Clinical Vignette Competition

2015

Utah ACP Residents & Fellows Committee

Kencee Graves, MD – Chair
Scott C. Woller, MD FACP
Rosane Fernandez, MD
David Glodowski, MD
FALL BANQUET & CLINICAL VIGNETTE PROGRAM
TUESDAY, SEPTEMBER 22, 2015
University of Utah | Health Sciences Education Building | Alumni Hall

5:00 PM  WELCOME & OPENING REMARKS
Residents & Fellows Committee

5:30 PM  PRESENTATIONS
Atrial Chorionic Gonadotropin
Presented by: Wade Brown, MD

Black Ears After a Binge
Presented by: Jason Carr, MD

Time = Brain: Code Stroke Leads to a Near Miss
Presented by: David Gill, MD

A Woman With Nausea, Vomiting, and Intermittent Urticaria
Presented by: Russell Johnson, MD

A Curiosity of Calcium
Presented by: Andrew Justice, MD

6:20 PM  ANNOUNCE RUNNERS-UP AND 1ST PLACE

6:30 PM  CLOSING COMMENTS – Adjourn for Dinner & Awards Ceremony
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.
THE HYPERTENSIVE-HYPOTENSIVE DILEMMA; A SINGLE DIAGNOSIS? | JASON ALLEN, MD [R3]

CASE PRESENTATION: Mr. C is an 84-year old male with past medical history of DM2, CAD, hypertension and hyperlipidemia who presents to the ER with one month of lightheadedness, dizziness, and recurrent syncope. One month ago he was seen in the emergency room for identical symptoms and received IV fluids after he was noted to have positive orthostatic vitals. However, despite a diligent attempt to stay hydrated, he continues to have dizziness with position changes. Over the past month, symptoms have progressed, and he is now confined to his bed, despite being a physically active person just a few months ago. He denies having any recent weight loss, abdominal pain or palpitations. He is admitted with persistent orthostatic hypotension.

PERTINENT PHYSICAL EXAMINATION FINDINGS: Orthostatic vital signs: lying 145/63, P 70s, sitting 106/43, P 80s, standing 87/50, P 110s with significant pre-syncopal symptoms. His cardiac, pulmonary and neurological examinations are normal. Throughout his stay, his systolic blood pressures range from 140 to 220s.

DIFFERENTIAL DIAGNOSIS: Initial differential is broad including medication effect, volume depletion, cardiovascular disease, adrenal insufficiency, infection, malignancy and neurological conditions including autonomic neuropathy.

PERTINENT STUDIES: Initial evaluation demonstrates normal CBC, CMP, TSH, troponin, EKG and telemetry. Echocardiogram and cosyntropin-stimulation testing are normal. HbA1c is 8.4. Head CT and brain MRI are also negative. Plasma metanephrines elevated at 1.91 (normal <0.49) and normetanephrine elevated to 4.13 (normal < 0.89). Abdominal MRI demonstrates a mass on the right adrenal consistent with a potential pheochromocytoma.

TREATMENT: He is initially treated with aggressive IV fluids, then midodrine, fludrocortisone and SSRI are initiated; all without improvement in his orthostatic hypotension. Once elevated metanephrines and adrenal mass are noted, surgical resection of the adrenal mass is performed and pathology confirms a pheochromocytoma (Figure 1). Following resection, Mr. C experiences rapid resolution of his orthostatic hypotension and no further syncope.

CONCLUSION: Orthostatic hypotension typically is related to intravascular volume depletion. Traditionally, findings of a pheochromocytoma are persistent hypertension. Orthostatic hypotension as the sole finding in a pheochromocytoma is not common. The exact method of action is not clear but hypothesized to be related to down-regulation of alpha-adrenergic receptors from persistently elevated metanephrines. Although not a common diagnosis, a pheochromocytoma should remain on the differential in those with resistant orthostatic hypotension.

