

2019 ACP Colorado Resident/Fellow Meeting

May 14, 2019

Saint Joseph's Hospital, Colorado



RESIDENT ABSTRACTS

PRESENTED:
MAY 14, 2019

Name: Kylie Adamek, MD

Presentation Type: Oral Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors: Christopher Caruso, MD/MPH

Abstract Title: Congenital Long QT Syndrome as a Cause of Polymorphic Ventricular Tachycardia in Young Adults

Abstract Information:

Introduction: Congenital long QT syndrome (LQTS) is an entity of rare medical consequence in otherwise healthy individuals and may often go undiagnosed without a provoking event. However, the abnormalities of ventricular repolarization significantly increase the risk of life-threatening arrhythmias and sudden cardiac death in healthy young adults. Appropriately diagnosing and treating LQTS has significant implications for morbidity and mortality.

Case Description: A 24-year-old woman with limited past medical history on OCPs presented following a traumatic trimalleolar fracture. Notably, she had no prior history of syncope and no family history of sudden cardiac death. She was taken to the operating room one day after the fracture for open reduction and internal fixation. After receiving one dose of ondansetron, she underwent sedation using propofol and fentanyl. Near the end of the procedure the patient became pulseless; telemetry monitoring showed polymorphic ventricular tachycardia (PMVT). She received two rounds of epinephrine with return of spontaneous circulation. Post-operative labs showed a potassium of 3.1mmol/L and a magnesium of 1.8mmol/L. Initial EKGs after the event revealed a QTc of 515ms and 550ms. Despite holding all QT prolonging medications, including narcotics, her QTc was consistently greater than 550ms on daily EKG. Accordingly, the patient was diagnosed with LQTS. She began nadolol therapy, but despite maximum doses of the beta-blocker she continued to have intermittent salvos of PMVT and frequent PVCs. Therefore, the patient underwent transvenous ICD placement. She tolerated the procedure well, allowing for definitive operative treatment of her ankle fracture. She was discharged on nadolol 40mg twice a day with follow-up in outpatient cardiology clinic.

Discussion: Congenital long QT syndrome is an uncommon channelopathy affecting repolarization of ventricular myocytes. After ensuring that metabolic derangements, medications, or other external influences are not causing a long QT interval, providers should consider congenital LQTS. Though our patient did not undergo testing for a channelopathy, she had a Schwartz score of > 5 due to her QTc > 480ms and history of Torsades de Pointes. This is indicative of a high probability (>80%) of congenital LQTS. Beta-blockers, especially nadolol, are a mainstay of therapy that reduce both sudden cardiac death and syncope by shortening the QT interval. As was the case with our patient, per Heart Rhythm

Society guidelines, ICD implantation is recommended in patients who have a history of cardiac arrest or continue to experience symptoms or signs of impending arrhythmia. This case highlights the recognition of congenital LQTS, as well as the medical therapies that can prevent fatal arrhythmias from reoccurring.

Name: Otavia Peixoto, MD **Presentation Type:** Oral Presentation

Residency Program: Saint Joseph Hospital/National Jewish Hospital

Additional Authors: Rebecca Adams, DO, PGY-1; Tamara Lunghay, MS3; Richard Miranda, MD, FACP

Abstract Title: A Rare Anterior Mediastinal Mass

Abstract Information:

Introduction: Anterior mediastinal masses have an extensive differential, including but not limited to: thymoma, malignancy, substernal goiter, parathyroid adenoma, benign soft tissue mass, fibroma, fibrosarcoma, and hernia. Anterior mediastinal AVMs are exceedingly rare with only 4 cases found in the literature. Other mediastinal AVMs are most commonly found in adult women in their 40's with a history of trauma or congenital disorder.

Case Description: We present a 39-year-old woman with a history of well-controlled asthma and anxiety who presented with two weeks of dyspnea on exertion and, positional, non-radiating chest pain at rest, who was found to have a large anterior mediastinal mass on CTA. Medical history includes a history of trauma (multiple MVAs and cheerleading accidents). She also reported a lifetime of joint laxity, and an increase in epistaxis and menorrhagia over the past few years. Her CTA showed a large anterior mediastinal mass that surrounded her bronchi, aortic arch, pulmonary trunk, and the majority of the heart. PET scan showed increased metabolic activity of the mass, consistent with large AVM versus vascular malignancy. It was thought that due to likely longstanding presence of the mass (since 2014 at least) it was unlikely to be malignant in this patient without other symptoms. Vascular surgery, thoracic surgery, and interventional radiology were all consulted and deemed the mass unresectable, and suggested referral to an interventional radiologist AVM specialist for consideration of a staged embolization procedure.

Discussion: Based on limited case reports, anterior mediastinal AVMs are typically incidental findings or found on evaluation of symptomatic mass effect. Two main risk factors include a history of trauma or Hereditary Hemorrhagic Telangiectasia/Osler-Weber-Rendu Syndrome. Patients with HHT account for up to 87% of all atraumatic AVMs. Diagnosis of mediastinal AVM can be confirmed with contrast CT or MRI, but angiography is ideal for surgical/interventional planning. Conservative management with watchful waiting is favored largely as AVMs are usually incidentally found are asymptomatic. Surgical resection, serial embolization as a gradual procedure, or a combination of the two

are options in the symptomatic patient. All cases in the literature recommended avoiding biopsy secondary to significant risk of bleeding. Of the cases that were biopsied, none revealed clinically significant results. AVMs of the anterior mediastinum carry risks for complications, mainly rupture and thrombosis. There is a relatively low risk for rupture, with only one case reported. The turbulence in the AVM also increases the risk for thrombosis, which should be considered if there is a sudden decline in oxygenation or increased symptoms. Steroids could be helpful in an acute symptomatic episode to help with spontaneous thrombosis.

Name: Luke Clifford, DO

Presentation Type: Oral Presentation

Residency Program: Parkview Medical Center

Additional Authors: Nicole Pisapia, DO, Lirio Polintan MD, FACG, Charles Ruzkowski MD

Abstract Title: The Hematoma Hypothesis

Abstract Information:

Introduction: Hematemesis secondary to bleeding duodenal ulcers is not uncommon. Known etiologies include NSAID use and *H. pylori*. Less commonly seen, however, is the development of an intramural hematoma secondary to duodenal ulcer bleeding. There is a proposed relationship between duodenal ulcerations and celiac disease in the literature, although it is not well understood.

Case: A 71-year-old female with a history of celiac disease presented with hematemesis of one-day duration. The patient reported ten days of progressive nausea, vomiting, and crampy abdominal pain after accidental gluten ingestion preceding her presentation to the emergency department. She compared her symptoms to prior episodes of accidental gluten consumption, but stated this episode was more severe. The patient denied any recent trauma. She reported daily use of aspirin 81mg but denied any other NSAID use, alcohol consumption, or liver disease. Physical exam was unremarkable other than moderate right upper quadrant and epigastric tenderness. Initial labs showed a moderate leukocytosis and macrocytic anemia with an elevated BUN but normal creatinine, INR, bilirubin, and platelet count. CT abdomen without contrast showed an 11.5cm rapidly developing duodenal mass associated with severe biliary distention most suspicious for a duodenal intramural hematoma. Gastroenterology and general surgery were consulted, and the patient was admitted to the intensive care unit. The patient was started on a protonix drip and an EGD was performed which revealed a large duodenal ulcer with two visible vessels and an adherent blood clot. The ulcer was treated endoscopically with epinephrine injections and placement of endoclips for hemostasis. She tested negative for *H. pylori* by stool antigen. She did not require surgical intervention and was ultimately discharged in a stable condition. Her repeat EGD one month later showed a well healing ulcer without signs of active bleeding.

Discussion/conclusion: The etiology of this patient's ulcer is probably related to her aspirin use. Interestingly, there have been a few case reports of duodenal ulcers and even perforations associated with celiac disease. The pathophysiology of this relationship

remains in question to date but we speculate the role celiac disease may have played in combination with chronic aspirin use in the above presentation. Trauma-induced duodenal hematomas are rare, even more so are non-trauma induced duodenal hematomas. Given this patient's history of celiac disease, malignancy was in the differential for her duodenal mass, given the known association with small bowel lymphoma. In spite of that, this was less likely given the rapid growth when compared to prior imaging, EGD findings, and clinical picture of GI bleeding. Overall, this case signifies an unusual presentation for a duodenal ulcer and non-trauma induced duodenal hematoma [likely] related to aspirin use with the possibility of celiac disease contributing to her disease burden.

Name: Samantha J. Allen MD

Presentation Type: Oral Presentation

Residency Program: Saint Joseph Hospital

Additional Authors: Juan N. Lessing MD, FACP; Mel Anderson MD, FACP

Abstract Title: “Doc, I can’t walk:” a reflexive diagnosis

Abstract Information:

Introduction : Thyrotoxic periodic paralysis (TPP) causes intermittent attacks of proximal muscle weakness and, rarely, respiratory muscle failure. Like other forms of periodic paralysis, it is a membrane channelopathy. Its pathogenesis involves a potassium channel regulated by thyroid hormone. Here we present a case of TPP in the clinic, emphasizing its unique characteristics.

Case Description: A 43-year-old woman presented with one week of weakness, palpitations, and heat intolerance. She had a history of panhypopituitarism following Rathke’s cleft cyst resection. Medications included levothyroxine, prednisone, transdermal estrogen, and medroxyprogesterone injections. She had tachycardia (HR 105) and her weight was down seven kilograms from three weeks prior. HEENT, chest, lung, and abdominal exams were normal. Bilateral thighs and calves were tender to palpation. Upper extremity strength was intact; lower extremity strength was symmetrically diminished. She could not rise from a chair or walk without support. Deep tendon reflexes were diminished in the upper extremities and absent in the lower extremities. Neurologic exam was otherwise normal. CBC was normal. ESR was 34 (0-20), CRP was 7.77 (≤ 3), and creatine phosphokinase was 190 (19-131). Potassium was 2.8 with an otherwise normal BMP. TSH was undetectable. Free thyroxine was 2.43 (1-1.85). Her exam and labs suggested a diagnosis of TPP. It was noted that her levothyroxine dose was increased 6 weeks prior due to an elevated TSH. She now disclosed that at that time, she had not been forthcoming about inconsistent levothyroxine use; therefore, the dose increase was inappropriate and caused iatrogenic hyperthyroidism complicated by TPP. Symptoms resolved quickly with potassium supplementation.

