Sudden Onset Altered Mental Status
Anees Daud, MD
Intermountain Medical Center

History
A 71-yr-old woman with a past significant history of relapsing-remitting multiple sclerosis, anxiety, and depression, presented to the emergency department with a one-day history of altered mental status. The patient’s husband and granddaughter provided the history. The patient was reportedly in her usual state of health until 9 AM on the day of presentation, when she began to act “strangely” and “confused.” The patient was altered in the ED as well, at one point taking off her clothes and walking around the ED.

Of note, the patient had recently been under significant stress because one of her daughters was going through a divorce and had moved in with her parents. Her medications included norco, duloxetine, bupropion, donepezil, pregabalin, levetiracetam, omeprazole, losamox, docusate, and occasional zolpidem.

Physical Exam and Diagnostic Studies
The patient’s vital signs were T 36.2C, BP 187/80, HR 116, RR 22, and SaO2 90% on room air. On exam, she was agitated, impulsive, attempting to climb out of bed, and unable to carry on a conversation. She was diffusely tremulous, especially in the upper extremities. She was diaphoretic, and skin was warm to the touch. Cranial nerves were difficult to test, but she had appropriate lateral EOMs, and no facial asymmetry. She had hyperreflexia in the biceps, triceps, and patellar tendons. Two to three beats of ankle clonus were present. She was tachycardic with a regular rhythm. Chest was clear to auscultation. Abdomen was soft, but tender diffusely, and bowel sounds were active.

Initial labs showed WBC 8.5K/μL (47.4% PMNs, 41.7% lymphs, 6% monos, 3.4% eos, 1.1% baso), Hg 14.4 g/dL, and Pt 302K/μL. The CMP was unremarkable, as were the TSH, lipase, hepatitis panel, and ESR/CRP. The urinalysis did not show evidence of a UTI, and urine toxicology screen showed synthetic opioids. Lactic acid was elevated at 3.7. Blood and urine cultures were obtained in the ED. EKG showed sinus tachycardia with a significant amount of movement artifact (Figure 1).

A chest x-ray and CT abdomen/pelvis were unremarkable. The ED consulted Neurology, who recommended an MRI brain/spine. This did not show any acute processes or new multiple sclerosis lesions. Lumbar puncture had normal protein and glucose, and was negative for WBCs and bacteria/fungi on gram stain.

Differential Diagnosis and Hospital Course
Initial diagnostic workup was targeted towards her history of multiple sclerosis and presence of a possible occult infection. She was started on broad spectrum antibiotics (vancomycin and zosyn) since infection was highest on the differential. The negative MRI and absent inflammatory cells in the CSF made active multiple sclerosis very unlikely. Infectious meninges/encephalitis was also unlikely based on those results. Blood/urine culture/CSF cultures were negative for bacteria, fungi, HSV, and VZV.

Since there was no improvement within the first 12-24hrs, other diagnoses were explored. Further history from the husband revealed that with the recent onset of home stressors, the patient had perhaps been taking higher doses of some of her medications.

After reevaluation of her history and clinical presentation, specifically her tremors, hyperreflexia, inducible clonus, agitation, and diaphoresis, a diagnosis of Serotonin Syndrome was made. Since the patient’s home serotonergic medications were already held on admission, she required only supportive management for her autonomic instability and agitation. This was managed primarily with haloperidol, lorazepam, and haloperidol, respectively. The patient’s mental status returned to baseline within the next three days, and she was discharged with improved clinical status and with home health physical therapy to recover from deconditioning.

Discussion
Serotonin syndrome is characterized by a clinical triad of mental status changes, autonomic hyperactivity, and neuromuscular abnormality. There is a significant amount of variability in the presenting symptoms. These can range from mild symptoms, which include akathisia and tremors, to severe symptoms, which include clonus, muscular hypertonicity, and hyperthermia (>40C) (Figure 2). The pathophysiology of serotonin syndrome is based on the hyper-activation of serotonergic neurons. In the central nervous system, they belong in the midline raphe nuclei which are located in the brainstem from the midbrain to the medulla. In the CNS, serotonergic neurons are responsible for wakefulness, affective behavior, thermoregulation, and motor tone. In the PNS, these neurons are responsible for regulation of vascular tone and gastrointestinal motility. This helps to explain the clinical findings in patients suffering from this syndrome.