Figure 1. H&E: Nest of cells typical of pheochromocytoma
CHIEF COMPLAINT: Muscle weakness

HPI: 81-year-old gentleman with Hx of SSS with pacemaker, MGUS, and atrial fibrillation who presents with a 3 month history of dyspnea, anorexia, and 31 pound weight loss. Over the past two months, he has noted progressive muscle weakness in his upper and lower extremities. The weakness then progressed to include numbness and increased pain with movement of his lower extremities. Workup was significant for CT chest with focal interstitial and alveolar opacities and an EGD that showed esophageal candidiasis. He was prescribed a two-week course of prednisone ending two weeks prior to presentation for suspected polymyalgia rheumatica. After the course of prednisone, he developed an abscess on his RLE that was incised and drained. He was prescribed a 10 day course of Keflex without improvement. Social history pertinent for working in his garden often.

PHYSICAL ABNORMALITIES: Exam was notable for two 2 cm well-circumscribed, mobile, erythematous tumors in his L axilla and L upper chest. He had 3/5 muscle strength in his RUE, 4/5 muscle strength in his LUE, and 3/5 muscle strength in his b/l LE. He had decreased sensation to light touch and pinprick most pronounced in his b/l LE. He had 2+ DTRs throughout.

LAB RESULTS: CRP and ESR were elevated at 21 and 61, respectively. Wound culture of R thigh revealed 2+ nocardia-like species.

DIFFERENTIAL DIAGNOSIS: The presumed diagnosis was nocardiosis. We performed CT scans of his head, neck, chest, b/l UE, abdomen/pelvis, and b/l LE. These scan were notable for 3 ring-enhancing masses in his brain, parenchymal abscess in his left lower lung, and innumerable abscesses in his b/l LE along with pyomyositis of the thigh musculature, all of which is consistent with disseminated nocardia. TEE was unremarkable. He underwent an immunodeficiency workup that showed MGUS, which was previously known.

TREATMENT: He was started on IV Bactrim, ciprofloxacin, and meropenem due to disseminated infection. After minimal improvement and worsening SOB, he transitioned to comfort care and died peacefully with more than 15 family members present in the room.

CONCLUSION: Without evidence of immunosuppression, we did not initially suspect nocardia, but learned that it is only an opportunistic infection in roughly 2/3 of patients. It is commonly found in soil and should be considered in patients who enjoy gardening. Nocardia commonly relapses despite appropriate treatment. It can disseminate to virtually any organ but has a predilection for the CNS.
PRESENTATION: A fifty-nine-year-old overweight male with depression and anxiety presented to the emergency department after “passing out.” On interview, the patient reported, “I went to the fridge this morning, felt weird, and passed out cold.” He clarified that he was standing in front of his refrigerator and then woke up on the ground. The patient denied preceding lightheadedness, rushing in his ears, vision changes, and diaphoresis. He denied loss of urine or injury to self. He also denied subsequent confusion or focal neurologic deficits. However, he did endorse feeling anxious and sweaty since the incident. In the emergency department, the patient’s heart rate was noted to be in the 120s-140s with atrial flutter on ECG. The patient denied a history of arrhythmia, palpitations, or past similar episodes.

ASSESSMENT: The patient was fluid resuscitated and placed on diltiazem with return to normal sinus rhythm. The etiology of this patient’s new onset atrial flutter was initially unclear. He presented without symptoms or signs of infection, as well as a bland urinalysis and an unrevealing chest X-ray. Sequential troponins were negative (0.01, 0.02, 0.02 ng/mL), and an eventual left heart catheterization revealed non-occlusive coronary artery disease. The patient presented with no history of COPD, and pulse oximetry in the emergency department showed an oxygen saturation of 96% on room air. The patient denied a history of significant alcohol consumption including no history of binge drinking. He reported no history of thyroid dysfunction, and presented with a TSH of 1.3 uIU/mL. The patient’s magnesium was within normal limits (2.0 mg/dL). He did report initiating a human chorionic gonadotropin (hCG) diet four days prior to presentation, however. He reported losing 12 pounds over 4 days.