Discussion: TPP findings may be suggestive of thyrotoxicosis. However, it is an important distinction to make. A useful distinguishing finding is deep tendon reflexes, which are absent or diminished in TPP but exaggerated in thyrotoxicosis. Treatment is potassium replacement, management of thyrotoxicosis, and treatment of the underlying thyroid disorder.

Name: Amanda Mika, DO

Presentation Type: Oral Presentation

Residency Program: Saint Joseph Hospital/National Jewish Hospital

Additional Authors: Sagarika Sinha, MD

Abstract Title: A Case of Non-Islet Cell Tumor Hypoglycemia

Abstract Information:

Introduction: Non-islet cell tumor hypoglycemia (NICTH) is a rare paraneoplastic syndrome seen in patients with tumors of mesenchymal or epithelial origin, most commonly, hepatocellular carcinoma. The tumor produces incompletely processed insulin-like growth factor-2 (IGF-2) which activates insulin receptors resulting in severe and difficult to treat hypoglycemia.

Case Description: A 64-year-old Vietnamese gentleman with hepatitis B and untreated hepatocellular carcinoma presented with dizziness and generalized weakness. He was found to have a blood glucose of 18 only responsive to a continuous dextrose infusion. CT imaging revealed large liver lesions and extensive cannon ball lesions in both lungs consistent with metastatic disease. He had numerous labs collected while hypoglycemic, which revealed low c-peptide, insulin and proinsulin levels with a normal beta-hydroxybutyrate level. Anti-insulin antibodies were negative. IGF-1 and IGF-2 levels were very low. Given these lab results, other etiologies of hypoglycemia such as insulinoma, exogenous insulin, or anti-insulin antibodies were ruled out. The clinical picture and lab findings were most suggestive of an IGF-2 secreting tumor. Given the rarity of this disease, management was guided mainly by case reports. Per our review, the most effective treatment is removal of the tumor. The case was discussed with interventional radiology, radiation oncology, and medical oncology. The patient was felt not to be a candidate for IR guided embolization, targeted radiation, or medical therapy given his significant extra hepatic tumor burden and dependence on continuous glucose infusion to prevent hypoglycemia. He was started on high dose steroids and had very mild improvement in blood glucose levels. He remained hypoglycemia with any attempt to wean his continuous dextrose 10% infusion however. Octreotide was then attempted in addition to steroids, but had no effect on his hypoglycemia. He was at that time started on recombinant growth hormone (GH) at 5mg subcutaneous daily (16 times a standard dose). Within the first 24 hours there was significant improvement in the patient's blood glucose levels. After two doses of supra-physiologic doses of GH, he was titrated off the continuous glucose infusion. He was discharged home on 5 mg GH daily and 40mg prednisone twice a day. Due to his good performance status and stable glucose levels he was referred to medical oncology for systemic palliative therapy.

Discussion: This case demonstrates the challenges of managing IGF-2 mediated hypoglycemia, and though rare, a potentially lethal complication of metastatic disease. Management of hypoglycemia with oral and subcutaneous medications can allow for safe discharge from the hospital with a manageable home regimen and, in an otherwise fit patient can allow them to be a candidate for

Name: Stephanie Franquemont, DO **Presentation Type:** Oral Presentation

Residency Program: Parkview Medical Center

Additional Authors: Ashley Allemon, DO; Hannah Mirzakhani, DO; Jennifer Forté, DO

Abstract Title: Missing the Vitamin D

Abstract Information:

Introduction:

Hypocalcemia is a known side effect of medications that reduce bone resorption to treat osteoporosis. Certain conditions, including renal impairment and vitamin D deficiency, can exacerbate the risk of this developing. We present a case of a 74-year-old female who was admitted for severe symptomatic hypocalcemia twelve days after receiving her first Denosumab injection.

Case description:

Patient is a 74-year-old female with past medical history significant for osteoporosis, invasive ductal adenocarcinoma (T1N0M0, status post left mastectomy), chronic kidney disease and cognitive impairment that presented to the emergency department with generalized weakness for the last week. She denied chest pain, palpitations, or seizures. Her family reported confusion beyond her baseline. The patient was unable to remember any of her home medications but records showed she had her first injection of Denosumab twelve days prior to presentation. On neurologic exam, she had normal strength and tone but fasciculations were noted in both lower extremities. She had negative Trousseau's sign but positive Chvostek's sign. Laboratory testing revealed a corrected calcium of 5.8mg/dL, 25-(OH) vitamin D of 12.9ng/mL, normal 1,25-di(OH) vitamin D and phosphorus, and appropriately elevated PTH. On outpatient record review, she had a calcium level of 8.7mg/dL two months prior to presentation but never had a vitamin D level checked. She was treated with intravenous and oral calcium supplementation for two days and was discharged home on continued oral calcium and cholecalciferol. Prior to discharge, her corrected calcium level was 7.6mg/dL and her symptoms of tremor and subjective weakness had resolved. One week after discharge, her calcium level remained stable at 7.9mg/dL.

Discussion:

Denosumab is a monoclonal antibody against activation of nuclear factor κ B ligand (RANKL) which inhibits osteoclast formation which reduces bone resorption and increases bone mineral density [1]. This medication has been shown to decrease calcium levels; especially in patients with predisposing risks

for hypocalcemia such as vitamin D deficiency and chronic kidney disease. The exact etiology of these factors increasing the risk of hypocalcemia with Denosumab is not clear [2].

This case highlights the importance of diagnostic testing and screening prior to determining ideal treatment for the specific patient. This patient had vitamin D deficiency, chronic kidney disease, medication non-compliance, and low medical literacy all of which should be included in the discussion of the ideal medical regimen. A checklist can be implemented to increase compliance with laboratory testing for vitamin D, calcium, and renal function prior to starting Denosumab. An informational packet can also be presented to provide a resource for patients to evaluate. Our clinic has implemented this check list and a contract for the patient and provider to sign prior to starting Denosumab to ensure appropriate testing has been done and understanding has been achieved.

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Name: Amy Beeson, MD

Presentation Type: Oral Presentation

Residency Program: University of Colorado Medical School

Additional Authors: Jonathan Schultz, MD, Josh Gannon, Julie Venci, MD, Anne Frank, Kaylynn Aiona

Abstract Title: Evaluating the cascade of care for latent tuberculosis infection at an urban primary care clinic

Background: Screening and treatment of latent tuberculosis infection (LTBI) is a key strategy for TB elimination in the United States. Screening and treatment based in primary care clinics may be beneficial for patients and allow for a broader reach than centralized TB clinics alone; however, the optimal strategy for scaling up testing and treatment in this setting is uncertain. From 2016-2019, providers at Denver Health's Federico Peña Clinic developed a new protocol for clinic-based LTBI treatment. We report on early outcomes of this intervention.

Methods: A retrospective chart review was performed from April 11, 2016 to Dec 18, 2019 to identify patients at risk of LTBI based on birth in a high incidence country, defined as active TB incidence of >20/100,000. Among these, patients with an interferon-gamma release assay (IGRA) test result were identified. Charts of patients with positive IGRA results were then reviewed in depth to characterize the treatment cascade, including evaluation for active TB, clinical decision to treat or not, treatment initiation (defined as an initial prescription for one of three LTBI treatment regimens) and treatment completion (defined as documentation of the final monthly monitoring visit). When patients did not start treatment or discontinued treatment without documentation of clinical rationale (such as pregnancy or low risk of progression), they were considered lost to follow up.

Results: There were 94,859 patient visits during the evaluation period, including 23,102 individual patients from 120 different countries. Of these, 6,566 (28%) were identified as at-risk for TB. 1,523 (23%) of these were screened for LTBI with an IGRA, of which 172 tests were positive (11%). Among patients with positive IGRA, 153 (88.9%) were evaluated with a chest x-ray, of which 145 (94.7%) did not show evidence of active TB, while 7 were indeterminate and one identified active TB. Of those with negative x-rays, 97 (66.9%) initiated treatment, 53 (36.5%) completed treatment and 25 (17.2%) are currently on treatment. Among those with positive IGRA who did not initiate treatment (N=75, 43.6%), a clinical decision not to treat was documented in 43 cases (57.3%) while 25 (33.3%) were lost to follow-up. Of 21 pregnant women with positive IGRA, only one had a plan to initiate treatment, while 6 remained pregnant, precluding treatment, and a majority (14) were lost to follow up post-partum.

Conclusions: While urban primary care clinics may contribute meaningfully to large-scale LTBI testing and treatment efforts, these interventions are complex. We noted significant gaps in both testing and

treatment at the Peña clinic, with the largest gap in testing individuals at risk. Forthcoming improvement efforts may include standardized screening, strengthening of the clinic-wide protocol to increase treatment initiation when indicated after a positive IGRA result, and targeted efforts to improve post-partum follow-up.

Name: Bree Smouse, MD **Presentation Type:** Oral Presentation

Residency Program: Saint Joseph Hospital/National Jewish Hospital

Additional Authors: Kevin Ozment, MD

Abstract Title: Semicircular Canal Dehiscence Masquerading as a Psychiatric Disorder

Abstract Information:

Introduction: Superior canal dehiscence syndrome (SCDS) is a rare disorder of the inner ear characterized by Tullio phenomenon, autophony, pressure-induced vertigo, and hyperacusis.

