel ultrasound approach to the empiric evaluation of the undifferentiated hypotensive patient. Am J Emerg Med. 2001;19:299-302.

2: Jones AE, Tayal VS, Sullivan DM, et al. Randomized, controlled trial of immediate versus delayed goal-directed ultrasound to identify the cause of nontraumatic hypotension in emergency department patients. Crit Care Med. 2004;32:1703-1708.

Quick Hit

Riley Grosso MD
University of Cincinnati R2

Ryan LaFollette MD
University of Cincinnati R3

Rapid Ultrasound for Shock and Hypotension (RUSH)

case

The patient is a 70 year old male with a past medical history of an unspecified hypercoagulable disorder, hypertension, and frequent falls presents from a nursing home with shortness of breath. The patient was just discharged from the hospital yesterday and is 7 days status post evacuation of a subdural hematoma by neurosurgery. He is still off of his Coumadin due to the bleeding risk after surgery. He started having shortness of breath this afternoon otherwise he is feeling well.

HR 133 BP 130/80 RR 30 SpO2 88% on RA 95% on 3L O2 T 101.3 F

Gen: appears stated age, alert, moderate distress
Resp: increased resp rate, no wheezes, rhonchi or rales, clear to auscultation bilaterally
Cardiac: tachycardic with a regular rhythm, no murmurs, rubs gallops
Neuro: alert and oriented x3, GCS 15, normal exam
CXR: normal
EKG: sinus tachycardia



see the video at tamingtheSRU.com

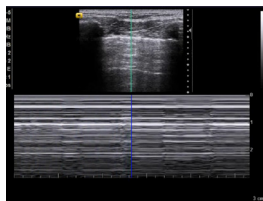
↑ His RUSH exam reveals

Answer on Page 15

Rapid Ultrasound for Shock and Hypotension (RUSH) can be thought of as the FAST exam for medicine patients. It is an exam developed for the rapid, goal directed assessment of the undifferentiated shock patient.¹ When studied, the RUSH exam improved the accuracy of diagnosis by 30% at 15 minutes after presentation of an undifferentiated patient in non-traumatic shock.² The mnemonic HI-MAP² explains both the components and sequence of the RUSH Exam

HI-MAP²

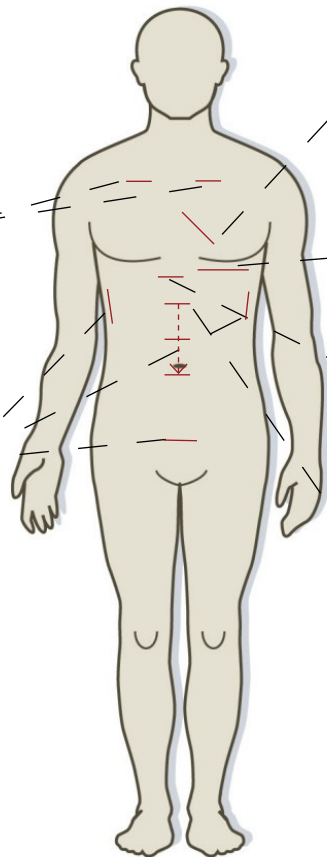
Heart - IVC - Morrison's (FAST) - Aorta - Pneumothorax



Linear (high frequency probe)

Pneumothorax
- 2nd intercostal space transverse view looking for lung sliding

Hemoperitoneum
- longitudinal views of Morrison's (hepatorenal), splenorenal and transverse retrovesicular views to rule out significant intra-peritoneal fluid



General (low frequency probe)

Effusion / Cardiogenic Shock
- parasternal long view for gross left ventricular contraction/function
- pericardial effusion

Right Ventricular Strain / PE
- apical 4 view; transverse view angled towards right shoulder

Hypovolemia
- longitudinal view of the IVC looking for respiratory variation (>50%)

AAA
- transverse views of supra-renal, infra-renal and aorta above iliac bifurcation >3cm

