ACP American College of Physicians
Leading Internal Medicine, Improving Lives
Utah Chapter

2016

Clinical Vignette Competition

UTAH ACP RESIDENTS & FELLOWS COMMITTEE

Kencee Graves, MD – Chair
David Glodowski, MD
Amanda Breviu, MD
Scott Holmstrom, MD

FALL CLINICAL VIGNETTE PROGRAM | WEDNESDAY, SEPTEMBER 28, 2016

University of Utah | Health Sciences Education Building | Alumni Hall

5:30 PM **DINNER & SOCIAL**

5:50 PM **WELCOME & OPENING REMARKS**
Residents & Fellows Committee

JUDGES
Yaw Boateng, MD
Amy Cowan, MD
Erik Riessen, MD

6:00 PM **PRESENTATIONS**

7:15 PM **ANNOUNCE RUNNERS-UP AND 1ST PLACE**

7:30 PM **CLOSING COMMENTS**
Residents & Fellows Committee

UTAH ACP RESIDENTS & FELLOWS COMMITTEE | MISSION STATEMENT

To Improve the professional and personal lives of Utah Residents and Fellows and encourage participation in the American College of Physicians – American Society of Internal Medicine.

1. **Foster Internal Medicine Resident's interest in the ACP – ASIM.**
   - Encourage ACP associate membership and a lifelong interest in ACP – ASIM.
   - Encourage representation on National and Local ACP subcommittees.
2. **Foster educational Opportunities for Internal Medicine Residents.**
   - Encourage participation in local and national ACP – ASLIM Associates Clinical Vignette and Research opportunities.
   - Organize the local competitions. Provide information on board review courses. Publicize local and national educational opportunities. Work with residency programs to improve residency education.
3. **Identify practice management issues for Internal Medicine Residents.**
   - Provide information for residents as they prepare to enter practice, such as practice opportunities and contract negotiation.
4. **Identify public policy concerns of residents.**
   - Monitor local and national health policy and how it relates to Internal Medicine and residency training.
5. **Encourage an interest in community service.**
   - Identify ways associates can become involved with community service in Utah.

CONTENTS

A RARE CAUSE OF CHRONIC DIARRHEA & ABDOMINAL PAIN | JEFFREY BANK, MD [R2]

CASE PRESENTATION: A 68 year-old Caucasian female with a history of IBS, allergic rhinitis, and migraine without aura presented with a 3-year history of episodic abdominal cramping, 3-5 loose, small-volume stools per day, and 30-pound weight loss. She also described episodic atypical chest pain, dyspnea, and recurrent episodes of syncope. Her exam was significant for mild tenderness to palpation of her abdomen and absence of skin lesions. She had an extensive workup including infectious stool studies, anti-TTG Ab, TSH/free T4, and serum immunoglobulins, all of which were unrevealing. CT abdomen, EGD, and colonoscopy did not reveal any mucosal abnormalities or bowel thickening. The endoscopic biopsies did not reveal evidence of eosinophilic esophagitis and the duodenal biopsies showed minimal intraepithelial lymphocytosis. Due to recurrent episodes of syncope along with chronic diarrhea, we obtained a tryptase level which was elevated at 18.2 ug/L (normal < 10.9 ug/L) and plasma histamine level which was elevated at 10 (normal < 8 nmol/L). C-kit point mutation was negative. She was subsequently given a diagnosis of mast cell activation disorder (MCAD). She had significant improvement in her symptoms after initiation of therapy with antihistamines, mast cell stabilizers and a leukotriene inhibitor.

DISCUSSION: Mast cells are known to have immunoregulatory function at the gastrointestinal mucosal interface. MCADs are characterized by accumulation of pathological mast cells in various organs and aberrant release of mast cell mediators. The most common cutaneous manifestation in patients with mastocytosis is urticarial pigmentosa (UP), which our patient did not have. Diagnosis of MCAD remains a challenge as it affects more than one organ system with waxing and waning symptoms. Gastrointestinal manifestations in patients with MCAD include abdominal pain, diarrhea, esophageal reflux, nausea, and vomiting. Our patient's recurrent syncope, weight loss, dyspnea, and migraines could all be explained by mast cell activation process. If a mast cell disorder is suspected, total tryptase levels are checked because mature tryptase is stored inside the secretory granules of mast cells and is released upon activation of mast cells. C-kit point mutations (most commonly D816V) and aberrant CD25 expression help differentiate monoclonal mast cell activation from mast cell activation disorder. Internists and gastroenterologists must consider mast cell disorders in their differential diagnosis of chronic diarrhea, especially when patients have symptoms involving more than one organ system.

END-ORGAN DAMAGE? THE PRESSURE'S ON! | MICHELLE BISCHOFF, MD [R2]

IDENTIFICATION: 61 year old man with history of anxiety

CHIEF COMPLAINT: nausea, vomiting, and headache

HISTORY: Headache was constant, sudden-onset, and associated with nausea, vomiting, and diaphoresis. A syncopal episode was witnessed by staff. He denies chest pain, dyspnea, abdominal pain, dysuria. He has never experienced an episode of these symptoms before. Patient reports recent elevated blood pressure reading at his psychiatrist's office.

PHYSICAL ABNORMALITIES: Blood pressure was 216/147 and heart rate was 131. Exam notable for anisocoria, tachycardia with regular rhythm, and 2/5 systolic murmur heard over LLSB.

LAB RESULTS: Troponin 10.73, TSH and free T4 normal, Utox negative; CT head unremarkable; CT Abd/Pelvis with 5.9 cm mass in right adrenal gland.

DIFFERENTIAL DIAGNOSIS: includes essential hypertension, stimulant intoxication, pheochromocytoma, adrenocortical tumor, Cushing's syndrome, hyperthyroidism, hyperaldosteronism, carcinoid

CASE PRESENTATION: 61 year old man with possible undiagnosed hypertension who presents with severe sudden-onset headache associated with nausea, vomiting and diaphoresis. In the ED had systolic pressures in the 200s with evidence of end organ damage: AKI, increased LFTs, and troponinemia to 10.7. Chest x-ray did not show evidence of a widened mediastinum and CT Brain without evidence of stroke. Abdominal CT showed 6 cm heterogeneous adrenal mass and no sign of renal artery stenosis. Hypertension initially managed with labetalol, clonidine, phenoxybenzamine, nicardipine gtt. Subsequent labs include AM cortisol 23.3 (high) after low dose suppression, but AM cortisol 7.9 (low) after high dose suppression, aldosterone/renin ratio normal, 5-hydroxyindoleacetic acid normal, plasma metanephrines 40.82 (normal <0.49), plasma normetanephrine >50 (normal <0.89), urine fractionated metanephrines 13700 (elevated), normetanephrines 11900, 27000s (elevated).

DISCUSSION: Pheochromocytoma is a rare but important cause of hypertensive emergency as alpha and beta blockers can acutely stabilize a patient and surgery is often curative. It is important to determine if other family members have had symptoms that could be consistent with pheochromocytoma, as familial pheochromocytoma indicates likelihood of MEN2 or von Hippel-Lindau disease. If a genetic disorder is present, screening for malignancies and yearly monitoring for recurrent pheochromocytoma should be performed.

CONCLUSION: Once pheochromocytoma was confirmed with labs, the patient was discharged on propranolol and phenoxybenzamine with plans for outpatient surgery in two weeks. He initially had significant hemodynamic instability on the operating table, but once the right adrenal gland was removed, his hemodynamic parameters stabilized. Since surgery, he no longer requires antihypertensive medications. Surgical pathology showed tumor cells positive for chromogranin, S-100, and GATA-3, consistent with pheochromocytoma.

MORE THAN NO FEELING | AARON CROSBY, MD [R3]

CASE HISTORY: A 30 year old woman presented to an emergency department for evaluation of numbness in her lower extremities and difficulty rising from a seated position. Her symptoms began three weeks prior and progressed to the point of being unable to walk. She also endorsed paresthesia from the nipples down, along with nausea and urinary retention. She had a history of hypothyroidism, obesity and a gastric sleeve procedure two years prior. This was converted to Roux-En-Y gastric bypass ten weeks prior to admission, after which she lost 53 pounds. Outpatient medications included levothyroxine and nutritional supplements.