DISCUSSION: Both subacute and overt hyperthyroidism are well-known causes of atrial fibrillation and flutter.1 Additionally, hCG is known to have activity at the TSH receptor with between 1.5 – 10 times the potency of TSH.2 This patient had recently started receiving daily IM injections of hCG. Though the patient’s TSH was within normal limits, it was lower than past levels (1.77 uIU/mL), and would not be expected to reflect the very recent initiation of an exogenous TSH analog. Additionally, the patient was attempting extreme calorie reduction (500 Calories per day) with resultant hypovolemia (BUN: CR of 20, HGB of 17.7g/dL, and loss of > 10 lbs. in one week). The patient’s hCG diet, with daily IM administration of a TSH analog and profound hypovolemia, was eventually determined to play a significant role in his new onset atrial flutter. After a complete workup, this patient was encouraged to discontinue his hCG diet and was discharged with a Holter monitor as well as cardiology follow up.

CASE PRESENTATION: A 54-year-old woman with a history of drug abuse, rheumatoid arthritis and thyroid ablation for unknown condition presents with two weeks of progressive painful, black skin lesions involving her ears, arms and thighs. Two weeks prior, she and her partner shared a batch of cocaine and both subsequently developed black skin lesions. Her only medication is PRN ibuprofen. She is otherwise asymptomatic.

EXAM: Vital signs are normal. She is agitated but non-toxic appearing. Exam otherwise notable only for necrotic appearing eschars involving her ears and limited areas (~5 cm²) of her arms and thighs.

STUDIES: CMP is within normal limits and CBC reveals leukopenia. Toxicology screen is positive for cocaine. She has elevated ESR, CRP, c-ANCA titer of 1:10240, low serum C4, positive ANA and dsDNA. Hepatitis C, cryoglobulin and HIV testing are negative. Skin biopsy consistent with a leukocytoclastic vasculitis.

DIFFERENTIAL: Given her elevated ESR, CRP and grossly positive c-ANCA titer, a small vessel vasculitis is possible, but no other organ systems are affected and an autoimmune vasculitis is unlikely to present concurrently. Further, the differential of necrotic skin itself is broad, but in this case is significantly narrowed by the history of a partner with similar skin changes. The shared element of intra-nasal cocaine is a risk factor for Hepatitis C, and associated cryoglobulinemic vasculitis, but testing for these is negative. Needle use can inoculate infections and cause complications such as sepsis or DIC, but her labs and clinical presentation are inconsistent with this. A shared drug-induced reaction would match the timeline and symptoms. Levamisole is a known adulterant in cocaine and has been reported to cause skin necrosis.

TREATMENT AND COURSE: She was treated with supportive, strongly encouraged to seek drug counseling, and discharged with plan for close follow-up.

CONCLUSION: Levamisole was removed from the US market in 1999 due to concerns for serious adverse reactions including agranulocytosis and skin reactions. It is still in use legally as a veterinary anti-helmintic and illicitly as an adulterant for powered cocaine due to favorable texture and perceived intoxication enhancing properties. Prior case reports of levamisole toxicity report skin necrosis, characteristically involving the ears, c-ANCA positive vasculitis and leukopenia in the setting of recent cocaine use, all of which are mirrored in our patient. Levamisole reportedly taints 80% of domestic cocaine and should be considered in the differential of a drug abuser presenting with necrotic skin.

IDENTIFICATION: Ms. H is a G4P3003 31-year-old female at 18 weeks gestation admitted with recurrent fever.

HISTORY: Ms. H had high rigorous fevers to 103F since week 16 of pregnancy. She described diffuse myalgias and new onset symmetric hand/foot swelling associated with burning neuropathy. The fevers occurred daily with no pattern. She had similar symptoms around week 8 of pregnancy; resolved after 3 weeks and a course of cephalaxin for suspected urinary infection. She had been hospitalized during her second pregnancy for shock, with no infectious organism identified. Her other pregnancies were uncomplicated. She had no other medical history. The patient was taking Tylenol, a prenatal vitamin and IV ertapenem started 5 days prior. Her husband frequently visited California for work. They had no pets and lived in an urban area.