Case Description: Ms. M is a 34-year-old female with a history of multiple sclerosis who presented to behavioral health at age 20 with complaints of “hearing odd noises” that caused significant distress, loss of sleep, and anxiety. She was diagnosed initially with generalized anxiety disorder and treated with SSRIs. As the symptoms worsened she began hearing grinding noises when turning her head side to side or when moving other joints in her body such as her elbows. She started hearing a “squishing sound” whenever she blinked her eyes and could hear her heart beat as though it were inside her skull. She returned to behavioral health, who diagnosed bipolar affective disorder due to depression and insomnia masquerading as mania. In early 2019 she developed difficulty with ordinary noises such as a knock at the door causing vertigo and she could not go outside without earplugs. Her PCP finally sent her for an audiology evaluation that showed decreased sensitivity to air conduction stimuli and increased sensitivity to bone conduction stimuli. Vestibular-evoked myogenic potential testing indicated that the patient might be suffering from superior canal dehiscence. Sub-millimetric temporal bone CT confirmed the diagnosis. She was scheduled for a right sub-temporal craniotomy with neurosurgery who repaired the defect in the right semicircular canal. She has had near-total resolution of symptoms on the right side and intends to proceed with left-sided repair.

Discussion: Proper diagnosis of superior canal dehiscence syndrome (SCDS) is vital as treatment with neurosurgical repair is highly successful and the differential diagnosis is broad. It is a considerably under diagnosed disease, as it was unknown prior to 1995 and effective imaging techniques have only been developed in the past two decades. High clinical suspicion is important, as the diagnosis of SCDS must be made radiographically. SCDS has been known to mimic otologic disorders such as Meniere’s disease, Eustachian tube pathology, and otosclerosis. Patients with SCDS have described years of misdiagnosis, typically seeing numerous physicians in an attempt to find an etiology. As the symptoms are often debilitating, many patients have described falling into depression. Many of the referrals for ENT and audiology have come from psychiatrists and PCPs suspecting that something larger than mental illness was at play. Given the often-unbearable symptoms, the multitude of unproductive doctor’s visits, and the frustration of the social limitations of the disease it is unsurprising that patients may first be diagnosed with depression or another psychiatric disorder. As most patients initially present to their PCP, increasing awareness of the disease will hopefully prompt physicians to consider SCDS and initiate appropriate work up much sooner in the disease course.

Name: Stephanie Franquemont, DO
Presentation

Presentation Type: Oral

Residency Program: Parkview Medical Center

Additional Authors: Spencer Grigg, DO; Daniel Lee, DO

Abstract Title: Sugar Shakes

Abstract Information:

Introduction: Hemiballism-hemichorea is a rare movement disorder with multiple known etiologies. It is characterized by high-amplitude, arrhythmic motions involving just one side of the body. We present a case of a 65-year-old female who presented to the hospital with uncontrollable movement of the left side of her body in the setting of poorly controlled diabetes with hyperglycemia.

Case description: Patient is a 65-year-old female with past medical history significant for type 2 diabetes mellitus, hypertension, and hyperlipidemia that presented to the emergency department with complaint of uncontrollable, violent movements of her left upper and lower extremity. She had no other focal neurologic complaints. She did present with hyperglycemia >400mg/dL and reported labile control of her diabetes over the proceeding months with her most recent hemoglobin A1c measured at 17.1% two months prior. A full neurologic exam demonstrated that the patient was alert and oriented appropriately, cranial nerves II-XII were intact bilaterally, and sensation was intact throughout. Motor exam revealed constant, arrhythmic movements of the left upper and lower extremity. Proximal and distal portions of both limbs were involved. No adventitious movements were noted on the right side. Formal strength testing and reflex testing was not possible with the ballismus. The patient underwent a magnetic resonance imaging (MRI) of her brain which showed not acute intracranial pathology. Ultimately, the movements improved on this admission with improvement of her blood sugar control. She was readmitted a short time later with recurrence of non-ketotic hyperglycemia and hemiballismus-hemichorea on her left side. At that time, a repeat MRI of her brain showed T1 hyperintensity of the right putamen. She was started on Tetrabenzine with improvement of her uncontrolled movements. Since discharge, she has been unable to continue Tetrabenzine due to cost and is now managing her symptoms with closer glycemic control.

Discussion: Hemiballism-hemichorea is a rare movement disorder and an even rarer complication of hyperglycemia. The estimated prevalence of this condition is <1/100,000 of patients with nonketotic hyperglycemia. The classic presenting triad includes: unilateral involuntary movements, contralateral striatal abnormalities on neuroimaging, and hyperglycemia [1]. The pathophysiology for hemiballism-hemichorea is not well understood. Proposed mechanisms include exacerbation of chronic small vessel

disease, microhemorrhages, or damage due to hyperviscosity of the blood with hyperglycemia. Patients with other risk factors for cerebrovascular disease (hypertension, hyperlipidemia, carotid stenosis) appear to be a greater risk [2,3]. Hemiballism-hemichorea does not appear to occur with acute hyperglycemia but rather prolonged poor glycemic control. The treatment of hemiballismus-hemichorea is anchored in treatment of the underlying cause. Stabilization in glycemic control can stop the adventitious movements. Medical treatment with neuroleptics or central-monoamine depleting agents, such as Tetrabenzine, can be used to treat severe or debilitating symptoms though they are often not necessary [3].

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Name: Andi Hudler, MD

Presentation Type: Oral Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors: Natalie Held, MD, Samantha King, MD, Mark Kearns, MD

Abstract Title: Streptococcal Purulent Pericarditis Presenting as Cardiac Tamponade with Obstructive Shock

Abstract Information:

Introduction: Bacterial pericarditis is a rare condition that carries a mortality rate of up to 40%. While historically bacterial pericarditis was most frequently caused by *Streptococcus pneumoniae*, this has now become a rare etiology due to widespread use of antibiotics and immunizations. In this case, we describe a patient with *Streptococcus pneumoniae* purulent pericarditis causing obstructive shock that was successfully treated with drainage, antibiotics, and pericardectomy.

Case Description: A 59-year old man presented with three days of chest pain. No further history could be obtained as the patient rapidly developed respiratory failure necessitating emergent endotracheal intubation. Vitals at admission were temperature 36.8°C, heart rate 108 beats per minute, and blood pressure 111/77 mm Hg. Heart sounds were normal, there was no jugular venous distension, and lungs were clear on exam. Labs were pertinent for white blood cell count 28.4 k/uL, lactate 6.0 mmol/L, C-reactive protein 215 mg/L and troponin 0.61 ng/mL. Chest x-ray showed perihilar prominence and Kerley B lines. ECG showed ST elevations in the inferior and high lateral leads with mild PR depression. Urgent coronary catheterization revealed diffuse atherosclerotic disease without a culprit lesion. Echocardiography demonstrated a small pericardial effusion.

Ten hours after admission, the patient developed respiratory distress and tachycardia. Labs were significant for a lactate of 19 mmol/liter. Bedside critical care echocardiogram revealed a large pericardial effusion with diastolic right atrial and right ventricular collapse consistent with tamponade. Emergent pericardiocentesis yielded 600 mL of purulent pericardial fluid that grew *Streptococcus pneumoniae*. Blood and respiratory cultures also grew *Streptococcus pneumoniae*. CT scan of the chest demonstrated small simple effusion and mild atelectasis. Notably there was no radiographic evidence of pneumonia or empyema. He was treated with ceftriaxone and pericardial drain placement, ultimately undergoing a pericardial stripping hospital day fourteen. The patient received a four-week course of ceftriaxone.

Discussion: This patient developed pneumococcal purulent pericarditis in the absence of radiographic evidence of pneumonia or empyema, which was successfully treated with antibiotics and drainage of the pericardial space. This case highlights the importance of early recognition and treatment of purulent pericarditis. Furthermore, it highlights the importance of critical care ultrasonography to expedite the evaluation of shock. Diagnosis can be difficult as patients often present with nonspecific symptoms and can deteriorate rapidly due to cardiac tamponade and

septic shock. Appropriate management includes a prolonged course of antibiotics along with drainage of the pericardial space.

Name: Windian Jubair, MD

Presentation Type: Oral Presentation

Residency Program: Parkview Medical Center

Additional Authors: Stephen Groendyk, DO, Daniel Long, DO, B. Aruni, MD

Abstract Title: A Case Report of Spontaneous Hemopericardium as a result of Anticoagulation

Abstract Information:

Introduction: Rivaroxaban has been an increasingly popular direct oral anticoagulation (DOAC) for the prevention of thromboembolic stroke in patients with atrial fibrillation. Of the adverse effects of this medication, the risk of major bleeding is the most serious. This is documented to occur at a rate of 3.3% [1]. The documented locations in the various studies such as ROCKET AF trial, Bleeding Events in the Pooled Analysis of EINSTEIN DVT and EINSTEIN PE studies include Intracranial, retroperitoneal, intraocular, and intraarticular bleeding. With the increasing use of this medication, other locations of life-threatening bleeding are being reported. To date, there have been 4 prior case studies that have described spontaneous hemopericardium as a result of Rivaroxaban use [2,3,4,5]. In this case study, we present yet another episode of this rare complication.

Case Presentation: A 91-year-old female with past medical history of atrial fibrillation on Rivaroxaban, presented with a 7 days history of worsening shortness of breath and fatigue. She was initially suspected to have pneumonia at an urgent care clinic and started on azithromycin. When this failed to resolve her symptoms, she presented to the ED where a CT chest diagnosed a large pericardial effusion. The diagnosis was then confirmed by echocardiogram, without evidence of tamponade. Rivaroxaban was held, pericardiocentesis was performed and 1100cc of serosanguineous fluid was drained. Her shortness of breath resolved. Pericardial fluid analysis showed presence of both red and white blood cells with no evidence of malignancy. The most likely causative factor was determined to be pericardial hemorrhage, a rare complication of anticoagulation therapy. The pericardial drain was removed, she was discharged home with close follow up and without anticoagulation for her paroxysmal atrial fibrillation.