PHYSICAL ABNORMALITIES: Vital signs: normal. She appeared well. Cranial nerves: normal. Sensory exam: decreased sensation and proprioception below the T4 dermatome. Motor/reflex exam: normal upper extremity strength and reflexes, with 2/5 strength and absent reflexes in the lower extremities. She was unable to stand.

DIFFERENTIAL DIAGNOSIS: Polyneuropathy secondary to inflammatory demyelination (Guillain-Barre) vs. thiamine, copper or B12 deficiency vs diabetes. Rhabdomyolysis. Myelopathy. Hypothyroidism. Spinal cord compression. Neurosyphillis. Heavy metal toxicity.

TEST RESULTS: CBC, BMP, CK, heavy metals, RPR, ANA, B12 and hemoglobin A1c were unremarkable. TSH was 14 uIU/mL with free T4 0.9 ng/dL. MRI of brain and spine revealed mild disc herniation. LP was normal with normal oligoclonal bands and myelin basic protein. EMG with nerve conduction studies was consistent with motor predominant axonal neuropathy. Thiamine was low at 39 nmol/L (normal 70 - 180), and serum copper was borderline low at 80 ug/dL (normal 80 - 155).

FINAL DIAGNOSIS: Beriberi secondary to post-gastric bypass thiamine and copper deficiency

DISCUSSION: Neurologic complications occur in 5% of bariatric surgery patients. Neuropathy can be secondary to compression from loss of subcutaneous fat, inflammatory demyelinating neuropathy, and axonal neuropathies. Thiamine deficiency presents as early as six weeks post-operatively, and can manifest as encephalopathy (Wernicke's) and/or motor predominant polyneuropathy (beriberi). Copper deficiency also presents as polyneuropathy. EMG with nerve conduction studies differentiates acute inflammatory demyelinating polyneuropathy from the axonal neuropathy of nutritional deficiency. Patients with symptomatic thiamine deficiency should receive parenteral thiamine. After a course of parenteral thiamine, copper and an inpatient rehab stay, the patient discharged home. Her sensory symptoms resolved, and she was ambulating with a gait aid.

IDENTIFICATION: Patient is a previously healthy 41 year-old female who presents with fatigue and headache.

CHIEF COMPLAINT: Headache

HISTORY: Patient is a previously healthy 41 year-old female who presents with malaise, fever, and severe headache for 60 hours. She recently returned from a trip to Bali and reports onset of these symptoms upon traveling home. Her headache is retro-orbital and associated with photosensitivity. Other symptoms include diffuse myalgias and arthralgias. Her immunizations were up-to-date, except for malaria treatment. Her teenage son, who traveled with her, also experienced similar symptoms which resolved prior to their return home. She does endorse history of multiple mosquito bites.

PHYSICAL ABNORMALITIES: Exam was notable for fatigue and photosensitivity. Macular rash on upper chest and back was noted, along with petechiae on buttocks.

LAB RESULTS: Initial laboratory results were remarkable for a white blood cell count of 1,860 with an ANC of 1400/uL. Other cell lines were also low. Cell counts decreased to a nadir of WBC 990, ANC of 419, and Platelets of 55,000. CMP, including liver function tests, was largely within normal limits. A lumbar puncture was performed and CSF studies were unremarkable. Peripheral smear for malaria was negative.

DIFFERENTIAL DIAGNOSIS: The presentation of fever, headache, and neutropenia in the setting of recent travel was suggestive of acute viral infection. The differential diagnosis considered includes: Dengue fever, Chikungunya, Zika Virus, Malaria, typhoid fever, West Nile, rickettsial infections, and leptospirosis.

CASE PRESENTATION: Acute onset fever, retro-orbital headache, and neutropenia in a previously healthy female who recently traveled to Indonesia. Testing returned positive for Dengue, Type 1 and diagnosis was consistent with Classical Dengue Fever.

DISCUSSION: Dengue fever was clinically suspected with retro-orbital headache, fever, and myalgias presenting 3-14 days after travel in an endemic region. Although Dengue virus is only one possible diagnosis when considering post-travel systemic febrile illness, it has been found to be the second-most common diagnosis in this differential. Other illnesses that are potentially treatable, such as malaria, must be ruled out when laboratory testing is available.

Conclusion Infection will provide long-term protection against this particular Dengue serotype; however, it offers little protection against the other 3 serotypes of Dengue virus. The risk of severe disease (such as hemorrhagic fever or shock) is significantly higher during secondary dengue virus infection. Thus, the patient was advised to utilize extensive anti-mosquito strategies if she were to travel to an endemic area.

CASE PRESENTATION: A previously healthy 72-year-old male was evaluated for a 9-month history of progressive anemia and thrombocytopenia (9.6 and 76 respectively), fevers, and weakness. Previous diagnostic workup was significant for normal iron and folate studies, a mild B12 deficiency, and a bone marrow biopsy showing mild atypical changes w/o objective diagnostic abnormalities. Infectious studies were negative and his cytopenias did not improve with B12 supplementation.

Due to progressive symptoms, the patient was admitted in June. Laboratory studies showed an elevated LDH(1084) and low Haptoglobin(<10) concerning for hemolysis and his platelets continued to be low. Steroids were initiated resulting in the resolution of his fevers with no improvement in his platelet count. Therefore, the patient was initiated on Rituxan, NPlate, and IVIG, again without improvement of his platelet count. Positron emission tomography revealed focal uptake in the cecum and diffuse mild splenic uptake with mild splenomegaly. Colonoscopy revealed no significant findings.

The patient was again admitted in August for continued symptoms. Due to his fevers, splenomegaly, elevated ferritin (825), and cytopenias, a diagnosis of hemophagocytic lymphohistiocytosis was considered. Soluble IL-2 receptor was found to be markedly elevated (12060). Due to his refractory thrombocytopenia, HLH, and hypermetabolic spleen on PET, the patient underwent splenectomy. Pathology revealed hemophagocytosis as well as marginal zone lymphoma with transformation to Diffuse Large B-Cell Lymphoma.

PHYSICAL ABNORMALITIES: Exam was notable for splenomegaly and intermittent and diffuse purpura.

DIFFERENTIAL DIAGNOSIS: The patient's anemia and thrombocytopenia were initially thought to be due to nutritional deficits. Given the negative initial work up, immune thrombocytopenic purpura (ITP) was initially felt to be the cause of the patient's thrombocytopenia. Later studies, suggested hemolysis as a cause of his anemia, however the specific etiology was unknown. In the end, HLH secondary to suspected transformed indolent lymphoma was the cause.

TREATMENT: The patient was initiated on R-CHOP + Etoposide for treatment of DLBCL and HLH. Blood counts have improved with treatment.

CONCLUSION: HLH is a syndrome that is characterized by inflammation and tissue destruction due to excessive immune activation, which can present as a single episode or relapsing disease. HLH is often fatal without treatment, which includes identification and treatment of the underlying cause, or primary treatment with etoposide and dexamethasone. Common etiologies of HLH include genetic mutations (usually presenting in childhood), infection, malignancy (usually lymphoma), and rheumatologic disorders.

DON'T FORGET THE SALT | SARAH GILLIGAN, MD [R2]

IDENTIFICATION: 80 year old male

CHIEF COMPLAINT: Weakness, diarrhea

HISTORY: The patient is an 80 year old male with COPD, BPH, OSA, hypogonadism, and chronic back pain on opiates who presented with 5 days of weakness and diarrhea. For 4-5 months, he had decreased appetite, weight loss, and worsening memory. Work-up by his primary care provider had been unremarkable as to a reversible cause of his memory loss, so with a family history of Alzheimer's, that was presumed to be his diagnosis. In the 5 days prior to admission, he had increased generalized weakness that had progressed so that he had been unable to get out of bed. This was accompanied by worsening anorexia, non-black, non-bloody diarrhea, and difficulty urinating without other symptoms of pain, nausea, vomiting, fevers, or chills. He denied any recent medication changes, travel, or sick contacts.

PHYSICAL ABNORMALITIES: Physical exam was remarkable for bradycardia (rate 56) and cool extremities, though with 2+ peripheral pulses throughout. He was oriented to person and place but not time with the remainder of his neuro exam unremarkable.

LAB RESULTS: Labs showed mild normocytic anemia and thrombocytopenia, both within baseline for the patient, sodium of 120 (previously WNL), chloride of 91, and creatinine of 1.25 (within baseline for patient). Troponin was negative. Serum osmolality 252, urine osmolality of 338 and urine sodium of 98 (urine studies performed after saline hydration). EKG showed first degree AV block and chest and abdominal CT showed no acute processes.