PHYSICAL ABNORMALITIES: She was ill appearing, with tachycardia and fever. Skin exam was notable for livedo reticularis of the lower extremities and a painful, blanching, scattered papules on the dorsal aspect of the bilateral knees and forearms. She had brisk lower extremity reflexes with normal strength and sensation.

DIFFERENTIAL DIAGNOSIS: Initial diagnostic considerations were broad, including drug reaction, infectious or inflammatory process. Subacute endocarditis and coccidiomycosis were the primary infectious suspects. An autoimmune disease such as vasculitis was considered equally likely.

LAB AND IMAGING RESULTS: She had a leukocytosis of 11.5 with neutrophilic predominance. ESR was 65 with a CRP of 28. CK and aldolase were within normal limits. Extensive infectious testing was negative, including blood cultures, HIV and hepatitis panel. She had negative ANA and ANCA, RF and CCP. ECHO was unremarkable. Dermatopathology revealed a necrotizing vasculitis of a medium-sized vessel. There was neutrophilic infiltrate and C3 and IgM deposition in the vessel wall.

DISCUSSION: Ms. H was diagnosed with cutaneous polyarteritis nodosa (CPAN), a rare condition with unknown incidence and etiology. CPAN more often effects females and runs a chronic, relapsing but benign course. Common presenting findings are livedo reticularis, tender subcutaneous nodules or cutaneous ulcerations, with extra-cutaneous manifestations such as fever, myalgia, arthralgia and neuropathy. The diagnosis requires clinicopathologic correlation. Mild cases are treated with NSAIDS, escalating to corticosteroids and immunosuppressant drugs in those with more severe features. Our patient was started on IV then PO steroids and quickly defervesced with drastic improvement in symptoms.
CHIEF COMPLAINT: Abdominal pain

HISTORY: 61 yo male with a history of HTN, and T2DM presenting with abdominal pain, nausea and vomiting for past 3 weeks. Abdominal pain described as diffuse, dull and constant. No aggravating or alleviating factors. No association with food. Vomit bilious, no blood. Normal bowel movements, without blood or melena. He was admitted to the hospital 4 weeks prior with chest pain, underwent ACS rule out and eventually diagnosed with community acquired pneumonia, discharged on azithromycin.

PHYSICAL: Afebrile, BP 120s/70s, HR 80s, on RA | Stoic, sedated, easily arouses | CTAB | RRR no m/g/r | no edema | Diffuse abdominal tenderness, no rebound, guarding, distension or hepatosplenomegaly | Diaphoretic, no jaundice

INITIAL LAB RESULTS: WBC 25.7 (44% eosinophils), hgb 16.7, plt 134 | Na 129 Cr. 1.65, Ca 11.8 | AST 106, ALT 87, AP 390, TB 1.0, Prot 8.5, Alb 4.1

DDX: Presumed hypereosinophilic syndrome, likely due to eosinophilic leukemia. Differential included DRESS with recent azithromycin; other leukemia, lymphoma, or malignancy; autoimmune hepatitis.

CASE PRESENTATION: Admitted with hepatitis and peripheral eosinophilia. Further evaluation resulted in determining he had peripheral eosinophilia 4 weeks earlier (eosinophils of 27%) when he was diagnosed with CAP. At this point he was felt to have hypereosinophilic syndrome with multiorgan involvement. This prompted additional hematologic work up at same time of hepatitis work up. Hepatitis work up unrevealing. Peripheral blood smear resulted in hypereosinophilia, but normal eosinophils. CT abdomen showed liver changes consistent with acute hepatitis with enlarged gastrohepatic and retroperitoneal lymph nodes, felt to be reactive. Review of previous CT chest showed right pulmonary nodules which were felt to be infectious/inflammatory in nature. Bone marrow biopsy obtained with flow cytometry unrevealing for leukemia or lymphoma. Review of the bone marrow showed clusters of large, pleomorphic cells with high nuclear to cytoplasmic ratio and prominent nucleoli; consistent with a metastatic carcinoma. PET CT showed 3 right hypermetabolic lung nodules, widespread hypermetabolic lymph nodes in neck, chest and abdomen, diffusely enlarged and heterogeneously hypermetabolic liver (consistent with hepatitis), and diffusely hypermetabolic red marrow. Supraclavicular lymph node biopsied which revealed small cell carcinoma.