Conclusion: Of the 4 other documented cases 3 of them had potentiating factors of major bleeds such various CYP3A4 inhibitors or potential confounding pericardial diseases. This is only the second case in history of spontaneous hemopericardium due to anticoagulation in the absence of any other risk factors. Rivaroxaban has been approved for thromboembolic prophylaxis in a fib and is widely used for this indication. as use becomes widespread, a growing number of rare complications have been documented. it is vital to be vigilant when starting DOAC therapy for bleeding events in uncommon locations.

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Name: David Kerbl, DO

Presentation Type: Oral Presentation

Residency Program: Sky Ridge Medical Center

Additional Authors: Gregory Hicks, MD, and Jonathan Sherman, MD

Abstract Title: Platypnea-Orthodeoxia Syndrome: A Case Report of a Rare Clinical Disease

Abstract Information:

Introduction: Platypnea-Orthodeoxia Syndrome is a rare clinical condition characterized by dyspnea and hypoxemia when assuming a seated or standing position. It is caused by an intracardiac shunt, pulmonary arteriovenous shunts, or ventilation-perfusion mismatch in the lungs.¹ Cardiac causes include a patent foramen ovale (PFO), atrial septal defect, or atrial septal aneurysm.² Diagnosis is often challenging, however there are several case reports in the literature of patients who are cured of their dyspnea and hypoxemia after repair of the intracardiac shunt.¹⁻⁶

Case Description: An 80-year-old male with a history of a PFO, coronary artery disease, bilateral pulmonary emboli, chronic obstructive pulmonary disease (COPD) and pulmonary hypertension presented to our center with repeated admissions for hypoxemia and dyspnea related to physical activity. The patient was noted to have near normal diffusion capacity on his pulmonary function testing, making COPD unlikely to be the cause of his hypoxemia.

The patient underwent right heart catheterization (RHC) in October 2019 demonstrating a right atrial pressure of 19/11 with a mean of 10mmHg and 58% oxygen saturation. Left atrial pressure was noted to be 20/11 with a mean of 10mmHg and 95% oxygen saturation in both the LA and periphery. Right ventricle end-diastolic pressure was 20mmHg and pulmonary artery pressure was 88/28 with a mean of 50mmHg. As the patient was supine for the duration of the procedure, no significant shunt, either right to left or left to right was appreciated. Interestingly, the patient was then noted to have marked hypoxia while recovering from RHC when he assumed a seated or standing position.

At this point, it was hypothesized that the patient may be suffering from platypnea-orthodeoxia syndrome and was subsequently referred for transesophageal echocardiogram (TEE). A small amount of right to left flow was noted at baseline across his PFO in the supine position. However, upon assuming the seated position, the patient became markedly hypoxic with significantly increased Doppler flow across a slightly enlarged PFO. The patient underwent PFO closure in November 2019 with no evidence of residual shunt on TEE and with complete resolution of symptoms.

Discussion: In the setting of elevated right atrial pressures at baseline, right to left shunting is postulated to arise by transient fluctuations in right atrial (RA) pressures coupled with diminished RA compliance when assuming the seated position. The clinical scenarios involving hypoxic lung disease, decreased compliance of the right heart, or increased right-sided filling pressures allow for this transiently increased elevation in RA pressures.⁷ The diagnosis of platypnea-orthodeoxia remains challenging and can be easily missed via a “gold standard” right heart catheterization. A

high clinical suspicion is paramount in establishing the diagnosis, as appropriate intervention often results in complete resolution or improvement of symptoms in a vast majority of patients.⁷⁻⁹

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Name: Kylie E. Adamek, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors:

Abstract Title: I'm sensing a pattern here

Abstract Information:

Learning Objectives:

- Recognize medication use as a cause of Brugada pattern
- Distinguish between Brugada pattern and Brugada syndrome

Introduction: Brugada syndrome is a major cause of sudden cardiac arrest and sudden cardiac death in the structurally normal heart. Distinguishing Brugada syndrome from the EKG characteristics that often accompany it has important clinical implications.

Case Description: A 60-year-old male with a medical history of Parkinson's disease, depression, and anxiety presented to the hospital following an intentional drug overdose. His wife reported that he had taken 7 pills of zolpidem, 5 pills of lorazepam, and several (>10) pills of paroxetine approximately 2 hours prior to arrival. He denied any chest pain, shortness of breath, or dizziness. Notably, he had no history of syncope and no family history of sudden cardiac death. Vital signs and physical exam were unremarkable. Labs demonstrated mild hyponatremia but were otherwise normal, including troponin. An EKG was performed, demonstrating a down-sloping ST elevation and T-wave inversion in V1 (Figure 1). EKG was repeated approximately 4 hours later and was found to have coved ST elevations, T-wave inversions, and a pseudo-right bundle branch pattern in V1 and V2 (Figure 2). These findings were repeated on subsequent EKGs over the next 36 hours. The patient remained asymptomatic. On day 3 of admission, the abnormal EKG findings had resolved (Figure 3). The patient's hospital course was uneventful and he was discharged to an inpatient psychiatric unit.

Discussion: Brugada syndrome is a sodium channelopathy involving cardiac myocytes that increases the risk of ventricular arrhythmias. Notably, it is defined by both characteristic EKG findings as well as clinical criteria. While multiple EKG abnormalities can be associated with Brugada syndrome, the classic finding is a coved ST segment in >1 of V1-V3 followed by a negative T wave. Taken with a history of sudden cardiac death, sustained ventricular arrhythmias, or other associated clinical criteria, it is classified as Brugada syndrome. Should none of the aforementioned clinical criteria be met, it is referred to as Brugada pattern. As was the case with our patient, Brugada pattern has been shown to be elicited in the presence of many classes of drugs. While it is more common to occur with medications that block cardiac sodium channels, it has also been demonstrated in many antidepressants, including paroxetine. This case highlights the importance of the clinical history in those presenting with typical EKG findings of Brugada syndrome as well as the consideration of Brugada pattern in those with possible provoking medications.



Figure 1. EKG on admission



Figure 2. EKG 4 hours after admission



Figure 3. EKG 72 hours after admission

Name: Brian Daily, MD

Presentation Type: Poster Presentation

Residency Program: Saint Joseph Hospital/National Jewish Hospital

Additional Authors: Katie Kopel, MD

Abstract Title: An Unusual Case of Miotic Pupils

Abstract Information:

Introduction: Common etiologies for miotic pupils include medication toxicity (opioids, benzodiazepines, clonidine, pilocarpine and organophosphates), pontine infarct, uveitis, Horner's syndrome and neurosyphilis. We present an unusual case of miotic pupils.

Case Description: A 67-year-old man with dementia and alcohol use disorder was brought to the ED by ambulance after his family became concerned about increased confusion, ataxia and visual hallucinations. EMS noticed he had miotic pupils, but naloxone was ineffective.

- Vitals: 90.6 F, HR 66, BP 149/94, RR 20, 96% on room air
- Exam: In the ED he was agitated, hallucinating and pulling out IV lines. Head was atraumatic. No neck stiffness. Miotic pupils bilaterally. Weak extraocular movements. Moving extremities spontaneously. Following some commands. RRR. CTAB.
- Labs: CBC unremarkable, sodium 141, potassium 2.5, glucose 66, magnesium 1.5, total bilirubin 0.4, AST 138, ALT 43, albumin 2.2.

Hospital Course: on admission the patient was suspected to have Wernicke's encephalopathy. Unfortunately, due to a nationwide shortage of IV thiamine, he was not able to receive IV thiamine until hospital day 6. During this time other potential etiologies were ruled out. CTA head and neck, and MRI brain, were negative for a pontine stroke. TSH and free T4 were normal, and a random cortisol was normal. Serum syphilis antibody was negative. CSF studies were negative for infection. He was initially delirious, with alternating agitation and somnolence, but this improved with IV thiamine, as did his hypothermia, hypoglycemia and extraocular muscles. A second dose of naloxone was given in vain, but after an extended course of IV thiamine his hypothermia and miotic pupils eventually resolved.

Discussion: Wernicke's encephalopathy (WE) is classically characterized by altered mental status, ophthalmoplegia and ataxia. The more sensitive Caine criteria requires only two of these, or one of these plus malnutrition. Patients with WE can also develop hypothermia and miotic pupils. Most patients have alcohol use disorder, although many other medical conditions can cause malnutrition and WE. The following information on treatment of WE is from an excellent 2016 review article by Flannery et al. WE is caused by thiamine deficiency. Normally the body contains 30-50mg of thiamine, with a required daily intake of 1.5mg. Since alcoholics have very poor GI absorption, oral replacement is woefully inadequate, accomplishing uptake of only 5mg per day, so IV replacement is needed. Flannery et al. recommend 200mg-500mg every eight hours for three days, along with IV folate and magnesium. Although it is often said that thiamine must be given before dextrose, the evidence for this rule is minimal, and the risks of hypoglycemia must be considered as well.

Name: Ashley Allemon DO **Presentation Type:** Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Lauren Sanders DO, Andrew Van Boening DO, Muath Dawod MD,

Abstract Title: More than just a toothache: a rare site of metastatic cancer

Abstract Information:

Introduction: Colon cancer is one of the most common malignancies with an estimated incidence of 100,000 new cases this year. Furthermore, it is estimated that approximately 33% of patients diagnosed with colon malignancy develop metastatic disease. The lungs, liver and axial skeleton are the most common sites of metastases. Spread to the oral cavity including the gingiva is extremely rare, accounting for less than 1% of cases in colon cancer and thus can be very challenging to diagnose. Here we report a case of heavily treated metastatic colon cancer patient who developed gingival metastasis.

Case Presentation: A 53 year old woman underwent segmental resection of the colon in 11/ 2005 and was diagnosed with stage IIIB colon adenocarcinoma. She received adjuvant chemotherapy with oxaliplatin and fluorouracil for a total of six months. However she developed metastatic disease to the liver, lungs and peritoneum in 12/2010 and was treated with different lines of chemotherapy as well as with external beam radiotherapy. In 8/2019 she started to have mouth pain that was getting progressively worse and later associated with non-healing maxillary gingival ulcer. Eventually she had dental evaluation with biopsy of the ulcerated area showing malignancy consistent with metastatic colon adenocarcinoma.