DIFFERENTIAL DIAGNOSIS: The patient was initially felt to have progression of his dementia with possible super-imposed viral gastroenteritis. His hyponatremia was thought to be hypovolemic due to poor oral intake and diarrhea vs. SIADH secondary chronic back pain.

DISCUSSION: Review of the patient's medical records revealed a head CT performed 5 months prior to admission showing a pituitary macroadenoma as well as testosterone levels that were undetectable. Work-up for his dementia had included vitamin levels and TSH, but a free T4 had not been performed. Additional labs revealed cortisol level of < 0.8 and free T4 of 0.57 with TSH of 1.44. MRI of the brain demonstrated persistence of a 1.8 cm pituitary macroadenoma with resulting panhypopituitarism and myxedema coma based on lab studies and exam. The patient was started on prednisone and levothyroxine with normalization of his sodium levels and improvement in his energy and cognition.

GREEN APPLES IN THE HEART, LIVER, AND KIDNEYS | ANDREW HAHN, MD [R2]

FINALIST

CHIEF COMPLAINT: Mr. M is a 75 yo M with CKD stage III, OSA, and HLD who presented with abdominal pain and jaundice.

HISTORY: 5 weeks prior to admission, Mr. M developed fatigue, epigastric abdominal pain, nausea, and anorexia. He was seen by his primary care provider and was diagnosed with PUD secondary to H. pylori infection. He was started on augmentin, flagyl, and omeprazole. Upon beginning treatment, he became jaundiced with worsening epigastric pain and was transferred to the Huntsman.

PHYSICAL ABNORMALITIES: HEENT: scleral jaundice present | Abd: protuberant abdomen with ascites present. Non-tender | Ext: 2+ pitting edema of BLE

LAB RESULTS: Na- 131, K- 4.7, Cl-105, HCO3- 21, BUN- 45, creat- 2.68 | TProtein- 6.3, albumin- 1.9, TBili- 8.9, AST- 159, ALT- 83, alk phos- 1899 | UA: protein- 100

DIFFERENTIAL DIAGNOSIS: Cirrhosis, HF, nephrotic syndrome, primary liver disease

CASE PRESENTATION: After his initial presentation, a diagnostic paracentesis showed a SAAG > 1.1. Infectious hepatitis panel (HAV, HBV, HCV, VZV, HSV, CMV), ERCP, and MRCP showed no evidence of obstruction. TTE demonstrated an EF of 67% with mid-cavitary obstruction. Urine studies were notable for spot protein to creatinine ratio of 2.1 g/g. UPEP was positive for monoclonal free lambda light chains consistent with Bence Jones proteins. SPEP/IFE had an M-spike in the gamma region that accounted for 2.1 g/dL of the total 2.35 g/dL. Renal then performed a biopsy that showed amyloidosis, AL lamba-type (positive Congo-red deposits) with cardiac, renal, and hepatic involvement. He was started on dexamethasone and bortezomib with rapid improvement in his LFTs.

DISCUSSION: New onset anasarca generates a broad differential that includes nephrotic range proteinuria. While there are numerous etiologies of nephrotic range proteinuria, amyloidosis is a rare cause that internists should be cognizant of. Early diagnosis of amyloidosis can significantly improve outcomes. If the disease progresses to cardiac involvement, amyloidosis carries a grim prognosis. Diagnosis with only renal involvement allows for prompt treatment to prevent further fibril deposition and more end-organ dysfunction. This case demonstrates the classic presentation for renal, cardiac, and hepatic involvement from amyloidosis.

CONCLUSION: Amyloidosis is a rare disease that can have renal, hepatic, cardiac, or neurologic involvement. Early diagnosis of amyloidosis can lead to improved clinical outcomes by preventing further end-organ dysfunction. Thus, internists and hospitalists should be sure to keep amyloidosis on their differential for proteinuria.

A PERNICIOUS CAUSE OF HEMOLYSIS | STEPHEN JENKINS, MD [R3]

HISTORY: A 64-year-old woman with hypothyroidism presented to the hospital with one month of fatigue and exertional dyspnea. She also reported dark urine, swelling in her legs, numbness in her feet and yellowing of her skin. She had not seen her primary care physician for several years. She had received intravenous iron in the past for iron deficiency anemia. She denied any hematemesis, hematochezia or melena.

PHYSICAL EXAM: She was an obese, disheveled woman with scleral icterus and sublingual jaundice. Cardiac exam revealed tachycardia and a II/VI systolic murmur. She had 3+ pitting edema up to the knees bilaterally. She had a normal neurologic exam except for a mildly ataxic gait and diminished vibratory sensation in the lower extremities.

LABORATORY: Her hemoglobin was 5.4 g/dL, with a mean corpuscular volume of 130 fL. She had a total bilirubin of 4.2 mg/dL, with direct bilirubin of 0.6 mg/dL. Lactate dehydrogenase was 1713 U/L, haptoglobin was undetectable, and her reticulocyte index was 0.2%. Coombs test was negative. Serum folate was > 24, but serum cobalamin was < 50. Iron studies were normal. Serum methylmalonic acid was 9.86 umol/L and homocysteine was 68 umol/L. Peripheral smear showed occasional schistocytes, megaloblastic hypersegmented neutrophils and anisopoikilocytosis.

DIFFERENTIAL DIAGNOSIS: The differential for hemolytic anemia includes congenital hemoglobinopathies and acquired hemolytic anemias. A negative Coombs test made an autoimmune hemolytic anemia unlikely. The presence of schistocytes on the peripheral smear was concerning for a microangiopathic hemolytic anemia, such as thrombotic thrombocytopenic purpura. However, this patient had normal platelet and renal function. She did have profound cobalamin deficiency, as evidenced by an undetectable serum cobalamin level and elevated serum methyl-malonic acid. The most common cause of cobalamin deficiency is auto-antibodies against gastric parietal cells or achlorhydria causing malabsorption.

DISCUSSION: This patient had megaloblastic anemia, evidence of hemolysis, and cobalamin deficiency. The most parsimonious diagnosis was pseudothrombotic microangiopathy with intramedullary hemolysis, which can occur with severe cobalamin deficiency. This patient had positive intrinsic factor antibodies and parietal cell antibodies, consistent with pernicious anemia.

CONCLUSION: This patient received two units of packed red blood cells and was started on intramuscular cobalamin. At her follow-up visit, her hemoglobin, serum cobalamin, LDH, haptoglobin and bilirubin were normal. Her fatigue and dyspnea had improved, but she continued to have problems with ataxia and loss of vibratory sensation.

IT'S ALL ABOUT TIMING: AN ULCERATING RASH IN AN HIV-1 INFECTED MALE | RUSSELL JOHNSON, MD [R4]

HISTORY: A 41-year-old man with a recent diagnosis of HIV presented to clinic with ulcerating rash on the left ankle, right foot, and lateral chest. He had been evaluated by dermatology one month prior for an annular, hyperpigmented skin rash that was unresponsive to topical steroids and antifungals. The lesions became ulcerative and productive of sero-sanguinous drainage.

ROS: positive for intermittent bright red blood per rectum.

PHYSICAL EXAM ABNORMALITIES: 15cm ulcerated lesion on dorsal aspect of right foot with foul-smelling sero-sanguinous discharge. Similar lesions were present on left chest wall (25cm) and left ankle (8cm). No genital, anal, or mucosal lesions.

LAB RESULTS: Complete blood count revealed hemoglobin of 12.5 g/dL, MCV of 70.9 fL, and serum iron of 23 ug/dL. CD4 count was 629 cells/uL and HIV-1 viral load of 73,000 copies/mL.

DIFFERENTIAL DIAGNOSIS: Pyoderma grangrenosum, secondary syphilis, Mycobacterium ulcerans, pyoderma vegetans, disseminated leishmaniasis.

CASE PRESENTATION: He was admitted to the hospital and biopsies were obtained. Tissue cultures and smears were negative for acid-fast bacilli, fungi, and spirochetes. The biopsy showed lichenoid interface changes, numerous plasma cells, and endothelial cell hyperplasia consistent with secondary syphilis, specifically lues maligna. Rapid plasma regain (RPR) titer was 1:1024 and fluorescent treponemal antibody absorption test was positive.