The diagnosis of Serotonin Syndrome is based entirely on clinical criteria (Figure 3). My patient met the criteria based on the presence of tremor and hyperreflexia, and the presence of inducible clonus, agitation, and diaphoresis. The management consists of removal of the offending agents, and supportive care.

The agitation can be controlled with benzodiazepines, which blunts the hyperadrenergic state (mortality benefits have been shown in animals). For severe cases (extreme hyperthermia, muscular hypertonicity), management may include sedation, neuromuscular paralysis, and intubation to prevent rhabdomyolysis.

References

Images courtesy of Intermountain Medical Center
Takotsubo Cardiomyopathy: A Case Report
Ashley R. Donaldson, MD
Intermountain Medical Center

A 78 year old female with a history of diabetes mellitus, chronic kidney disease and chronic uric tract infections presented to the Emergency Department via EMS following two episodes of hypoglycemia. On the night prior to admission, the patient’s son had difficulty awakening her. He checked her blood glucose, which was 40. An amp of dextrose administered by EMS improved her glucose to 113 and she remained at home the rest of the evening.

At 5:00 the morning of admission the patient was again difficult to arouse. In the ED her blood glucose was 26. She was given another amp of dextrose; her blood glucose increased to 120. Three hours later she was again hypoglycemic with a blood glucose of 21. Following dextrose she was started on a D5W infusion.

The patient reported starting glimepiride 3 months prior. She denied any history of hypoglycemic events but did endure experiencing a “panic attack” one month earlier while getting a CT scan. She stated this episode felt similar to a hypoglycemic event, with racing of her heart, dizziness and lightheadedness.

The morning after admission the patient complained of epigastric pain and shortness of breath. Her pain was similar to that of heartburn. She stated both of these symptoms were new but felt similar to her prior panic attack. She also reported heart palpitations, fevers, and chills, but denied substernal chest pain, pain radiating to her back, nausea or vomiting.

Physical Exam/Diagnostic Studies

On physical exam the patient’s blood pressure was 146/85, heart rate 110, temperature 38.9, and respiratory rate 24. She was a thin, elderly female who appeared to be in moderate distress secondary to dyspnea. She had increased JVD and a 2/6 systolic murmur heard best at the left lower sternal border. Her lungs had bibasilar crackles, respiratory effort was increased, and she was using O2 via nasal cannula at 2L/min. Her abdomen was soft, mildly distended and non-tender to palpation with normoactive bowel sounds present. She was mildly diaphoretic, but no cyanosis, clubbing or edema was noted in her extremities.

Laboratory data demonstrated a troponin elevation of 0.86 and CK-MB of 1.8. Complete blood count showed WBC of 16.8 and hemoglobin in 10.2. Serum chemistries were notable for BUN 185 and Cr 1.70. An ECG was obtained, showing left bundle branch block, unchanged from prior ECG (Figure 1).

History

The differential diagnosis for this patient’s presentation of epigastric pain with dyspnea included acute coronary syndrome, pneumonia, takotsubo cardiomyopathy, and gastroesophageal reflux disease. Given the concern for a cardiac event she was started on ACS protocol.

Over the course of admission her troponin and CK-MB levels continued to rise, peaking at 2.11 and 5.2 respectively, 24 hours after initial onset. Chest x-ray showed no significant cardiopulmonary abnormality (Figure 2). Transthoracic echocardiography results showed an ejection fraction of 33%, segmental wall motion abnormalities, a severely hypokinetic/akinetic right ventricular apex, and tricuspid regurgitation. Myocardial PET perfusion scan was subsequently performed, demonstrating a small, fixed apical defect with-out evidence of peri-infarct ischemia. Apical akinesis was also noted both at rest and with stress, suggestive of takotsubo phenomenon.

She was discharged home on a beta blocker, aspirin, and ACE inhibitor and has been following with Cardiology in the Heart Failure Clinic. It was concluded that her episode of takotsubo cardiomyopathy was likely incited by her hypoglycemic shock on initial presentation.