DISCUSSION: After diagnosis of small cell lung cancer patient was started on high dose steroids for eosinophilia and shortly afterwards chemotherapy. Unfortunately developed tumor lysis syndrome with multi-organ failure. Admitted to HICU requiring aggressive support. Died shortly later after cardiac arrest.
CHIEF COMPLAINT: Shortness of breath

CASE PRESENTATION: Patient is an otherwise healthy and active 71-year-old female diagnosed with breast cancer (Stage I, ER+, PR+, HER2+) a few months prior to presentation who was experiencing shortness of breath. She had undergone lumpectomy and started chemotherapy. Her treatment plan included combination paclitaxel and trastuzumab chemotherapy, followed by radiation therapy.

After eight weeks of chemotherapy, her major side effects were nausea and weakness. However, in the week prior to presentation, she had increasing shortness of breath with minimal exertion. Her symptoms were accompanied by cough and difficulty sleeping. One night, she awoke with shortness of breath, chest heaviness, wheezing, and coughing. This led her to seek further care.

PHYSICAL ABNORMALITIES: Vital signs were notable for new-onset hypoxia, (80% on room air), which improved with 3L nasal cannula. She appeared fatigued, but was breathing comfortably. She had crackles in her lung bases bilaterally, along with jugular venous distention. Her cardiovascular exam was otherwise normal.

LAB RESULTS: Initial blood counts and chemistries were unremarkable. Her troponin was negative and her B-type natriuretic peptide was 156.

DIFFERENTIAL DIAGNOSIS: The most concerning diagnosis was a pulmonary embolism given her active cancer history. The CT-PA was negative for a clot, but did show interstitial pulmonary edema. An echocardiogram then confirmed the diagnosis of new-onset systolic heart failure, with ejection fraction of 27%. This was in contrast to a relatively normal echo obtained two months prior. Then, CT coronary angiography was performed and was negative for coronary artery disease.

DISCUSSION: While the most likely cause of cardiomyopathy in general is ischemic disease from coronary artery disease, the most likely etiology of this patient’s symptoms was trastuzumab therapy. This agent is thought to decrease myocyte contractility, which can lead to an echocardiographic reduction in systolic function, but usually not clinical heart failure. This is in contrast to anthracycline chemotherapeutics, which lead to myocyte damage in a dose-dependent fashion, and often cause heart failure. Trastuzumab-related cardiotoxicity is usually reversible upon discontinuation of the drug, and can be re-challenged if needed.

CONCLUSION: Trastuzumab was temporarily discontinued, and she was started on ACE-inhibitor and beta-blocker therapy. While these are used in traditional systolic heart failure, data are not strong in this particular situation. However, the principles likely still apply. Patient’s symptoms improved and follow up echocardiogram is scheduled.
CHIEF COMPLAINT: 48 yo male with HTN, HLD, hypothyroidism presented to urgent care with acute onset R sided facial numbness, droop, and dysphagia.

HISTORY: For approximately 1 week he suffered cough, petechial hemorrhages on face, lethargy, and insomnia for which he was prescribed amoxicillin. Symptoms persisted until day of admission when he developed acute onset R sided facial numbness, droop, and dysphagia. Presented to urgent care and was noted to be markedly confused with global aphasia. Telesstroke was activated and patient was given NIHSS score of 5 (moderate stroke severity). CTA was negative for ischemia or hemorrhage. Prior to administering tPA, stat labs revealed platelets of 10. After repeating lab, it was decided tPA was contraindicated and patient was transferred to UofU for further treatment and evaluation.