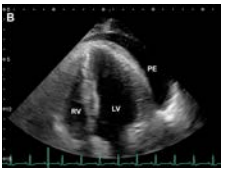
TREATMENT COURSE: The patient was given 2.4 million units of benzathine penicillin intramuscularly. Within twenty-four hours the patient's lesions showed improvement with cessation of drainage. By hospital day three the lesions showed near-complete resolution and patient was discharged home. He was given two more weekly benzathine penicillin injections for the treatment of lues maligna. At six month follow up his lesions had resolved.

DISCUSSION: Lues maligna was first described in the 1800's and represents a rare but severe form of secondary syphilis, now most commonly associated with recent HIV infection. The lesions of lues maligna have an ulceronodular appearance, unlike the classic mucocutaneous eruption of secondary syphilis. Lues maligna was diagnosed based on serology, characteristic pathology, and dramatic response to penicillin. He met three of four diagnostic criteria for lues maligna, only lacking in a severe Jarisch-Herxheimer reaction with penicillin treatment. Often the underlying clue to diagnosis of lues maligna is a recent or concomitant diagnosis of HIV, though the precise pathologic mechanism is not known.

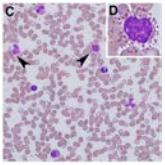
CONCLUSION: Multiple ulcerative cutaneous lesions in a patient with recent HIV infection or risk factors for HIV should always prompt investigation for lues maligna as well as HIV infection.

FINALIST

CASE PRESENTATION: A 50-year-old woman presented with chest pain, dyspnea at rest, associated fever, chills, night sweats and left periorbital swelling (Figure 1A). She had a past medical history significant for adult-onset asthma, hay fever, sinusitis, and 9 surgeries for recurrent nasal polyps. On presentation, transthoracic echocardiogram revealed a significant pericardial effusion with tamponade physiology (Figure 1B). Urgent pericardiocentesis was performed and 800ml of straw-colored fluid was drained. She recovered well with no residual pericardial effusion after removal of pericardial drain and the patient was subsequently discharged.



Ten days later, the patient presented with a syncopal episode. On admission, echocardiogram and chest x-ray showed a recurrent pericardial effusion, and a large pleural effusion. Complete blood count was significant for 45% eosinophilia (Figure 1C, D). Thoracentesis was performed and one liter of fluid was removed, which showed 77% eosinophils. After this procedure, she had residual chest pain, dyspnea, cough, generalized aches and chills. The patient was referred for video-assisted thoracoscopic surgery (VATS) with pericardial window for drainage of pericardial and pleural effusions, and a lung and bone marrow biopsy for further diagnosis and treatment guidance.



PHYSICAL EXAM: Exam revealed distant heart sounds, and absent breath sounds with dullness to percussion below midback bilaterally. Skin demonstrated dermatographia.

LAB RESULTS: Initial WBC was 12.8% with 30% eosinophils. Peripheral blood smear showed significant eosinophilia (Figure 1C, D). CRP was 21, ESR 81, RF 104. T. cruzi, Strongyloides, ANA, anti-MPO, and ANCA antibodies were negative. Cytology, flow cytometry, and bone marrow aspirate showed no evidence of a hematolymphoid malignancy.

DIFFERENTIAL DIAGNOSIS: Presumed diagnosis was Eosinophilic Granulomatosis with Polyangiitis (EGPA, or Churg-Strauss), based on history of nasal polyps, adult-onset asthma, allergic rhinitis, eosinophilia, and chest pain. Patient met 3/6 ACR criteria for EGPA on admission (4/6 criteria is 99.7% specific)[1]. Other DDx included Chagas disease (periorbital swelling, Mexico travel), Löeffler's syndrome (pleural effusions), leukemia, Strongyloidiasis or other parasitic infections.

TREATMENT: Patient was started on high-dose prednisone after bone marrow biopsy. Within 1d of steroid initiation, eosinophilia resolved and the patient showed significant clinical improvement. Cyclophosphamide was added before discharge to help prevent symptom recurrence.

CONCLUSION: EGPA is a rare, necrotizing systemic vasculitis associated with asthma and hypereosinophilia[1,2]. ANCA positive EGPA frequently presents with peripheral neuropathy and renal manifestations, whereas ANCA negative EGPA often includes cardiac symptoms, perhaps suggesting 2 distinct disease etiologies[3,4]. Cardiac tamponade and periorbital swelling are two unusual presentations of this disease, according to the literature[3,5].

(1) Masi, A. T. et al. The American College of Rheumatology 1990 criteria for the classification of Churg-Strauss syndrome (allergic granulomatosis and angiitis). Arthritis Rheum. 33, 1094–1100 (1990). (2) Greco, A. et al. Churg-Strauss syndrome. Autoimmun Rev 14, 341–348 (2015). (3) Yano, T. et al. Cardiac tamponade leading to the diagnosis of eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome): a case report and review of the literature. Heart Vessels 30, 841–844 (2015). (4) Sablé-Fourtassou, R. et al. Antineutrophil cytoplasmic antibodies and the Churg-Strauss syndrome. Ann. Intern. Med. 143, 632–638 (2005). (5) Ameli, F., Phang, K. S. & Masir, N. Churg-Strauss syndrome presenting with conjunctival and eyelid masses: a case report. Med. J. Malaysia 66, 517–519 (2011).

I CAN'T GO TO PROM LIKE THIS | CAROLINE LAGGIS, MD [R1]

CHIEF COMPLAINT: Mouth ulcers

HISTORY OF PRESENT ILLNESS: Patient is an 18-year-old female with history of Systemic Lupus Erythematous who presented to the inpatient service from dermatology clinic for worsening oral mucosal lesions. She first noticed these lesions 3 days prior and they had become increasingly painful to the point that she could no longer eat or drink. Patient also noted subjective fevers, chills, night sweats, and a splotchy rash within the same time period.

Patient's SLE was characterized by numerous rashes of various morphologies recalcitrant to immunosuppressive treatment. Her regimen included methotrexate, hydroxychloroquine, mycophenolate mofetil, dapsone, pulse IV solumedrol, and oral prednisone. Of note, she was admitted 6 weeks prior for inpatient infusion of rituximab. On further ROS, patient reported 7-8 episodes of diarrhea daily since the time of infusion.

PHYSICAL ABNORMALITIES: Vital signs were all stable and within normal limits. Her HEENT exam was notable for several round shallow ulcers scattered throughout the oral cavity that were white in color and several centimeters in diameter. She had associated bilateral cervical lymphadenopathy. Dermatological exam revealed mottled, violaceous macules over her abdomen and lower extremities consistent with post-inflammatory hyperpigmentation. On abdominal exam, she had diffuse mild tenderness to deep palpation.

DIFFERENTIAL DIAGNOSIS: The initial impression of the primary and rheumatology teams was that this presentation likely represented a lupus flare. However, with her immunosuppressed state, infectious etiologies and hematological malignancy were also considered.

LABS: Basic labs were notable for lack of significant abnormalities. SPEP was without evidence of monoclonal banding. Comprehensive immunological and infectious disease labs were sent. Most interestingly, her CMV IgM and IgG antibodies returned elevated. Given her ulcers and diarrhea, the primary concern became active CMV infection.

DISCUSSION AND CONCLUSION: Knowing that elevated CMV antibodies do not imply active infection, the GI team was consulted for tissue diagnosis. Colonoscopy revealed a multitude of small round ulcers throughout the colon highly suspicious for CMV colitis.

This case highlights the importance of considering active infection when patients on immunosuppressive therapy for autoimmune conditions present with a worsening clinical picture. Because of potential similarity or vague nature of presentation, it is an easy mistake to assume new symptoms represent a flare of an underlying condition or a side effect of medication. Immunosuppressive therapy was gradually scaled back during this patient's hospitalization and she was referred to Infectious Disease for further evaluation and treatment.

PLATYPNEA-ORTHODEOXIA SYNDROME IN A LUNG TRANSPLANT PATIENT | M. SYDNEY LEGUYADER, MD [R1]

FINALIST

INTRODUCTION: Platypnea- orthodeoxia syndrome is an uncommon clinical syndrome defined by dyspnea and hypoxia in the erect position and improvement of dyspnea and hypoxia when supine. The most common etiology is an underlying intracardiac shunt such as a patent foramen ovale or atrial septal defect. This is a case of platypnea-orthodeoxia in a patient with delayed lung allograft dysfunction manifesting as bronchiolitis obliterans syndrome without evidence of intracardiac shunting.