Differential Diagnosis/Hospital Course

Discussion

Takotsubo cardiomyopathy is a relatively rare event, although its incidence has increased in recent years. It is commonly triggered by one of three inciting factors including iatrogenic causes, neurologic events, and emotional or physical stressors. Iatrogenic causes are numerous, including sympathomimetic, alpha adrenergic, anticholinergic, and para-sympatholytic medications. Similarly, neurologic events that cause catecholamine release and sympathetic activation have been associated with takotsubo. (3) All of these factors can cause an increase in catecholamine levels. The apex of the myocardium has a greater vulnerability to sudden surges in catecholamine levels and thus results in the hypokinesis noted on perfusion scans of takotsubo patients. (3) Emotional/physical stressors are common in the general population, but few patients develop takotsubo, thus indicating predisposing factors also play a role.

Risk factors for takotsubo cardiomyopathy are categorized into cardiovascular (HTN, smoking, HLD, CAD), associated comorbidities (endocrinopathies, malignancy), and endothelial dysfunction, (atherosclerosis, systemic inflammatory disorders, estrogen deficiency). (2) The diagnosis is most common in post-menopausal females, likely because of the decreased estrogen state, as estrogen protects against myocardial damage during ischemia/reperfusion through induction of endothelial nitric oxide synthase.

Although primarily a diagnosis of exclusion, Mayo Clinic has proposed four diagnostic criteria: 1) transient hypokinesis, akinesis, or dyskinesis in the left ventricular mid segments with or without apical involvement, 2) absence of obstructive coronary disease or acute plaque rupture, 3) new ECG abnormalities or elevation in troponin and 4) absence of pheochromocytoma and myocardiitis. (1)

Management of takotsubo cardiomyopathy includes beta blockers, ACE inhibitors, aspirin, diuretics, nitrates and calcium channel blockers, as well as identification and prevention of predisposing factors and inciting events. (4)

References

1. Pollicino, MD, Ph.D, F. C Greco, MD, C Virdis, MD, PhD, G Rosano, MD, PhD, C Giando, MD, and J. C. Kaski, DSc, MD, FRCP. “Takotsubo Syndrome (Oscillating Cardiomyopathy): An Interpreting Clinical Condition in Search of Its Identity.” The American Journal of Medicine.


Figure 1. ECG with left bundle branch block.

Figure 2. Chest X-ray without evidence of cardiopulmonary abnormality.
Downhill Esophageal Varices in a Patient with SVC Stenosis
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History

A 46-year-old Polynesian female with a history of end stage renal disease secondary to hypertension, on hemodialysis, presented to the emergency department with a one day history of epigastric abdominal pain, two episodes of hematemesis, and melonic stools.

One similar episode in 2013; an EGD at that time showed evidence of gastric ulcers. She had previously been on a PPI, but not currently. No recent illnesses or sick contacts. She had taken a total of 1600 mg of ibuprofen over the last week. Previous history of heavy alcohol use 20 years ago, but none currently. There was no known history of cirrhosis or hepatitis. She had a history of a failed renal transplant in 2000, a transplant nephrectomy in 2003, as well as multiple tunneled dialysis catheter placements and bilateral upper extremity AV grafts for dialysis access.

Physical Exam/Diagnostic Studies

On exam, afefe, pulse 100’s, BP 151/100. 90s on room air. Overweight but in no acute distress. Notable facial edema and prominent external jugular veins, as well as dilated veins over her anterior chest. Abdomen was tender to palpation over the periumbilical region, but was negative for ascites or hepatosplenomegaly. 1+ pitting edema in the extremities, bilateral AV fistulas in the upper extremities with a palpable thrill. No neurologic defects, no asterixis.

Initial labs demonstrated a normal white count, hemoglobin of 10.1 (baseline around 15), platelets 205. INR 1.1, albumin 3.3, alkaline phosphatase 87, AST 13, ALT 7. Hepatitis workup was negative.

Endoscopy showed large dilated varices in the proximal esophagus with a ruptured area (Figure 1). CT abdomen/pelvis showed differential hepatic attenuation with intensely enhancing collateral veins of the anterior abdominal wall and recanalization of the umbilical vein (Figure 2). CT chest with contrast showed occlusion of the superior vena cava with collaterals in the anterior thoracic and abdominal wall with retrograde flow of contrast from the right subclavian vein into the collaterals, followed by retrograde flow into a patent recanalized umbilical vein (Figure 3).