Discussion: Oral cavity metastasis in solid malignancies, including colon cancer, is extremely rare and is associated with poor prognosis. Case series reported median survival ranging between 3.7 and 8.3 months. Patients usually present with pain, bleeding or non-healing ulcer. As symptoms are not specific, diagnosis requires thorough physical exam and high index of suspicion. Once diagnosis is confirmed treatment options in terms of resection, external beam radiotherapy or chemotherapy need to be considered depending on the extent of the disease and other sites of involvement. If oral metastasis is discovered, it is predominantly found in the mandible, however, our patient was found to have a metastatic lesion to the maxilla making her case even more unusual. Our patient received external beam radiotherapy for palliation as the lesion was painful and unresectable with good pain relief. In conclusion this report describes a case of colon cancer with gingival metastasis which is extremely rare. It emphasizes the importance of thorough examination and prompt biopsy to establish the diagnosis in a timely manner so proper treatment can be initiated.

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Name: Samantha Allen, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado Medical School

Additional Authors: Elena Lebduska, MD

Abstract Title: Secondary Hypertension: An Essential Diagnosis

Abstract Information:

Introduction: Hypertension is a ubiquitous condition. At the time of diagnosis of hypertension, all patients should undergo a basic workup for risk stratification and screening for secondary hypertension. Renal disease is one of the most common causes of secondary hypertension and should be evaluated with serum and urine labs, and ultrasound to assess for obstruction and evaluate kidney size and morphology. Here we describe a case of autosomal dominant polycystic kidney disease diagnosed in a patient with no known family history of the disease who presented with mild hypertension.

Case Description: A 58-year-old man presented to his new PCP to establish care. He had recently visited a health fair and was told his blood pressure was high. He had not seen a doctor for ten years and had no complaints. He denied any past medical history and was not on any medications, including NSAIDs. He was a current ten pack-year smoker but denied alcohol and drug use. He worked as a landscaper. His mother had lung cancer and his father had COPD; both were heavy smokers. Physical exam was notable for blood pressure 144/94 and BMI 27; exam was otherwise normal. Labs were notable for serum creatinine 2.7 (GFR approximately 30) and hemoglobin of 13.3 with MCV 92; lipid panel was normal. Urinalysis showed moderate protein; spot urine protein:creatinine was 0.2. EKG was normal. He underwent renal ultrasound, which showed normal bladder and polycystic kidneys, and MRI, with findings consistent with autosomal dominant polycystic kidney disease (ADPKD).

Discussion: Initial workup to evaluate for target organ damage, screen for a limited number of causes of secondary hypertension, and assess cardiovascular risk is indicated at the time of diagnosis of hypertension. This should include serum chemistry, urinalysis, lipid panel, and EKG. A finding of elevated serum creatinine should prompt urinalysis with microscopy as well as serum and urine electrolytes. Renal ultrasound should be considered to assess size, morphology, and symmetry, and rule out obstruction. In this case, obstruction was considered likely given the patient's age-associated risk of BPH. Obstruction was ruled out with ultrasound, and a surprising and important diagnosis was made. Classically, a diagnosis of ADPKD is made in a patient with a positive family history and is heralded by flank pain and palpable kidneys on exam. However, up to 25% of patients with the disease have a negative family history, and the majority of patients are asymptomatic. ADPKD is the cause of less than 1% of cases of secondary hypertension. Conversely, hypertension is the most common clinical manifestation of ADPKD and is often present before renal impairment. Blood pressure management is key in slowing the decline of renal function in ADPKD.

Name: Tulsi Shah, MD

Presentation Type: Poster Presentation

Residency Program: Saint Joseph Hospital/ National Jewish Hospital

Additional Authors: Sagarika Sinha, MD

Abstract Title: A benign immune activation leading to multi-organ failure

Abstract Information:

Introduction: Hemophagocytic lymphohistiocytosis (HLH) syndrome is a disease process characterized by progressive unchecked immune activation, leading to an over-activation of normal histiocytes and lymphocytes. HLH can present as an inherited condition or as acquired, following a trigger (eg viral illness, lymphomas or autoimmune conditions). In this report we present a case of a previously healthy male who quickly progressed to multi-organ failure likely secondary to HLH.

Case Description: A 63-year-old Eritrean male with relevant past history of idiopathic T cell immunodeficiency presented initially for progressive malaise, weakness and B symptoms. His initial exam was notable only for cachexia, generalized weakness, and wide based gait. An extensive work up for his symptoms was notable for pancytopenia, with lymphopenia worse than baseline, an infectious work up that was negative, elevated ferritin, a CD25 level that was elevated to 99,600 pg/ml, and splenomegaly on abdominal CT scan. A bone marrow biopsy was read as having B cell lymphoma without evidence of hemophagocytosis. Given his lab findings he was diagnosed with HLH, possibly triggered by underlying lymphoma, and started on chemotherapy. Despite early therapy, his hospital course was complicated by shock and lactic acidosis that eventually progressed multi-organ failure and cardiac arrest. He was transitioned to comfort care and passed away.

Discussion: The pathophysiology behind HLH is not well understood but currently believed to be an inappropriate immune reaction with overactive T cell and macrocyte activation leading to systemic inflammatory response. The diagnosis is made by the presence of five of the following features: fever more than seven days, splenomegaly, cytopenias, hypertriglyceridemia or hypofibrinogenemia, hemophagocytosis (as demonstrated by tissue biopsy), elevated ferritin level, or elevated CD25 (aka sIL2ra). Our patient, while having a negative bone marrow biopsy, met six of the above criteria.

Currently there are no prospective trials to guide treatment of HLH in adults. The approach to treatment is generally to treat any underlying source and control the immune activation. Case reports had shown response to etoposide in few patients with HLH and after larger studies is considered standard of care. Etoposide containing chemotherapy should be considered particularly for patients with lymphoma triggered HLH as in our patient.

Overall, HLH has a high mortality rate especially without treatment, with average survival about two months from diagnosis. This is increased in cases of malignancy-associated HLH. Largely this poor prognosis is related to delays in diagnosis. Treatment should not be delayed, though despite treatment many patients have poor outcomes.

Name: Andrew Van Boening, DO **Presentation Type:** Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Ashely Allemon, DO, Maksim Grachev, DO, William Grigg, DO, Michael Harris, Dom Widian Jubair, MD, Meghan Timmerman, DO

Abstract Title: From the Lung to Finger: Acrometastasis in a Community Hospital

Abstract Information:

Introduction: Acrometastasis; metastases to distant bone, is a rare presentation of malignant disease. Overall, 30% of metastatic disease involves the bone, with about 1 to 3% of the total cases in the hand. Approximately 21% of the cases of acrometastasis to the hand occurs in the distal phalanx with the primary metastatic disease most commonly originating from the lung, followed by the kidneys and breasts. The following illustrates a case that initially presented a finger ache that was found to have metastatic lung cancer.

Case Presentation: The case began with a 78-year-old female with a known history of stage III lung cancer with metastases to the brain that presented to her primary care physician for “finger ache” that had begun approximately 2 months prior. She was treated with antibiotics for a presumed infection. After little improvement with antibiotic therapy, the site was subsequently incised and drained; no drainage was expressed. Given the lack of resolution, she presented to the emergency department with a chief complaint of worsening pain of her left second phalanx. At the time, there was concern for osteomyelitis versus metastatic disease to the joint. An MRI demonstrated near complete destruction of the second distal phalanx. Orthopedic surgeon was consulted; after extensive discussions with the patient and family, an amputation of the area was performed. Pathologic evaluation of the phalanx demonstrated poorly differentiated squamous cell carcinoma, deeply invasive.

Discussion: In a patient with a known history of metastatic disease, there are many clinical factors to bear in mind when new symptoms arise. The incidence of metastatic disease to the hand is rare; it accounts for about 0.1% of metastatic disease to the bone. Nevertheless, it should be considered in the differential. While the exact pathophysiology is still unclear, it is thought to be secondary to tumor cell migration triggered by proinflammatory cytokines. Acrometastasis carries a poor prognosis with an average life expectancy of about 7 months. Even with an overall poor prognosis, early detection and diagnosis can allow for proper treatment planning and improvement of the quality and possible quantity of life.

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Name: Casandra Duarte, MD

Presentation Type: Poster

Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors: Gisella Pere, MD

Abstract Title: A Fast Decompensation

Abstract Information:

Introduction: Nocardiosis is often diagnosed in immunocompromised patients on high dose corticosteroids and TNF-alpha inhibitors. While seen in patients with rheumatologic disorders or transplant patients, it can also be seen in patients with inflammatory bowel disease.¹⁻⁵ Nocardiosis typically presents with either pulmonary, CNS, or cutaneous involvement. When the GI tract is involved it typically is in the form of liver abscesses.⁶ There are no documented cases of decompensated cirrhosis due to *Nocardia* until the present case.

Case Description:

The patient is a 59-year-old male with a past medical history of difficult to control ulcerative colitis treated with methotrexate, adalimumab, and budesonide, and cirrhosis in the setting of primary sclerosing cholangitis who was directly admitted from hepatology clinic for an expedited liver transplant evaluation. Three months prior to admission the patient noticed worsening of his liver function, experiencing ascites, hepatic encephalopathy, and esophageal varices requiring banding. On admission the patient underwent a thorough work up to evaluate causes for his decompensation. The patient denied any infectious symptoms, was afebrile with a WBC count of 6.0 cells/L, negative urinalysis, chest radiograph without focal consolidations, and triple phase CT demonstrated a cirrhotic liver with patent hepatic vasculature. Diagnostic paracentesis had <250 PMNs. Given his negative work up for a cause of decompensation the process for expedited transplant evaluation was started.