CASE PRESENTATION: A 34 year old woman with a history of cystic fibrosis, status post bilateral lung transplant 11 years prior, presented to the pulmonary clinic with progressive shortness of breath. She reported a non-productive cough and dyspnea with minimal exertion for eight weeks. She did not require supplemental oxygen at baseline, and was admitted to the MICU after developing a 10L oxygen requirement.

Bedside evaluation revealed a resting oxygen requirement of 10L high flow nasal cannula to maintain SpO2 93% when erect and SpO2 98% on ambient air when supine. She reported feeling profoundly dyspneic when erect with resolution of symptoms when supine. FEV1 was significantly decreased to 20-30% from a baseline of 88%.

CT chest revealed extensive bronchiomalacia with large volume air trapping on the expiratory phase in the bilateral lower lobes. Bronchoscopy revealed no airways amenable to stenting, BAL analysis did not reveal an infectious process, and transbronchial biopsy was non-diagnostic. Transthoracic echocardiogram with bubble study was negative for right to left inter-atrial or intrapulmonary shunting.

Due to concern for pseudomonas pneumonia, initial treatment consisted of two weeks of IV cefepime followed by two weeks of IV meropenem without improvement in symptoms or FEV1. This patient's bronchiomalacia and air-trapping were attributed to delayed allograft dysfunction from bronicholitis obliterans syndrome. Her immunosuppressive treatment (tacromilus, mycophenolate) was augmented with a three day course of 1g IV methylprednisolone daily. Her dyspnea gradually improved and oxygen was successfully weaned.

DISCUSSION: This case illustrates a pulmonary etiology for platypnea-orthodeoxia not previously described: a ventilation/perfusion mismatch due to significant bibasilar air trapping from bronchiomalacia secondary to bronchiolitis obliterans syndrome. This resulted in shunt physiology in the lung bases. Shunting was most pronounced when the patient was sitting erect, resulting in increased perfusion of the non-ventilated lung bases and severe dyspnea and hypoxia. This is the first reported case of platypnea-orthodeoxia syndrome due to bronchiolitis obliterans syndrome without an underlying intracardiac shunt or pulmonary arteriovenous malformation.

THE WAY TO A MAN'S HEART IS THROUGH HIS PANCREAS | DAWN MILLER [MS4]

FINALIST

CASE PRESENTATION: A 52-year-old man with a history of hypertension, gastroesophageal reflux disease, migraines, and schizoaffective disorder presented with vomiting, lower abdominal pain migrating to his back, intermittent substernal chest pain, and tachycardia.

PHYSICAL EXAM ABNORMALITIES: The patient had an unremarkable exam aside from tachycardia and mild lower abdominal tenderness. Blood pressures were equal in both upper extremities.

Four serial electrocardiograms showed ST elevations in inferior and lateral leads which improved over several hours. Serial troponin levels were normal. The chest x-ray showed no acute processes. D-dimer was elevated, but he did not have pleuritic chest pain or hypoxia. His electrolytes were consistent with pre-renal azotemia. He had leukocytosis with a left shift. His lipase levels were within normal limits.

The patient underwent coronary catheterization, which showed largely patent coronary arteries (20-30% stenosis in the left anterior descending artery, and 40% stenosis in the right coronary artery). A transesophageal echocardiogram showed a small pericardial effusion over the posterior left ventricle.

A CT scan of the abdomen revealed a gallstone in the neck of the gallbladder without distension, diffuse pancreatic edema, and peripancreatic mesenteric fat stranding consistent with pancreatitis.

DIFFERENTIAL DIAGNOSIS: Taken together, it was thought that the patient had lipase-negative pancreatitis with concurrent pericarditis presenting with transient ST elevations.

TREATMENT: He was treated with Dobhoff tube feedings and an eventual cholecystectomy, which resulted in full resolution of signs and symptoms.

DISCUSSION: This case demonstrates the diagnostic challenges of a case of pericarditis associated with pancreatitis, and serves as a reminder of non-coronary reasons for ST elevations.

It is known that pericarditis can cause transient ST elevations[1]. Although pericarditis is often caused by viral infections, there is evidence that pericarditis can also develop in the setting of pancreatitis. The suggested mechanism is the release of pancreatic enzymes leading to fat necrosis followed by serositis[2,3].

There is also evidence showing physiologic changes with pancreatitis alone can cause ST elevations. Circulating pancreatic enzymes are thought to cause transient hyperkalemia, resulting in ST changes. Furthermore, the rami supplying the gallbladder and the heart are connected via intermediate neurons, which allows for a vagally-mediated reflex in the presence of biliary tree disease. When prolonged, this signal can cause ST changes[4].

A better understanding of the link between pancreatitis and pericarditis may produce a more expedient diagnostic approach, thus lowering costs and shortening patient morbidity.

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WHEN OCCAM'S RAZOR WAS WRONG | SNEHA PARIKH, MD [R3]

CHIEF COMPLAINT: Hypoxia

HISTORY: The patient is a 74 year old male with CAD, neurosarcoidosis, and optic neuritis on prednisone who was found unresponsive at home by family. He was hypoxic to 40% when EMS arrived, and EKG showed anterior STEMI. He was taken emergently to the cath lab and had a stent placed to his LAD. The patient was then admitted to the CVICU where he remained hypoxic and became increasingly hypotensive requiring vasopressor therapy.

Upon further questioning, it was learned that the patient had increasing cough and shortness of breath for a week. He denied any fevers.

PHYSICAL ABNORMALITIES: Over the next 12 hours after admission, the patient became febrile, tachycardic, hypotensive, and tachypneic. He was somnolent but easily arousable and fully oriented. Cardiac exam was essentially normal. Lung exam revealed diffuse crackles in all lung fields with moderate respiratory distress.

DIAGNOSTIC STUDIES: Labs were notable for leukocytosis and acute kidney injury. Echo demonstrated severely reduced LVEF though Fick cardiac output was normal. Chest x-ray showed bilateral patchy opacities and pulmonary edema.

DIFFERENTIAL DIAGNOSIS: The patient was initially thought to have cardiogenic shock and pulmonary edema secondary to STEMI. However, his presentation was also concerning for septic shock secondary to pneumonia, including opportunistic infections given steroid use.

CASE PRESENTATION: Based on the chest x-ray findings, the patient was first diuresed. However, his respiratory status continued to worsen overnight, and repeat chest x-ray showed persistent opacities. He also became febrile with increasing vasopressor requirements. The patient was placed on BiPAP and started on broad spectrum antibiotics. Pulmonology was consulted and bronchoscopy was performed. He was intubated for the procedure and was unable to be extubated afterward. Given that his clinical picture pointed more toward septic shock rather than cardiogenic shock and his respiratory status did not improve with broad spectrum antibiotics, there was increasing concern for PJP in the setting of steroid use. Bactrim was started empirically, and bronchoscopy studies later returned positive for PJP. The patient was then transferred to the MICU for further management of septic shock and respiratory failure. He ultimately developed multi-organ dysfunction syndrome, and the family decided on no further escalation of care due to his poor prognosis. The patient died in the hospital several days later.

CONCLUSION: As this case demonstrates, the most parsimonious explanation for the patient's multiple problems cannot always be applied.

A CASE OF HANTING IN THE WOODS | MATTHEW PETERSEN [MS3]

CASE PRESENTATION: The patient is a 56-year-old previously healthy woman admitted to an outside hospital with a one-week history of fevers, chills, headaches and two observed syncopal episodes. Patient had not recently travelled outside the US, reports no history of substance abuse and no sick contacts. She lives alone in rural Montana and has had multiple zoonotic exposures including rodents, cattle, horses and deceased rabbits killed by her cat.

On admission, patient endorsed nausea, shortness of breath, arthralgias and myalgias. In the ED, fluids were given, but her course worsened considerably. The patient experienced rapid respiratory decline with an increased A-a gradient and a CXR consistent with acute respiratory distress syndrome (ARDS). She received veno-venous Extrecorporeal Membrane Oxygenation (ECMO) cannulation at the outside hospital prior to being transferred to the University of Utah.