Differential Diagnosis and Hospital Course

Initial differential diagnosis included peptic ulcer disease, H. pylori, gastritis, or uremic bleeding. Following endoscopy, esophageal varices secondary to portal hypertension was considered, though patient had no history of cirrhosis and had intact hepatic function on initial labs.

She was monitored briefly in the ICU for re-bleeding and was started on octreotide and PPI drips for variceal bleeding. CT imaging suggested that repeat dialysis catheter placements had led to scarring of the SVC, subsequently causing increased collateral flow and development of varices.

Interventional radiology performed a trans-mediastinal reconstitution of the superior vena cava with 3 stents placed from the subclavian vein to the superior vena cava, with restoration of venous flow. Patient reported improvement in her facial flushing and edema immediately following the procedure and was discharged home.

Discussion

"Downhill" esophageal varices in patients without portal hypertension were first described in 1965. The dilated veins result from obstruction of the superior vena cava, directing blood flow caudally towards the ayzygoes vein or the inferior vena cava. These findings have been observed in various head and neck malignancies by causing SVC compression, as well as systemic vasculitis syndromes such as Behcet's. Iatrogenic causes such as pacemaker insertions and hemodialysis access have also been reported.

The incidence of downhill varices in end-stage renal disease patients is rare, with only 8 cases reports such patients. The 8 patients presented with gastrointestinal bleeding, but had normal liver chemistries. Localized edema and superficial venous engorgement was seen in 4 out of the 8 patients. All 8 patients had histories of angioplasties and AV fistula constructions with revisions, as well as a history of multiple temporary hemodialysis catheters placed in their subclavian veins.

Management is largely aimed at removing the obstruction to allow return of venous flow through the SVC. In dialysis patients, percutaneous radiological SVC angioplasty with or without stent placement was performed in five of the 8 case reports, with three developing restenosis.

References

Causes of UGIB: An Uncommon Presentation in an Elderly Patient
Mark Kaeppler MD, Kevin Whitehead MD
University of Utah, Dept. of Internal Medicine

HIPI
An 85 y/o F with history of multiple DVT/PE, s/p IVC filter, presents from a rehab center with 1-2 days of “coffee ground” stool. She denies abdominal pain, but does endorse dizziness/lightheadiness. This is her third episode of bleeding within the past 4 months. She has undergone an EGD twice, both at an outside hospital, and she’s unsure what was observed.

She was recently admitted with a UTI and AKI/hyperkalemia, thought 2/2 overdiuresis related to her history of HFGPEF. She has been attempting to regain her strength at the rehab facility, though is no longer independent.

Anticoagulation history: 2nd PE by 2009 (provoked), plan for lifelong anticoagulation, on Warfarin for some years, RLE DVT in 2014. IVC filter placed that year. Was stable on Warfarin, later switched to Xarelto; GI bleeding begins, taken off anticoagulation.

Interestingly, she also describes that she experienced recurrent episodes of epistaxis in her childhood/teenage years, but did not otherwise suffer from major bleeding until recently. Her granddaughter experienced bleeding as a postpartum complication.

Initial Work-up
VS. Afebrile, HR 76, BP 135/51, sat’ing well on RA
The patient is an ill-appearing woman, morbidly obese, with limited mobility, though in no acute distress. Inspection of the oropharynx is shown (Figure 1). There is no significant JVD, negative HJR. Cardiopulmonary exam was unremarkable. Abdomen was soft and non-tender. Rectal examination revealed non-bleeding external hemorrhoids. She is globally weak with 2+ pre-tibial edema noted, stable from previous.

DDx and Hospital Course
DDx: PUD, duodenal ulcers, esophageal varices, esophagitis, Mallory Weiss tear, angiodysplasia, malignancy

Adequate access (2X large bore IVs) is obtained in the ED. She is typed and screened and 2 units of PRBCs are made available. She received some IVF, related primarily to dizziness, though orthostatic is found to be negative. She is started on an IV PPI. GI is consulted and plans for an EGD in the morning.

The next day’s EGD findings are included (Figure 2).

Figure 1: Mouth
Figure 2: Distal esophagus, left; Gastric antrum, right

Numerous angioectasias (>30) are identified throughout the upper GI tract. The patient undergoes colonoscopy a day later that identifies further angiodysplasia.