Three days later, the ascitic fluid culture grew acid-fast bacilli. This result was not communicated to the team; culture data was found on chart review five days later. Given the presence of non-neutrocytic bacterial ascites, repeat paracentesis was performed. Infectious Disease was consulted given the concern for *Nocardia* or *Actinomyces* infection. A broad work up revealed multiple pulmonary cavitary lesions and a ring-enhancing lesion in the right occipital lobe. He was removed from the transplant list given concern for widely disseminated infection. The ascitic fluid speciated as *Nocardia farcinia* and he was started on appropriate antibiotic therapy. He was monitored with serial paracentesis and imaging for resolution of nocardiosis. He has completed six months of antibiotic therapy with improvement in his liver function. At this point he no longer needs to be considered for liver transplant.

Discussion: This case is the first of its kind to describe nocardiosis in an immunocompromised host as the cause for decompensated PSC cirrhosis. This demonstrates the importance of a wide differential when evaluating a cause for decompensation. Had he not undergone diagnostic paracentesis he would have been transplanted with a likely fatal outcome he would not have undergone imaging to reveal the brain and pulmonary abscesses. This case also highlights the

potential risks of anti-TNF-alpha therapy and high-dose corticosteroids in increasing a patient's risk for nocardiosis.⁷

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Name: Sagarika Sinha, MD

Presentation Type: Poster Presentation

Residency Program: Saint Joseph Hospital

Additional Authors: Alex Steinberg, MD, Michael Morton, MD, FACP

Abstract Title: Call Me, Maybe: Improving Cross Cover Paging

Abstract Information:

Background: The Saint Joseph Hospital Internal Medicine residency teams use two cross cover pagers for after hour paging to the overnight team. Between 7am and 6pm, the primary resident caring for the patient should be paged rather than the cross cover pager. During the day, the on-call team for the day carries these pagers and answers cross cover pages that come to them and triages or redirects these calls to the primary team.

Purpose: The goal of this quality improvement project was to streamline communication and reduce redundant work by decreasing the number of cross cover pages inappropriately sent between 7am and 6pm when the primary day team should be contacted directly.

Methods: Cross cover pager data was analyzed for how often the cross cover pager was paged during the day. A survey was sent to nursing staff to identify areas of trouble regarding pager communication. With this data, we theorized clear terminology and nursing and resident education could reduce redundant work and improve ease of communication. We added a “relationship” in Epic for the primary provider to select called ‘First Call (7 AM- 6 PM)’. Nurses and residents were educated on using this relationship, effective April 2, 2019. New interns joining the internal medicine residency received education on the topic as a part of their orientation. Cross cover pager data was analyzed again after the intervention.

Results: Initial evaluation of the problem revealed in the months of July and August 2017, there were 107 and 118 inappropriate pages, respectively. A nursing survey (34 responses) showed the majority of nurses use the PER in Epic to determine who to call about patient care. They commonly described finding multiple providers listed on the PER with similar titles like “resident”. Nurses said this was confusing and reported an average of 1-2 times a week that the wrong number was paged and had to re-page to contact the appropriate provider for patient care. After the intervention, adding the “First Call (7 AM- 6 PM)” relationship in April, 2019, pager data was reanalyzed July 2019 and August 2019 with 78 and 87 inappropriate pages made each month, respectively. Following our intervention, there was a 25% reduction in inappropriate pages from July 2017 to July 2019 and 26% reduction in inappropriate pages August 2017 to August 2019. As a secondary measure we noted that text pages were repeated less often than numeric pages with 5.46% of numeric pages repeated and 1.26% of text pages repeated when averaging May, July, and August 2019 data.

Conclusion: Based on preliminary data, our intervention of clearly identifying on the PER the primary contact person on the team for a patient reduced to number of pages to the cross-cover pager during the day. Additionally, text pages sent to the cross-cover pager were repeated fewer times than numeric pages indicating quicker response to text pages.

Name: Lena Bazhenova, DO

Presentation Type: Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Eric Elliot, DO

Abstract Title: Slightly Loopy: A Simple Explanation for a Complicated Case

Abstract Information:

Introduction: Encephalopathy is characterized by altered mentation, which may include deficits in cognition, alertness, attention, and consciousness. The frequency with which it is encountered, especially in the elderly population, as well as the broad differential, makes identifying the exact underlying cause and the appropriate treatment a formidable challenge.

Case Description: A 64-year-old male with a complex medical history (including CVA and two intracranial bleeds) presented to the hospital after a fall and was found to have a fracture of the left hip. He was taken to the OR on the same day and had the fracture surgically repaired under general anesthesia. Following surgery, he remained somnolent and unable to answer questions. He was also hypoxic and was requiring 8L via oxy-mask to keep oxygen saturation above 88%. The ICU team was consulted. Patient had a chest CTA which revealed acute pulmonary emboli in the right upper and lower lobes. Lovenox 30mg bid was initiated, and this was changed to Heparin drip once the patient was 48 hours post his surgery. He was also noted to have some fevers shortly following surgery. His UA was positive for nitrites, large leukocyte esterase, WBCs, bacteria, and urine culture was positive for E. coli. He was started on Ceftriaxone. It was believed the patient's encephalopathy was the result of the anesthesia he received during surgery, the hypoxia that occurred following surgery, and the UTI. However, his encephalopathy persisted several days after surgery, long after the anesthesia should have worn off and the hypoxia had resolved. He had also been receiving treatment for his UTI. He had two head CTs, which showed no evidence of new stroke or bleeding. MRI of the brain could not be obtained because patient had a pacemaker. Patient had a 24-hour EEG, which showed nonspecific abnormalities. Lumbar puncture remained a consideration if patient did not improve and no other explanation could be found. At this point, Ceftriaxone was changed to Ciprofloxacin. Patient started to become more alert, and later the same day he was sitting up in the chair and was much more interactive with his wife and the nursing staff.

Conclusion: This case illustrates how many different factors can contribute to encephalopathy and that it can be difficult to identify the cause. Encephalopathy is a known, albeit frequently overlooked, side effect of cephalosporin antibiotics. As an example, a 2013 retrospective study had reported a 15% rate of encephalopathy associated with cephalosporins. In addition to Ceftriaxone, patient also received Cefazolin during surgery. This patient had multiple plausible explanations for his encephalopathy. Therefore, cephalosporin-induced encephalopathy was not the first that came to mind. Recognition of this side effect and prompt discontinuation of the offending drug is important to prevent extensive workup and possible invasive procedures and to decrease hospital length of stay.

Name: Casandra Duarte, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors:

Abstract Title: An Elevated Caused of Headache

Abstract Information:

Introduction:

- 1) Recognize the signs, clinical manifestations, and lab abnormalities associated with Acquired Hemophilia A.
- 2) Develop a systematic approach for the work-up of isolated aPTT elevation.

Case Description: A 73-year-old man presented with retro-orbital headache and confusion. He was normotensive, drowsy, with left hemiparesis and dysarthria. CT showed right basal ganglia hemorrhage with midline shift and intraventricular extension. He denied anticoagulation use or history of coagulopathy. Hb was 8.7, platelets 392,000, PT 12.4 (INR 1.1), elevated PTT at 101 with anti-Xa level at zero. Factor VIII activity was undetectable, with negative lupus anticoagulant and normal Factor XI and IX activity. Factor VIII inhibitor was elevated, consistent with acquired Factor VIII deficiency. While recombinant Factor VIII was obtained, activated Factor VII, prednisone and cyclophosphamide were administered. He remained stable and Factor VIII activity improved to 172% at discharge.

Discussion: Acquired Hemophilia A (AHA) is caused by autoantibodies against Factor VIII. The incidence, 1 per 1.5 million, is bimodal peaking in the 20-30s and 70s [1]. Typical presentation is mucosal/subcutaneous bleeding with no history of coagulopathy [2, 3]. Intracranial hemorrhage (ICH) is uncommon [4-9]. AHA occurs when antigen-presenting cells take up Factor VIII and interact with B cells to produce autoantibodies against Factor VIII. Normally, Factor VIII interacts with thrombin to bind Factor IX, activating thrombin and initiating the intrinsic pathway. Understanding the lab abnormalities is crucial for diagnosis of this rare disease. The first clue is prolonged aPTT that does not correct with mixing studies, indicating the presence of an inhibitor. Factor VIII activity will be undetectable; presence of Factor VIII inhibitor confirms the diagnosis. Treatment is immediate coagulopathy reversal and prolonged immunosuppression to eradicate the inhibitor. For severe bleeding, coagulation factor replacement is required. With high inhibitor titers, exogenous Factor VIII may not be sufficient. Therapy with prothrombin complex concentrate or Factor VII may be required [2]. Porcine Factor VIII may be effective as its structure is unrecognized by autoantibodies [9]. Immunosuppression with steroids and cyclophosphamide is usually successful. 70-90% of patients with AHA present with life-threatening bleeding with a 5-10% fatality rate [1-2]. In case reports of AHA with ICH, 3 of the 6 patients expired [9]. AHA is a rare etiology of ICH requiring quick identification and treatment. The diagnosis and cure in this case hinged on the thoughtful and systematic work-up of an elevated PTT.

Name: Alex Steinberg, MD **Presentation Type:** Poster Presentation

Residency Program: Saint Joseph Hospital

Additional Authors:

Abstract Title: DKA Without the Sugarcoating

Abstract Information:

Introduction: Canagliflozin (brand name Invokana), and the other sodium-glucose cotransporter-2 (SGLT-2) inhibitor used in type 2 diabetes have been increasingly used off label in type 1 diabetes (T1DM)¹. We present a case of euglycemic DKA in a patient with well-controlled T1DM on an insulin pump and Canagliflozin admitted to the ICU. While euglycemic DKA is a known complication of SGLT-2 inhibitors, the profound metabolic derangements presented in this case highlight a severity of disease that could go under appreciated or even unrecognized.