PHYSICAL ABNORMALITIES: On exam, patient had a temperature of 98.1, pulse 105, respiratory rate 14 and blood pressure ranging from 70/40 to 130/70. She was alert, oriented and cooperative on admission, but respiratory distress required sedation and intubation. Cardiovascular exam was grossly normal, demonstrating regular rate and rhythm without appreciable murmurs. Lung fields were diffusely course with inspiratory rales.

LABS, IMAGING, PATHOLOGY AND MICRO: Chest x-ray showed 5-lobe alveolar and interstitial infiltrates. CBC and BMP were within normal limits. Bronchoalveolar lavage showed hemorrhagic bronchus. Respiratory cultures showed no growth. Patient's infectious workup was negative for Hepatitis C, Adenovirus, HSV 1 and 2, influenza A and B, parainfluienza, and RSV. Urinalysis, blood, viral respiratory panel and fungal cultures showed no growth. After discharge, Hantavirus antibodies were found to be positive while Francisella Tularensis antibodies were negative.

DIFFERENTIAL DIAGNOSIS: Given the patient's symptoms and imaging, causes of acute respiratory distress syndrome were considered including sepsis, inhalation injury, aspiration and severe pneumonia. Ultimately, the patient's history of infectious prodrome, pulmonary symptoms and zoonotic exposures led to a thorough infectious workup. Exposure to rural rodents and their droppings was strongly suggestive of Hantavirus.

TREATMENT: No definitive treatment or cure exists for Hantavirus, the patient received supportive respiratory care in the ICU including intubation, oxygen therapy and ECMO. Approximately one week after admission patient was discharged to home without supplemental oxygen.

CONCLUSION: Hantavirus is a relatively rare condition occurring mostly in rural areas with exposure to the droppings of infected rodent hosts. Because pulmonary complications are life-threatening, early recognition through detailed history taking and laboratory studies is crucial to a full recovery.

ALLERGIC RESPONSE TO IVF HORMONES | REBECCA POWELL [MS3]

IDENTIFICATION: Patient is a 23 year old female with no previous medical history who is G4P3, with her current pregnancy 6 weeks post IVF implantation, serving as a surrogate.

CHIEF COMPLAINT: Presented with 1 week of worsening SOB, cough and chest pain.

HISTORY: Patient was in her previous healthy state with no medical complaints until 1 week prior she developed shortness of breath. It progressed to pleuritic chest pain and dizziness. She denied dry eyes, dry mouth, malar rash, oral ulcers, dysphagia, or raynauds. No history of chronic diarrhea or pneumonia. Her only medications were aspirin 81mg qd, progesterone supplement bid x4 week, progesterone injection 2ml x4 week, and estradiol 0.2ml x4 week.

PHYSICAL ABNORMALITIES: Vitals normal | Only abnormality was diminished inspiratory effort secondary to cough.

LAB RESULTS: Outside CT angio from two days prior to presentation showed peripheral ground glass opacities and consolidation with bilateral hilar lymphadenopathy. No PE | Bronchoscopy showed an eosinophilic cell % of 55 on differential | ANA, RF panel, ENA 5, ANCA and UA were all unremarkable.

DIFFERENTIAL DIAGNOSIS: Presumed diagnosis was eosinophilic pneumonia due to peripheral consolidations on CT and case reports of eosinophilic pneumonia resulting from use of IM progesterone. This was confirmed with bronchoscopy results showing primary eosinophilia. PE had been ruled out by CT angio of the chest. Cryptogenic organizing pneumonia wasn't favored due to hemoptysis. Pneumonitits secondary to autoimmune disease or pulmonary vasculitis was unlikely due to no evidence or history of autoimmune disease.

CASE PRESENTATION: 23 yo female, serving as surrogate, six weeks post IVF implantation. IM progesterone was started two weeks before symptom onset. At OSH ED, D-dimer was elevated and CT angio of chest reveled bilateral peripheral ground glass infiltrates and no pulmonary embolism. She was admitted for evaluation. Bronchoscopy with BAL admission showed 55% eosinophils on cell differential. IV solumedrol was started. Patient improved on steroids and was discharged.

DISCUSSION: The acute eosinophilic pneumonia was likely caused by IM progesterone, and a literature search reveled several similar cases. This patient's symptoms occurred 2 weeks after initiation of IM progesterone with acute respiratory illness of less than one week duration, similar to the other case reports. The common presenting symptoms were shortness of breath and cough.

CONCLUSION: Rapid recovery occurs within days after stopping IM progesterone preparation and starting steroid therapy. The differential diagnosis of acute pneumonia-like symptoms during IVF pregnancy should include eosinophilic pneumonia.

FROM HEMATOMA TO SARCOID TO CANCER: DIAGNOSTIC DIFFICULTIES IN A NON-ENGLISH SPEAKING PATIENT | HEIDI SAXTON [MS4]

HISTORY: The patient is a 64 year old woman from Puerto Rico who was in good health until four months ago, when she developed shortness of breath and was found to have "water on the lung," which was diagnosed and treated at a Puerto Rican hospital. Three months ago, she developed pain and swelling in her right leg, was diagnosed with a clot, and was started on warfarin. The pain and swelling persisted, and so she traveled to Utah one week ago for further evaluation. She also noted worsening trouble breathing and constipation. The history was obtained overnight using a virtual interpreter. The patient did not have her medical records, and the outside hospital in Puerto Rico was not contacted.

PHYSICAL ABNORMALITIES: Examination was significant for tenderness and non-pitting edema in her right lower extremity, decreased breath sounds on the right, firm bilateral inguinal lymphadenopathy, and splenomegaly.

LAB RESULTS: Abnormalities included INR 4.4, LDH 920, calcium 15.5, low PTH, low 25-hydroxyvitamin D, and high 1,25-hydroxyvitamin D.

DIAGNOSIS: CT L-spine showed enlargement of psoas musculature consistent with hematoma, or less likely tumor. This finding in the setting of elevated INR was concerning for retroperitoneal bleed, and anticoagulation was reversed. Chest CT revealed a large pleural effusion and hilar mass. Thoracentesis showed an exudative effusion and no malignant cells, biopsy of the mass was negative for malignant cells, and PTHrp returned normal. Sarcoid and infectious etiologies were then considered. The team's findings and plans were communicated through virtual interpreters. PET scan showed metabolic hyperactivity in the right retroperitoneal mass and right mediastinum. When a new Spanish-speaking member of the team communicated the concerns for possible malignancy, it was discovered that the patient had undergone inconclusive bone marrow biopsies in Puerto Rico and was anticipating a diagnosis of cancer. CT-guided biopsy showed diffuse large B-cell lymphoma.

TREATMENT: The patient will undergo six rounds of R-CHOP chemotherapy.

CONCLUSION: DLBCL is the most common form of non-Hodgkin lymphoma. Based upon the International Prognostic Index for NHL, this patient is in the high-risk group, giving her a 3-year overall survival rate of 59%. Having a Spanish-speaking individual on our team made us better able to direct our inquiry and ascertain the patient's history and insight into her disease than we previously could, even with immediately available virtual translation resources.

A PUZZLING STORY OF HEMOLYTIC ANEMIA | MIRANDA TRACY, MD [R2]

CASE PRESENTATION: The patient is a 64 year old female who presented to the ED with two weeks of dyspnea on exertion, nausea, bilateral leg swelling, LUQ abdominal pain, dark urine, and yellow skin along with 1 month of fatigue. The patient's family mentions that she has been very weak, having trouble with a single step into her house and using walls to help walk around the house. Her vitals are pertinent for 93% O2 on 2 L. On exam she has slight sclera icterus of the lower sclera and jaundice of the face. She has a 2/6 systolic murmur heard best at LLSB. Her LUQ is tender to palpitation. There is 3+ pitting lower edema bilaterally. Neuro exam pertinent for weakness of the lower extremities and diminished ankle reflexes, proprioception and sensation not completed. Labs are pertinent for a TBili of 4.2 with normal liver enzymes, hgb of 5.3 with an MCV of 130, LDH of 1713 and haptoglobin <10 and reticulocyte count of 1.5%. The patient was given 2 units of PRBC for anemia, admitted and started on steroids for presumed diagnosis of autoimmune hemolytic anemia. A peripheral smear showed occasional schistocytes and spherocytes. The combination of patient's jaundice, elevated bilirubin, low hemoglobin and schistocytes on smear pointed suggested microangiopathic hemolytic anemia. The patient's symptoms, however, did not fit TTP or HUS. Because patient was Coomb's negative and had an initial MCV of 130, B12/folate was done with a resulting B12 of <50. The patient ate a varied diet and had no past surgeries to suggest B12 deficiency. A gastric parietal cell IgG was largely elevated, along with methylmalonic acid and homocysteine levels suggestive of pernicious anemia.