None of the lesions are found to be bleeding; no interventions are performed.

Her hospital course is not otherwise complicated. Her Hgb downtrends, but she does not reach transfusion threshold (7.0 g/dL). She is discharged in stable condition after 4 days, back to the rehab facility.

Outpatient Follow-up
The patient is seen by Dr. Whitehead twice following her admission, related to concern for Hereditary Hemorrhagic Telangiectasia (HHT).

Her Curacao criteria score is 2 (suspected), based on the physical exam findings of multiple telangiectasias and a family member with HHT. As it turns out, her granddaughter had undergone genetic testing, identifying an Endoglin gene missense mutation. While a certain Endoglin gene mutation is considered diagnostic for HHT, this one is not. Her spontaneous epistaxis was considered a minor contributor to establish the diagnosis.

Further management includes:
1. Aggressive treatment of IDA with IV iron repletion.
2. Continue PPI in the setting of the gastric/duodenal/colon angioectasia.
3. IVC filter in place, no plan to remove. No anticoagulation for now Warfarin could be considered. Advised to avoid Xarelto!
4. Consider screening for brain and liver AVM. Currently awaiting abdominal US results, given the known HFGPEF and concern for liver AVM.
5. Screen family members for HHT; attempt to identify others with the variant Endoglin mutation, thus proving it pathologic in her family’s case.

Discussion
Hereditary Hemorrhagic Telangiectasia (HHT), aka Osler-Weber-Rendu syndrome, is an autosomal dominant condition that results in abnormal proliferation of blood vessels. It affects 1 out of every 5-10K people of the general population.

It tends to be a disease of the young and most often presents with recurrent episodes of epistaxis in childhood or puberty. In fact, >95% of those affected suffer from epistaxis by age 40. This tends to contribute more to IDA and problematic bleeding than from a GI source.

Other less common—but often more severe—manifestations of HHT include:
1. Pulmonary AVM, ~50% of patients
2. Stroke, most often 2/2 pulmonary AVM (paradoxic emboli)
3. Cerebral abscess, 2/2 pulmonary AVM (thought b/c of loss of “filter function”)
4. Heart failure, 2/2 liver AVM (high output state)

Interestingly, literature also supports that HHT patients suffer from a hypercoagulable state, which is why we should consider anticoagulation in the future in our patient.

Diagnosis in adults relies on fulfillment of the Curacao criteria, which includes:
1. Spontaneous and recurrent epistaxis
2. Multiple mucocutaneous telangiectasia
3. Visceral involvement
4. First-degree relative with HHT

[0-1 = unlikely, 2 = suspected, 3-4 = definite]

Genetic testing provides confirmation of diagnosis and currently relies on finding one of three pathologic gene mutations: Endoglin, ALK-1 or SMAD4. 15% of families have no identified mutation, but this doesn’t exclude the diagnosis.

Care of these patients relies on having a high index of suspicion to identify high-risk lesions, such as lung and brain AVMs. Coil embolization can be employed to treat symptomatic and anatomically favorable lesions, particularly pulmonary and brain AVMs. Brain AVMs can also be treated with surgical excision and/or radiation therapy.

There are a number of other medical therapies that are being explored as well.

It is important to screen family members. While debate exists regarding when family members should undergo genetic testing, all family members should be screened using the aforementioned Curacao criteria.

References


UpToDate.com, Topic: Hereditary Hemorrhagic Telangiectasia

A special thanks to Dr. Whitehead for his help with this poster!
Right Ventricular Papillary Fibroelastoma
Adam Kilian MD1, Laura Upton-Kilian BA1, Robert Patrick Davis MD PhD2, Jack Morshedzadeh, MD1,3
1Department of Medicine, University of Utah
2Department of Surgery, Duke University
3Department of Cardiovascular Medicine, University of Utah

Introduction
Cardiac papillary fibroelastoma (CPF) is a rare, albeit the 2nd most common, benign cardiac neoplasm. Historically, CPFs were typically described from autopsy findings, although since the introduction of echocardiography, the diagnosis of these tumors has increased in living patients. CPFs rarely contribute to symptoms, although they present potential for serious morbidity, particularly among patients with large, mobile lesions. We report a case of a 69 year-old man who presented with NSTEMI and was found to have a large right ventricular papillary fibroelastoma with a highly mobile stalk.