Case Description: A thirty-five-year-old woman with T1DM presented with a week of rapidly increasing fatigue, extreme thirst, nausea, and vomiting. She also described sour breath and frothy urine. Four days prior she had started a low carbohydrate and grain free diet. She had a 10-year history of T1DM, treated with an insulin pump, canagliflozin, liraglutide, and metformin. She denied recent episodes of hyperglycemia. Admission labs showed a metabolic anion gap acidosis with a pH of 6.99, anion gap of 25, glucose of 178, with ketones in the blood and urine. Serum alcohol and salicylates levels were undetectable. She was admitted to the ICU and treated on the DKA protocol with infusions of insulin, dextrose, crystalloid, and bicarbonate as well as potassium supplementation. She had resolution of her acidosis and symptoms in twenty-four hours.

Discussion: Sodium-glucose cotransporter-2 (SGLT-2) inhibitors, including Canagliflozin, first approved in 2013, are some of the newest oral medications used in T2DM. These medications prevent glucose reuptake in the proximal renal tubule leading to glucose loss through the urine. In the Endocrinology literature DKA is a known complication of Canagliflozin in patients with T1DM^{2,3}. This case of euglycemic DKA presents an important learning opportunity to critical care providers, since the metabolic derangements in DKA go beyond the effects of hyperglycemia alone. Indeed, the unbridled ketosis that leads to ketoacidosis is driven by intracellular hypoglycemia due to hypo-insulinemia. This severely limits the available substrate for cellular respiration. As our patient decreased her carbohydrate intake, her insulin pump adjusted, giving her progressively less insulin. With less insulin, less glucose was transported intracellularly and more glucose was lost in the urine, having been blocked by the SGLT-2 inhibitor. This explains her classic symptoms of foaming urine and dehydration without hyperglycemia. Treatment of euglycemic DKA should follow the same protocol for hyperglycemic DKA: replacement of depleted substrates with transfusion of both D10 and insulin.

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Name: Luke Clifford, DO

Presentation Type: Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Chase Bartholomew, DO, Rajiv Singh, DO, Stephanie Murphy, DO

Abstract Title: The “Aneurysm” That Did Not Pop

Abstract Information:

Introduction: Trigeminal neuralgia is a facial pain disorder hallmarked by unilateral recurrent paroxysms of lancinating pain restricted to the somatosensory distribution of the trigeminal nerve. It has a slight predilection for females, its prevalence increases with age, and the annual incidence is estimated to be 4 to 13 per 100,000.

Case: A 58 year-old female with a past medical history of herpes zoster, dental caries, herpes labialis, and bells palsy presented to her primary care provider to establish care with a chief complaint of left-sided neck and facial pain. She endorsed several years of a sensation that she described as “an aneurysm is going to pop”. She felt this along the left side of her neck and face, described as a “lightning/severe spasm” in quality, lasting seconds in duration, 10/10 severity, and daily to weekly episodes. The pain had been previously diagnosed as neck spasms and managed with chiropractic treatments, numerous over the counter pain medications, ice, heat, and stretching exercises, but remained refractory to treatment. Given her lack of improvement, her symptoms were attributed to poor dentition—and she was encouraged to follow up with her dentist. Her pain occurred randomly, although sometimes associated with brushing her teeth, which created a moderate aversion to dental care. She denied any associated conjunctiva injections, headaches, prior facial/neck trauma, skin changes, hearing loss, tinnitus, or rhinorrhea. Her physical exam was unremarkable other than poor dentition. The patient was diagnosed with trigeminal neuralgia given her symptoms and clinical picture, with a less likely differential diagnosis of multiple sclerosis, migraines, cluster headaches, temporomandibular joint dysfunction, giant cell arteritis, dental pain, and postherpetic neuralgia. A facial MRI was obtained looking for sources of her trigeminal neuralgia, which was unremarkable, thus, she was categorized as likely idiopathic trigeminal neuralgia. She was started on carbamazepine 100mg twice daily, and has since largely remained symptom free to date without need for up titration.

Discussion/conclusion: This case demonstrates an unusual chief complaint for a rare etiology of facial pain. With thorough questioning she largely endorsed traditional features of trigeminal neuralgia. Unfortunately, a delay in diagnosis led to prolonged morbidity and possible worsening of dental care, which further highlights the need for good history taking and differential diagnosis. Etiology wise, we speculate reactivation of either varicella zoster virus or herpes simplex virus type 1, but this connection seems to be supported by only a number of case reports. Interestingly, her

prior bells palsy was left-sided and could conceivably be related to the same viruses. Ironically, her work up did not identify any aneurysm "going to pop".

Name: Larry Liriano, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors: Chantal O'Brien, MD

Abstract Title: Cerebrospinal Fluid (CSF) Leak as a Secondary Cause of Chronic Headache

Abstract Information:

Introduction: ICHD-3 classifies CSF leak as headache attributed to low cerebrospinal fluid 7.2. The annual incidence is 5/100,000, with peak at 40 years old, but it can be diagnosed at any age. The most common etiology is a dural defect, most commonly low cervical and/or thoracic. Headache may be described as thunderclap, bilateral in occipital regions and neck, or it could be in any region, typically worsening at the end of the day with photophobia or blurred vision, facial pain, tinnitus, imbalance, and spinal symptoms. It has shown to improve with the Valsalva maneuver in supine position. Multiple risk factors include hyper-mobile joints, head and neck trauma, spinal surgery, or previous lumbar puncture (LP).

Case Description: A 46 years old man with history of spontaneous left pneumothorax came to the clinic with persistent headaches after a cycling accident 4 months before consultation. He hit his head while he had his mountain bike strapped to his shoulder when swung around and struck left side of his head. He denied loss of consciousness with episode and when he arrived home, he noticed low grade headache and dizziness. Symptoms persisted for a week and gradually increased. He went to his primary physician who recommended head CT scan, which was reported unremarkable. Tapering of prednisone and gabapentin were recommended. 3 weeks after the initial incident, patient had improvement of his symptoms and returned to his cycling routine. Unfortunately, headache pattern increased every time he was riding with extreme light sensitivity and sensation of "whooshing" in his ears and feeling that his brain was "sliding down." Head CT was repeated and reported unremarkable. Because of his persistent headaches, brain MRI was done and reported unremarkable with subsequent LP reporting opening pressure of 4 cmH₂O. Because suspected headache attributed to low CSF, blind blood patch was done with headache relieve lasting only one week; no benefit from OTC. Upon physical examination, bilateral retro orbital and occipital headache with daily pattern, worsening in the evening/night time accompanied with bilateral tinnitus. Symptoms improved in supine position. BP 145/80 mmHg, pulse 84, temp 37.1 C, BMI 22.02 kg/m². Neurological examination and laboratories were unremarkable. Spine CT myelogram done reporting: extrathecal collection in thoracic spine T7-T8 suggesting CSF leak with osteophytes C6-7 and spur T9-10. Targeted blood patch was done, and gradual improvement was reported.

Discussion: Magnetic resonance imaging abnormalities may be present in 90% of patients with CSF leak reporting parenchyma enhancement when gadolinium is administered or descending of the cerebral tonsil. CT myelogram or spine MRI has the higher sensitivity when initial brain MRI is unremarkable. Physicians should be aware of secondary headaches like CSF leak, because early diagnosis and treatment will relieve chronic pain in these patients.

Name: Ricki Kumar, DO

Presentation Type: Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Logan Reimer, DO, Hannah Mirzakhani, DO, Ashley Allemon, DO

Abstract Title: Running on Empty: Limiting Platelet Transfusions in Unresponsive ITP

Abstract Information:

Introduction: Immune thrombocytopenic purpura (ITP) is a development of autoantibodies against platelets, causing destruction and a count of typically less than 100,000/ μ L. ITP is one of the most common causes of thrombocytopenia; bleeding is a common cause of mortality in these patients. The risk of bleeding increases if the count falls below 20,000/ μ L, often requiring a transfusion. Here is discussed a patient who, despite repeated platelet transfusions, had a platelet count below 1,000/ μ L. This raised the ethical dilemma of how long to treat a seemingly treatment-unresponsive patient in the setting of limited resources and a shortage of platelets.

Case presentation: W.L., an 89-year-old male with history including prostate cancer presented to the emergency department (ED) for oral blood blisters noticed by his dentist; he also admitted to mild rectal bleeding and mild bruising for two days prior. In the ED, his platelet count was 1,000/ μ L, stool guaiac positive. CT head was negative for intracranial hemorrhage. Exam significant for painful subconjunctival hemorrhage in left eye, and diffuse petechiae on lower extremities. Hematologist recommended treatment with IVIG, steroids, and transfusion of two units of platelets. Platelet count increased to 42,000/ μ L, then began to fall despite treatment and daily transfusion. Bone marrow biopsy showed monoclonal B-cell population without evidence of underlying lymphoproliferative disorder. ITP is rarely associated with CLL. Starting rituximab and romiplostim failed to increase platelet counts. Bacteremia, pleural effusions, and a tricuspid valve vegetation were discovered after patient developed fever and dyspnea. Brain MRI for encephalopathy showed small acute lacunar infarcts. By this time, patient had been transfused 21 units of platelets and 8 units of packed erythrocytes. Platelet transfusions were stopped as counts remained <1,000/ μ L. Ultimately the decision was made to pursue palliative hospice care and the patient died a few days after he was transferred to hospice care.

Discussion: This case has both medical and ethical implications. In the case of ITP unresponsive to IVIG, high dose steroids, Rituximab and Romiplostim, splenectomy is the treatment of choice if platelets remain <20,000/ μ L with signs of bleeding. However splenectomy carried a high risk of bleeding given platelet count remained <1,000/ μ L. There was not any known autoimmune condition, toxic exposure, or signs of viral or bacterial infection prior to admission. The tricuspid valve vegetation could have been due to staphylococcus epidermidis bacteremia, however may also have been from his dental procedure 3 weeks prior to admission. An ethical dilemma surfaces in a patient who is full code with diagnosed ITP requiring several units of platelets in a community hospital with limited resources. In this case, the decision was made to stop platelet transfusions.