The patient was started on 1 mg of B12 daily. She was discharged to a skilled nursing rehab for intense physical therapy due to balance issues secondary to subacute combined degeneration.

DISCUSSION: The patient's severe anemia was due to intramedullary hemolysis from pernicious anemia, a rare presentation of severe vitamin B12 deficiency. It is thought intramedullary destruction of red blood cells may relate to endothelial damage from elevated homocysteine levels as a result of pernicious anemia. Careful attention should be paid to the possibility of vitamin B12 Deficiency in patients with severe anemia and hemolysis. Fortunately, this patient had a high MCV suggestive of macrocytic anemia resulting in the appropriate diagnosis.

WHEN THE CAUSE OF ABDOMINAL PAIN IS HIDING SKIN DEEP | ASHLEY TRANE, MD [MS4]

FINALIST

IDENTIFICATION: The patient was a previously healthy, 23-year-old male who presented with abdominal pain, bloody diarrhea, and a lower extremity rash.

CHIEF COMPLAINT: Abdominal pain with a rash

HISTORY: The patient was a 23-year-old previously healthy male who presented to the emergency room with a 10-day history of a lower extremity rash and a 6-day history of left-sided abdominal pain. He described the rash as a progressively increasing group of large, red, pleuritic bumps. His rash started on his ankles and spread to his upper thighs with isolated lesions on his elbows. His left-sided abdominal pain started 4-days later. It was diffuse and colicky in nature and accompanied by bloody diarrhea. He denied any nausea, vomiting, fevers, URI symptoms, or mouth ulcers.

He initially presented to an outside hospital where a CT scan and colonoscopy showed terminal ileitis. There were several ulcers in the distal terminal ileum and rectum consistent with Crohn's ileocolitis. He was started on IV steroids. However, his abdominal pain continued so he presented to our hospital for an additional evaluation.

PHYSICAL ABNORMALITIES: Our exam was notable for a maculopapular, pustular rash on his legs, buttocks, and elbows. Several purpuric plaques contained a central erosion. His abdomen was diffusely tender throughout. No mucous membrane ulcers or lesions were noted.

DIAGNOSTIC RESULTS: A terminal ileum biopsy demonstrated multiple PMN's with no ulceration or granulomas. A skin biopsy showed neutrophilic infiltrate in the dermal layers with a predominantly superficial perivascular distribution.

DIFFERENTIAL DIAGNOSIS: Given the terminal ileocolitis and bloody diarrhea, there was an initial concern for Crohn's disease with pyoderma gangrenosum. Other considerations included infectious enteritis such as EHEC. The skin biopsy was sent for direct immunofluorescence analysis. It showed IgA and C3 deposition in the dermal blood vessel walls consistent with an IgA vasculitis. He was diagnosed with Henoch-Schonlein IgA leukocytoclastic vasculitis.

CONCLUSION: While 90% of HSP patients are less than 10-years-old, HSP needs to remain on the differential for adults presenting with a purpuric rash on the extensor surfaces of the extremities, followed by a colicky abdominal pain. The incidence in adults varies from 3.4 to 14.3 cases per million. Given that adults have a more severe course and are more likely to develop long-term renal disease; early diagnosis and steroid treatment are imperative.

DIGITAL ISCHEMIA AND NECROSIS AS A PARANEOPLASTIC SYNDROME | ISABEL VANDERSLICE [MS3]

CHIEF COMPLAINT: "Fingers feel weird and are black and blue"

HISTORY: A 51-year-old woman with no significant past medical history presented to her primary care provider with 1.5 months of sensory change in finger tips that progressed to a Renaud's-like cold sensitive vasoconstriction, and was started on amlodipine.

PHYSICAL ABNORMALITIES: She returned with necrosis of the fingertips and was hypoxic, tachycardic, and febrile, with oxygen saturation of 75%. Palpable supraclavicular adenopathy was discovered on exam.

LAB AND IMAGING RESULTS: Chest x-ray showed bilateral effusions suggesting pulmonary edema, yet transthoracic echocardiography showed normal LV and RV function. CTA of the abdomen and pelvis to evaluate for vasculitis found mesenteric and retroperitoneal lymphadenopathy. US guided biopsy of the supraclavicular node demonstrated metastatic adenocarcinoma suspicious for a colorectal primary (CK20+, CDX2+). She was also found to have a grossly positive ANA 1:1280 and elevated SSA Ab of 228. APLAS testing was negative. Colonoscopy revealed a fungating partially obstructing large mass found on pathology to be moderately differentiated adenocarcinoma.

DIFFERENTIAL DIAGNOSIS: Autoimmune diseases related to vasculitides were high on the differential, especially with her high ANA and SSA Ab; however, paraneoplastic syndrome was also considered and was found to be most likely when CA125 antigen labs became positive, and was confirmed by colonoscopy and biopsy.

TREATMENT: A port placed and FOLFOX was initiated (J Clin Oncol. 2004 Jan 1;22(1):23-30.) as soon as possible for the treatment of adenocarcinoma of the colon. Hydroxychlorquine, NTG paste, amlodipine, prednisone, and anticoagulation were initiated by rheumatology as workup by oncology began.

DISCUSSION: Digital ischemia leading to necrosis, rare and mostly associated with autoimmune conditions such as SLE and Sjogrens, generally does not prompt investigation for a paraneoplastic process, but may represent an underlying malignancy. Complete workup with rheumatology, vascular, GI, and oncology were required to make a diagnosis in a previously healthy woman with no other symptoms, personal risk factors or family history of colon cancer.

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PYRIDOSTIGMINE POISONING THE PACER | ANDREW WALKER, MD [R2]

CASE PRESENTATION: This is a 72 year old Caucasian male with a history of myasthenia gravis (MG) on pyridostigmine, atrial fibrillation on flecainide and warfarin, and CVA with residual left sided weakness who presented to the ED after he experienced orthostasis and fell getting out of bed. Approximately two weeks prior to this the patient noted increasing blurry vision and difficulty swallowing, both of which are typical symptoms of his MG. As such, he increased his pyridostigmine on his own accord from 180mg twice a day to three times a day and noted improvement in both his vision and swallowing. One week prior he began to note increasing fatigue and orthostasis resulting in a couple of falls. On the day of presentation the patient noted a blood pressure of 84/54 and a heart rate of 45 recorded with his home machine after one of these falls which prompted him to call EMS. In the ED the patient was confirmed to be hypotensive (89/54) and bradycardic (30's-40's).

PHYSICAL EXAM: Other than bradycardia, the patient had a normal cardiac exam. His neuro exam was negative for any ptosis or strabismus. He had 2/5 strength on his left upper and lower extremity which was chronic from his prior CVA.

NOTABLE OBJECTIVE DATA: Initial EKG demonstrated complete heart block with junctional escape at a rate of 48 BPM. A second EKG obtained 30 minutes later showed junctional rhythm at a rate of 41 BPM and no P waves. Electrolytes and troponin were all normal as was a non-contrast head CT. An echocardiogram was largely unremarkable.

DIFFERENTIAL DIAGNOSIS: Broadly speaking, CVA, sick sinus syndrome, ACS, and worsening MG were all considered, but felt less likely based on reassuring labs, imaging, and patient history. This left our working diagnosis of medication induced complete heart block.

TREATMENT: Initially his blood pressure responded to a small fluid bolus. He was then monitored in the cardiac ICU in the case pacing was needed. We held his pyridostigmine and he reverted to a sinus rhythm at a rate of 60-70. He received a dual chamber pacemaker 48 hours later.

DISCUSSION: This is a case of complete heart block secondary to pyridostigmine which the patient was taking at high doses for his MG. Implantation of a dual chamber pacemaker allowed the patient to restart this extremely helpful medication without its bradycardic side effect.

THE GREAT MASQUERADER PRESENTS AS RAPID VISION LOSS IN AN HIV-INFECTED MAN OFF HAART | LANA WEBER [MS3]

INTRODUCTION: A 42 year-old HIV-infected man, diagnosed in 2007 and off antiretroviral therapy for two years, presented to the Moran Eye Center at the University of Utah with five days of progressive, non-painful bilateral vision loss.