History
A 69 year-old male presented to an OSH with typical angina and exertional dyspnea of 6 weeks duration. He had a history of 30 py smoking, HTN, DM2, CAD, and AMI s/p 3 stents 15 years ago. His pain felt similar to his past Ml, and his ROS were otherwise negative or non-contributory. ECG showed no signs of ischemia and troponin level was < 0.01 ng/dl. TTE revealed normal biventricular systolic function with LVEF 70%, mild LVH, no valvular abnormalities, and a round echodense structure in the RV approximately 1.8 x 1.8 cm in size. CTA revealed no evidence of pulmonary emboli. The patient was then transferred to the University of Utah Hospital.

Physical Exam / Labs / Differential
Physical exam revealed an alert and comfortable man with a BMI of 29, blood pressure of 120/72 mmHg, heart rate of 55 bpm, an O2 saturation of 97% on room air, and normal exam (Kllip Class I). ECG demonstrated sinus bradycardia, and T-wave inversions in lateral leads. Troponin-I level peaked at 3.59 ng/dl. CBC, CMP, PT/INR, and lipid panel were normal. Blood cultures remained negative after 5 days. Per the ACS protocol, a heparin drip and ASA were started. Clopidogrel was held due to anticipated surgery to remove the mass. Initial differential diagnoses for the mass included: myxoma, papillary fibroelastoma, thrombus, or vegetation.

Imaging
Repeat TTE: 1.5 x 2.0 cm round pedunculated echodense highly mobile mass in mid-distal RV that appears solely connected to moderator band w/o connection to the TV or ventricular myocardium; on contrast images, the mass remained echodense and did not show obvious perfusion.

Further tissue characterization by cardiac MRI: (A) Hyperenhancement of the mass on T2-weighted double inversion recovery sequence suggests the presence of edema. (B) Absence of early arterial enhancement of the mass following gadolinium infusion on a T1-weighted gradient echo sequence suggests it is relatively avascular. (C) Delayed peripheral enhancement with incomplete central enhancement of the mass on phase-sensitive inversion recovery imaging 10 minutes after gadolinium infusion suggests it is fibrous. These findings reassuringly indicated that the tumor was benign; however, significant thromboembolic risk necessitated surgical excision. Coronary angiography revealed multi-vessel CAD. The decision was made to proceed with RV mass resection and coronary artery bypass grafting after optimizing medical therapy.

Discussion
• CPF can arise from anywhere in the heart, but 90% arise from valvular endothelium with a predilection for aortic and mitral valves.1 Although often found incidentally, CPF can present with symptoms caused by embolization of thrombus or the tumor itself as well as outflow obstruction; common presentations include angina, MI, CVA, mesenteric ischemia, acute valvular dysfunction, CHF, syncope, PE, and sudden death.1,4
• Typical echocardiographic features include: (A) The tumor is round, oval, or irregular in appearance, with well-demarcated borders and a homogenous texture; (B) < 2 cm in largest dimension; (C) ~50% have mobile stalks; (D) often associated with cardiac valvar disease.1 Histology confirms the diagnosis, which demonstrates variable amounts of fine elastic fibrils arranged in whorls in a hyaline stroma.2
• Decisions regarding the management of CPF depend on the size, location, mobility, and potential or strength of association of the tumor with symptoms. Patients with CPF who do not undergo curative resection experience a 50% mortality rate due to systemic embolization or outflow obstruction.2 Symptomatic patients and asymptomatic patients with larger (>1cm) CPF, especially if mobile, should be considered for resection.1

References

Acknowledgements: Special thanks to Brent D. Wilson MD PhD for selecting the MRI images and assisting with their captions.