Name: Tomio Tran, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado School of Medicine

Additional Authors: William Cornwell, MD

Abstract Title: Flash Pulmonary Edema as a Result of Alcohol Withdrawal during Decompensated Alcoholic Cardiomyopathy

Abstract Information:

Introduction: There is a dearth of information regarding management of patients with concurrent alcoholic cardiomyopathy and alcohol withdrawal. Heart failure and alcohol withdrawal are both states with excess sympathetic nervous system activity. Aggressive management of alcohol withdrawal and its hyperadrenergic state may be important in patients with poor cardiac reserve in alcoholic cardiomyopathy.

Case Description: A 33-year-old man with history of severe alcohol use disorder presented to the emergency department with dyspnea on exertion, orthopnea, and peripheral edema. He had no known history of cardiac disease. His last alcoholic drink was 12 hours prior to admission.

Physical exam showed volume overload, diffuse pulmonary rales, and increase work of breathing. He was found to have 4-chamber dilation of his heart and an ejection fraction of 10-15% on transthoracic echocardiogram. He was initiated on diuresis and placed on a Clinical Institute Withdrawal Assessment (CIWA) for alcohol withdrawal on admission.

As his withdrawal worsened, he had increasing respiratory distress. Computed tomography scan of the chest showed diffuse pulmonary edema. He eventually required intubation and had a prolonged course on the ventilator. He developed a ventilator-associated pneumonia and supraventricular tachycardia that ultimately resulted in pulseless electrical activity and death.

Discussion: Alcohol use-disorder and alcoholic cardiomyopathy are co-morbid conditions, thus patients may present in concurrent alcohol withdrawal and decompensated heart failure. There is a lack of information in the literature about the acute management of these patients. Current alcohol withdrawal protocols use a reactionary approach to treating withdrawal. In patients with alcoholic cardiomyopathy with reduced ejection fraction, it may be important to aggressively treat alcohol withdrawal early in order to reduce central nervous system sympathetic outflow as well as using afterload reducing agents to improve forward blood flow from the heart.

Name: Cole Seifers, DO

Presentation Type: Poster Presentation

Residency Program: Parkview Medical Center

Additional Authors: Ashley Allemon, DO, William Grigg, DO, Andrew Van Boening, DO, Charles Ruzkowski, MD

Abstract Title: A Hepatic Cause of Dyspnea

Abstract Information:

Introduction: Hepatopulmonary syndrome (HPS) can best be characterized by a triad of liver disease, abnormal arterial oxygenation, and evidence of intrapulmonary vascular dilatations. HPS is clinically diagnosed; the patient must have known liver disease with impaired oxygenation and known pulmonary shunting for the diagnosis to be considered. The following case highlights a 61-year-old male with a medical history including chronic obstructive pulmonary disease (COPD) and cirrhosis who was diagnosed with HPS.

Case Description: A 61-year-old male with a medical history including COPD on continuous supplemental oxygen and alcoholic cirrhosis presented to the emergency department with dyspnea and an increased oxygen demand. His dyspnea initially improved after receiving diuretics for pulmonary edema and undergoing a paracentesis for decompensated cirrhosis. He was not eligible for a thoracentesis due to coagulopathy. Despite optimal medical therapy, he required gradually increasing amounts of supplemental oxygen. Incidentally, he had a diagnostic echocardiogram with bubble study as part of an outpatient workup for exertional dyspnea about 12 weeks prior to this admission; this demonstrated a mildly elevated right ventricular systolic pressure and no note of a right to left shunt. During the inpatient echocardiogram with bubble study, the notice of late bubbles prompted a reevaluation of the previous outpatient echocardiogram. The presence of an intrapulmonary shunt led to the diagnosis of HPS. The gastroenterology service was consulted to evaluate the patient for a possible liver transplant; he was not a surgical candidate and proceeded with optimized medical management for his HPS.

Discussion: HPS involves abnormal arterial oxygenation (i.e., increased alveolar-arterial oxygen gradient) caused by intrapulmonary vascular dilatations in the setting of liver disease, portal hypertension, or congenital portosystemic shunts. There are likely several factors involved in the process including portal hypertension leading to the translocation of bacteria, triggering inflammatory cytokines and the release of vasoactive mediators ultimately leading to pulmonary

vasodilation and angiogenesis. The failing liver is not able to effectively metabolize and remove these vasodilators from circulation. This vasodilation leads to pulmonary capillary dilation, causing hypoxemia via ventilation-perfusion mismatch and mixed venous blood into the pulmonary veins and oxygen diffusion limitation. At room air, the partial pressure of oxygen is not sufficient for equilibration due to vascular dilation; this improves with supplemental oxygenation. Some studies have shown mortality of 41% over 2.5 years in patients with HPS. Liver transplant is the only effective treatment; some treatments that have shown some potential benefit include cyclooxygenase (COX) inhibitors, immunosuppressants, methylene blue, pentoxifylline, and transjugular intrahepatic portosystemic shunt (TIPS). Ultimately, this case demonstrated the effects of liver disease and its ability to cause resultant pulmonary side effects.

Name: Colt Williams, MD

Presentation Type: Poster Presentation

Residency Program: University of Colorado Medical School

Additional Authors: Matthew West, MD

Abstract Title: Evaluation of Acute Vertigo – Who Needs Imaging?

Abstract Information:

Introduction: Differentiating central from peripheral sources of acute vestibular syndrome is a challenging, yet critical skill for the physician when examining a patient with vertigo. In the case presented below, we will discuss several key physical exam findings that were undervalued and several that were not performed that not only could have rapidly clarified the patient's diagnosis, but also could also have prevented low value neuroimaging.

Case Description: A woman in her 20s with no past medical history presented to the emergency department after being awoken by the sudden onset of profound vertigo. Immediately upon awakening she noticed severe difficulty walking or standing due to her dizziness. She also reported intractable nausea and vomiting that was worsened with movement. She denied headache, neck pain, difficulty speaking, weakness, confusion, photophobia, or recent illness.

Upon arrival to the hospital she was afebrile and her vitals were notable only for sinus tachycardia of 108 beats/min. She was evaluated by the emergency room physician who noticed rightward jerk nystagmus on primary gaze. Her gait was unsteady and she required support due to the severity of her vertigo. The patient could not tolerate a Dix-Hallpike maneuver due to active vomiting and worsening of her symptoms with movement. Her rightward nystagmus persisted despite change to all directions of gaze and she had difficulty maintaining visual fixation due to her nystagmus. Torsional or vertical nystagmus were not noted. Cranial nerves were intact, finger-nose-finger, knee-heel-shin, and rapid alternating movements were normal. She displayed no focal weakness and no changes to sensation or speech. Her complete blood count, basic chemistry profile, and EKG were normal. A stroke alert was called for concern for posterior circulation infarct. The on-call neurology team evaluated the patient approximately 30 minutes after her arrival to the emergency department. Her National Institutes of Health Stroke Scale (NIHSS) was 0. CTA of the head and neck were normal. The patient was diagnosed with vestibular neuritis and was discharged home four hours after presentation. On follow up one week later, her symptoms had resolved and she was back to baseline functioning.

Discussion: Differentiating central from peripheral sources of acute vestibular syndrome is a challenging, yet critical skill for the physician when examining a patient with vertigo. In this case, there were several key exam findings that were undervalued and several that were not performed that not only could have rapidly clarified the patient's diagnosis, but also could also have prevented low value neuroimaging.

HiNTs, or the Head-Impulse-Nystagmus-Test of Skew, has been shown to reliably differentiate peripheral from central sources of an acute vestibular syndrome. During head impulse testing, the patient's head is rapidly turned 15° by the examiner and visual fixation is monitored.

An inability to fixate with a resultant saccade back to target suggests a peripheral disorder. Unidirectional nystagmus suggests a peripheral disorder. Finally, the lack of vertical skew of one eye after being covered then uncovered further reinforces a peripheral source of vestibular symptoms. A normal horizontal head impulse test, direction changing nystagmus in eccentric gaze, or skew deviation was 100% sensitive and 96% specific for vertebrobasilar stroke in 101 patients and outperformed acute MRI of the brain which was falsely negative in 12% of patients ¹.

Lateral medullary infarction, also known as Wallenberg Syndrome, is the most common syndrome associated with vertebral artery dissection or occlusion. While vertigo and nystagmus are two of its cardinal features, it is also characterized by hypotonia and ataxia of the ipsilateral arm, diplopia, ocular torsion, loss of pain and temperature sensation of the contralateral limb and trunk, and occasionally paralysis of cranial nerves IX-XII—all of which were absent in this patient.

Several validated tools to aid in the diagnosis of stroke do exist, including the Los Angeles Prehospital Stroke Screen (LPSS), Cincinnati Pre-Hospital Stroke Scale, and the Recognition of Stroke in the Emergency Room (ROSIER) Scale. The ROSIER scale evaluates for common stroke mimickers and inquires about specific neurologic deficits such as unilateral weakness and speech disturbance. The scale is scored from -2 to +5 with scores ≤ 0 showing a negative predictive value of 91% for infarct and 88% for TIA² in one study. However, these tools are heavily weighted towards recognizing anterior circulation deficits and are not as accurate for screening for posterior circulation or cerebellar infarcts. The paucity of reliable screening tools for posterior or cerebellar infarcts reinforces the importance of an extensive and targeted physical exam—including HiNTs testing—when acute vertigo is the predominant symptom ³.

Dizziness and vertigo are increasingly common presenting complaints to the emergency department. In 2013 US national costs for the evaluation of vertigo were estimated to exceed \$4 billion per year¹. Given the rising cost of healthcare combined with the emergency department's ease of access to neuroimaging, it is now more important than ever that we as providers be conscientious in our decision in whom to scan.

Had the patient's non-torsional, non-deviating nystagmus and normal neurologic exam been weighed more heavily, and had the clinician utilized the HiNTs test, the patient's correct diagnosis could have been made while avoiding excessive radiation, contrast exposure, and the non-insignificant financial burden of an exhaustive and emergent neurological workup for what was ultimately a benign and self-limiting condition.

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