HISTORY: His symptoms began with clouding of his vision in the left eye. At time of presentation, he was only seeing light-dark contrast and rough outlines of figures in both eyes. He was diagnosed with bilateral acute necrotizing retinitis, started on empiric therapy with acyclovir and sulfamethoxazole/trimethoprim and admitted to the University of Utah Hospital for systemic workup.

Notably, he denied a history of eye pain or pruritus, headache, fever, chills or symptoms of opportunistic infection. He did report URI symptoms one-month prior and loss of muscle mass over the past year.

PHYSICAL ABNORMALITIES: Physical exam revealed hand-motion vision at 10 cm in each eye. His pupils were sluggishly reactive to light. No skin rashes were noted. His left tympanic membrane was erythematous with reduced hearing.

DIFFERENTIAL DIAGNOSIS: Opportunistic infections were the most likely etiology with several viral, fungal and bacterial diseases considered and tested: herpes simplex, varicella zoster, cytomegalovirus, toxoplasmosis, tuberculosis and syphilis.

LAB RESULTS: Laboratory data revealed a highly reactive RPR (1:512) with CSF analysis showing elevated WBCs (14 cells per microliter), protein (91 mg/dL) and a reactive VDRL (1:1). His CD4 count was 182.

DISCUSSION: Syphilis has been called “the great masquerader” for its ability to present with nonspecific symptoms, such as this patient’s URI, that imitate various infectious and inflammatory conditions. Ocular syphilis, a clinical manifestation of neurosyphilis, can occur at any stage of the disease with uveitis being the most common presentation of vision loss. Unlike CMV retinitis, which has decreased in the post-HAART era, ocular syphilis has not decreased. , In March 2016, the CDC announced an increase in cases of ocular syphilis in the U.S. with 200 cases reported in 20 states in the prior two years.3 Aside from case reports, there is little evidence regarding the treatment and outcomes of individuals with HIV and ocular syphilis. A 2011 review found that 34/35 HIV-infected individuals with ocular syphilis regained their sight following treatment with IV penicillin.

CONCLUSION: The patient was diagnosed with ocular syphilis and initiated on 14 days of continuous infusion of 18 million units of penicillin G. This case illustrates the importance of vision screening in HIV-infected individuals as well as continued surveillance for STIs in this population.

(1) Moradi A, Salek S, Daniel E, Gangaputra S, Ostheimer TA, Burkholder BM, Leung TG, Butler NJ, Dunn JP, Thorne JE. Am J Ophthalmol. 2015 Feb;159(2):334-43.e1. doi: 10.1016/j.ajo.2014.10.030. Epub 2014 Nov 5. (2) Gonzalez-Lopez JJ, Guerrero ML, Lujan R, et al. Factors determining serologic response to treatment in patients with syphilis. Clin Infect Dis. 2009; 49:1505–1511. (3) Peterman, T, Workowski, K. Clinical Advisory: Ocular Syphilis in the United States. CDC.gov. (4) Tucker JD, Li JZ, Robbins GK, Davis BT, Lobo AM, Kunkel J, Papaliodis GN, Durand ML, Felsenstein D. Sex Transm Infect. 2011 Feb;87(1):4-8. doi: 10.1136/sti.2010.043042. Epub 2010 Aug 26. Review.

STAGNANT STONES AND BROKEN BONES | JOSEPHINE WRIGHT, MD [R1]

IDENTIFICATION/CHIEF COMPLAINT: 60-year-old post-menopausal woman with recurrent UTI sent from her PCP for elevated creatinine and hypercalcemia.

CASE PRESENTATION: The patient recently presented to the ED complaining of 1-week history of dysuria, bilateral flank pain, urinary frequency and urgency. She was found to have a UTI and was discharged with a prescription of Macrobid. However, when she returned home from the ED, she developed hematuria. She was afraid so did not start the antibiotics. Her symptoms persisted. She saw her PCP a few days later, at which point routine labs were drawn. She was advised to return for admission after labs came back with elevated creatinine and hypercalcemia. She states she has had recurrent UTIs, about once a year for the past 5 years, which usually resolve with oral antibiotics. However, she states her most recent UTI 9 months ago was 'more complicated' and she was found to have 'big stones which required surgery.' She thinks that she also had acute kidney failure at that time. Unfortunately she was lost to follow up and did not end up having any intervention. ROS was positive for fatigue, myalgia, anorexia, heartburn, depression and chronic constipation.

PHYSICAL ABNORMALITIES: The patient exhibited psychomotor retardation, flat affect and poor eye contact. Her exam was otherwise unremarkable. She did not have any abdominal tenderness or flank pain.

LAB RESULTS: Significant for WBC 11.2 (71% neutrophils), Creatinine 2.8, Calcium 11.1, PTH 70. UA with SG 1.011, pH 8, 823 WBC/hpf, 17 RBC/hpf, many bacteria, large LE. UCx positive for Proteus mirabilis, which was resistant to nitrofurantoin.

DIFFERENTIAL DIAGNOSIS: We suspected renal stones given recurrent UTI in the context of hypercalcemia. An abdominal KUB showed bilateral large staghorn calculi with dilation of the right renal pelvis by large stone. This was confirmed with renal ultrasound and CT KUB.

TREATMENT/OUTCOME: Urology was consulted. Unfortunately, her calculi were too large for percutaneous lithotripsy. They recommended bilateral ureteral stent placement for urinary decompression and definite stone management which would consist of multiple staged procedures. Unfortunately, the patient refused to undergo ureteral stent placement and left AMA. She will likely also need parathyroidectomy in order to treat the likely underlying cause of her stones.

CONCLUSION: The patient presented with the classic symptoms of hyperparathyroidism including 'moans, stones, groans, psychic overtones.' Her hypercalcemia may have contributed to her impressive staghorn calculi, which is the likely cause of her AKI and recurrent UTIs.

FINALIST

CASE PRESENTATION: Patient is an 80 year-old man who was recently seen in the ED for a painful vesicular rash over the right ear extending down the right side of his neck. He was diagnosed with herpes zoster and sent home with valacyclovir. He presents one week later, with his wife who reports that the patient has been extremely confused, disorganized and irritable. He may have missed a few doses of valacyclovir. The patient also complains of a diffuse pruritic rash, severe headache, photophobia, nuchal rigidity, chills and right ear pain, all of which developed around the same time as the altered mental status.

PHYSICAL EXAM ABNORMALITIES: Uncomfortable appearing man scratching his arms uncontrollably. Erythematous vesicular rash from the right mastoid along angle of the mandible to the neck. Yellow discharge in right ear canal, exam limited by tender external ear. Nuchal rigidity, unable to touch chin to chest. Diffuse, symmetric maculopapular rash over neck, chest, waist band and groin creases. Oriented to person, place and time but poor concentration, confused and repeated himself often. CN II-XII initially intact. However, one week later, he developed right eye ptosis and facial droop.

RESULTS: INR 2.7. Non-contrast head CT showed mild diffuse cerebral atrophy, no acute hemorrhage. MRI showed no acute intracranial process, meningitis or cerebritis. CSF was clear, colorless, glucose 109, protein 69.7, WBC 133 (83% Lymphocytes) HSV PCR negative, VZV PCR positive.

DIFFERENTIAL: Presumed diagnosis was zoster meningoencephalitis given altered mental status with meningeal signs in the context of recent painful vesicular rash. The distribution of the vesicular rash followed a C2-C3 dermatomal distribution. Bacterial meningitis was less likely given the slow progression of symptoms. Initial testing ruled out drug intoxication and CVA. With the development of ipsilateral facial paralysis, we suspected Ramsay Hunt Syndrome. As for the maculopapular rash, we suspect this represents drug rash; interestingly, a symmetrical drug-related intertriginous and flexural exanthema ('baboon syndrome') has been described with valacyclovir.

TREATMENT/CONCLUSION: Valacyclovir was discontinued, with subsequent complete resolution of the diffuse pruritic rash. The patient was treated with IV acyclovir. He was initially also started on empiric therapy for bacterial meningitis; this was discontinued after results of the LP indicated a viral etiology. Mental status and nuchal rigidity improved on IV acyclovir. Post-herpetic neuralgia treated with gabapentin. After development of facial paralysis, course of IV acyclovir was extended for a total of 4 weeks with initiation of concurrent steroid therapy.

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