Images courtesy of University of Utah Healthcare and ARUP Laboratories
U.S. resident physician use of smartphones in clinical care
Sonja Raaum MD, Andres Patino MD, Christian Arbelaez MD, MPH, Caroline Milne MD
1 Dept. of Internal Medicine, University of Utah, Salt Lake City, UT; 2 Dept. of Emergency Medicine, Brigham and Women’s Hospital, Boston; 3 Veteran’s Affairs Medical Center, Salt Lake City, UT

Introduction
• Smartphone use in clinical care has dramatically increased over last decade
• Use provides a potential opportunity for clinicians to improve care through efficiency and enhanced access to resources
• Use by clinicians is currently unregulated and there are few studies identifying patterns of use
• Trainees are likely early adapters

Objective
• Describe current patterns of use by resident physicians in primary care specialties

Methods
• Using current literature, a survey was designed and piloted in fall 2013
• IRB approval was obtained
• Online anonymous survey
• Distribution:
  → Utah residents in IM, EM, general surgery and family medicine
  → Brigham and Women’s residents in IM and EM
• Data collected included: demographics, smartphone ownership, frequency and patterns of use, barriers to use and perceived benefits

Results

Ownership, prior experience and perceptions
• 251 (97%) residents owned smartphones
• 246 (98%) of smartphone owners used them in clinical care
• Only 32 (12%) residents attended a medical school that provided or required purchase of smartphone; and only 50 (19%) have received formal training
• 127 (50%) desired further training on clinical use of smartphone apps
• 238 (93%) think smartphone use improves clinical care

Conclusions
• Smartphone ownership and use has increased since last survey of US residents in 2011 1,2
• Patterns of use highlight an incongruence with perceived utility and effect on patient care
• Few residents have been provided formal teaching on smartphones, but half are interested in learning opportunities
• Barriers that could be addressed through teaching include how to utilize resources in limited time, and awareness of availability of free apps through institution access
• Overwhelming majority think smartphone use improves care specifically in areas of diagnostic assistance, quality and patient safety

Future Areas of Study
• Further characterization of internet use
• Effect of interventions on smartphone use
• Further exploration on how smartphones impact clinical care

References

Table 1. Demographics

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Jaundice & abdominal distention in an alcoholic patient

Tara D. Rachakonda, MD, Dustin Armstrong, MD, and Megan O’Hara, MD
Intermountain Medical Center

History
A 44 year old female presented to the Emergency Department with a 2 week history of jaundice and 1 month history of abdominal distention and bilateral lower extremity edema. She had a 15-yr history of alcoholism, consuming nearly one pint of vodka per day and a 20 pack year smoking history. She also reported intermittent blood tinged sputum and dark-colored urine, as well as streaks of bright red blood in two episodes of emesis. Though she was honest about the extent of her alcohol abuse, she demonstrated limited insight into the severity of her condition.

Physical Exam/Diagnostic Studies
On exam, temp was 36.6, BP 98/61, HR 103, RR 18 and O₂ saturation 97% on room air. She was a thin, jaundiced female in no acute distress with a severely distended abdomen with positive fluid wave and shifting dullness. She also had epigastric tenderness to palpation without rebound or guarding. She had several spider angiomas on her chest.

Admission labs revealed a total bilirubin of 19.5, albumin 3.0, AST 206, ALT 21, alkaline phosphatase 422, and ammonia level of 67, CBC was remarkable for a platelet count of 54. Chemistry showed hypokalemia of 2.8, with normal creatinine. Coagulation studies were notable for a PT of 19.9 s, INR 1.7 and PTT of 42 s. Hepatitis panel was negative.

An abnormal, nodular liver with narrowing of the portal and hepatic veins was appreciated on abdominal ultrasound. A CT scan showed diffuse hepatocellular disease with lobulated masses involving the caudate lobe, but without arterial enhancement typically seen in hepatocellular carcinoma (HCC). An MRI demonstrated marked fatty infiltration of the liver with macronodular cirrhosis and fibrosis with no evidence of HCC.

Hospital Course
The diagnosis was severe alcoholic hepatitis with decompensated cirrhosis. MELD alcoholic hepatitis score was 42, with a 3-month mortality rate of 91.7%. Maddrey discriminant function score was 50.

The patient was started on prednisolone; however this was discontinued by hepatology due to the risk of infection. She also received ceftriaxone for SBP prophylaxis, as well as pentoxyfylline, lactulose and albumin support.

On hospital day #3, the patient developed coffee-ground emesis and underwent two upper endoscopies, which revealed a Grade I varix as well as alcoholic gastritis, but no evidence of active bleeding. The patient showed limited clinical improvement. Prior to discharge, her bilirubin reached a peak of 30 and her INR increased to 2.2. Eventually, the patient was placed on hospice and expired 11 days following discharge.

Discussion
Alcohol-related deaths are the 3rd leading cause of preventable deaths in the U.S; an estimated 30 years of life are lost per alcohol-related death.¹ Differential diagnosis for diagnosis for alcoholic hepatitis includes nonalcoholic steato-hepatitis, viral hepatitis, drug-induced liver injury, Wilson’s disease, alpha-1-antitrypsin deficiency and decompensated liver disease with HCC. Initial work-up includes CBC, CMP, coagulation studies, abdominal US, and infection screening; diagnostic paracentesis may be sent to rule out SBP & HCC.

Treatment options include prednisolone when the Maddrey discriminant function score ≥32 to improve short-term survival.² The Lille Model identifies non-responders early during corticosteroid therapy in order to trial alternative treatments.³ Pentoxyfylline may also reduce hepatic-related mortality, particularly from hepatorenal syndrome.⁴

Ultimately, any medical therapy must be coupled with abstinence, along with psychosocial and nutritional support in order to prevent further alcoholic liver damage.

References
A 82 yo Caucasian female with PMH of severe COPD, hypothyroidism, and post-herpetic neuralgia from shingles transferred to Intermountain Medical Center (IMC) from an outside hospital (OSH) for evaluation of R lower leg lesion not improving on antibiotics.

The patient initially presented to the OSH ED 2 weeks prior with a painful R leg wound which she thought was a spider bite. Pain worse with movement, better at rest. Associated symptoms included chronic diarrhea for 6 months and questionable blood per rectum. She denied fevers, chills, CP, nausea, vomiting.

She was treated with vancomycin and discharged. She presented 1 week later, and wound had progressed to 2 cm in diameter with necrotic center with additional 2 cm surrounding erythema. Wound culture was negative.

She was admitted and treated with cefalexin, clindamycin, trimethoprim-sulfamethoxazole and ceftriaxone, all without improvement in the lesions. She was transferred to IMC for more definitive evaluation and treatment.

Patient was admitted to medicine with differential diagnosis of cellulitis, pyoderma gangrenosum, venous stasis, arterial insufficiency, and vasculitis. Dermatology was consulted and performed biopsy, recommending broad-spectrum antibiotics until biopsy results return and IV methylprednisolone.

Biopsy returned citing “epidermal ulceration with dense dermal mixed inflammation including prominent neutrophils." Antibiotics were discontinued, and lesion was noted to improve quite dramatically on IV methylprednisolone (Image 2).

Gastroenterology was consulted and performed colonoscopy with clinical impression “possibly consistent with IBD.” Biopsy showed neutrophilic infiltrate with “moderate active colitis.” C. difficile and giardia antigen were negative. Patient received mesalamine with noted improvement in drainage.

Final diagnosis was pyoderma gangrenosum with inflammatory colitis (likely UC due to improvement following mesalamine).

Pyoderma gangrenosum (PG) is an uncommon neutrophilic dermatosis that is more common in middle-aged adults and women. Lesion starts as a small pustule typically on lower extremities that expands peripherally and ulcerates centrally. Biopsy is useful for to exclude other diagnoses.

Major Diagnostic Criteria (must have both):
1. “Rapid progression of a painful, necrotic cutaneous ulcer with an irregular violaceous and undermined border (1-2 cm growth per day or 50% increase in size within 1 month).”
2. Other cases of cutaneous ulceration excluded

Minor criteria (2+):
1. History of pathergy or clinical cribiform scarring
2. Systemic disease associations
3. Classical histopathology (sterile dermal neutrophilia, +/- mixed inflammation, lymphocytic vasculitis
4. Rapid response to steroid treatment

Recommended first-line therapy is systemic corticosteroids or cyclosporine (Level 3 evidence). Second-line therapy is infliximab 5 mg/kg (level 2 evidence).

Notable disease associations include IBD, hematologic disease (AML, CML, PV, myeloma), RA, Behcet's and chronic acute hepatitis. Workup for comorbidities is recommended in patients with PG.

### References
5. “Pyoderma Gangrenosum.” UpToDate Online Database. Accessed 08/11/14