On arrival to UofU, he was noted to have fever, AKI, anemia, mild leukopenia, and hyperbilirubinemia. MRI showed patchy irregularity bilaterally in putamen that was not contrast-enhancing and not consistent with a CVA. Regardless, patient was diagnosed with acute ischemic stroke and presumed hematologic malignancy and admitted to Neurology service.

PHYSICAL: Temp: 37-39 | Neuro: somnolent but opens eyes to voice, follows simple commands, global aphasia | CN: R hemiparesis and facial droop | DTR: normal

LABS: WBC 3.32 | Hemoglobin 10.2 (MCV 89) | Platelets 17 | Creatinine 1.4 | Bilirubin 2.4 (1.7 indirect) | LDH 800, Haptoglobin <10 | ADAMTS13 <5% | Peripheral Smear – schistocytes

TREATMENT COURSE: Patient had fluctuating neurologic symptoms with persistent encephalopathy. The next day, Hematology was consulted for possible malignancy, who noted patient had pentad of symptoms for TTP. He was urgently started on steroids and dialysis catheter was placed for plamapharesis. He responded rapidly to treatment. Unfortunately, patient had recurrence of disease requiring treatment with methylprednisone, vincristine, and rituximab. He improved and was discharged after 25-day hospital stay.

DISCUSSION/CONCLUSION:

Diagnosis: Thrombotic Thrombocytopenic Purpura - Acquired

Given acute onset of neurologic symptoms, patient was appropriately evaluated for stroke. However after this was ruled out with multiple scans, including perfusion imaging, a further differential should have been considered. Anchoring to a single diagnosis prevented prompt treatment for one of the few hematologic emergencies - TTP. Importantly hemiplegia, encephalopathy and aphasia are three of the most common neurologic manifestations of TTP (paresthesias and visual disturbances are seen as well). Patient presented with a perfect pentad (fever, AKI, microangiopathic hemolytic anemia, neurologic symptoms, and thrombocytopenia) which was confirmed with ADAMTS13. Treatment course was complicated and included corticosteroids, plasmapheresis, and chemotherapy. Splenectomy may be required for future recurrences, but currently he is asymptomatic without neurologic deficit. Etiology is unknown, but sinusitis is suspected as inciting trigger.
HISTORY: Patient is a 32 year old male with polycystic kidney disease in otherwise good health who presents with a one month history of fevers, chills and hemoptysis. He started with URI symptoms that progressed to night sweats, rigors, dyspnea, and productive cough with clear sputum. He later developed scant hemoptysis. He presented to an outside hospital 2 weeks ago and was found to have bilateral diffuse parenchymal opacities on chest CT. He was treated for community-acquired pneumonia with a 7 day course of levofloxacin.

Less than 2 weeks from discharge he presented to the University of Utah with fevers, dyspnea, hemoptysis and worsening bilateral ground-glass opacities on chest CT. Social history was notable for a recent move into a dilapidated house with significant rodent infestation. Multiple family members have sore throat, cough and dyspnea.

PHYSICAL ABNORMALITIES: Vitals significant for tachypnea, tachycardia, and temperature of 40.5C. Exam was notable for diffuse rales bilaterally without wheezes. No hepatosplenomegaly or sinus disease was appreciated.

DIAGNOSTIC STUDIES: Labs notable for WBC 6,000 with neutrophilic predominance and no eosinophilia. Hemoglobin nadired to 5.4. Platelets nadired to 115,000. Hantavirus serology was negative. Bronchoalveolar lavage showed increasingly sanguineous aspirate with each aliquot. Hemosiderin macrophages were found on cytology consistent with diffuse alveolar hemorrhage (DAH). ANA titer returned at 1:80. He also had a positive direct Coombs test and markedly elevated plasma hemoglobin of 28.8.

DIFFERENTIAL DIAGNOSIS: Initial differential for his DAH included infectious, inflammatory, and malignant causes. BAL was negative for viral, fungal or bacterial infections. Hantovirus pulmonary syndrome and pneumonic plaque were considered due to recent rodent exposure. With anemia on presentation, connective tissue disorders and malignancy were high on the differential.

DISCUSSION: After reviewing old records it was discovered he had a prior ANA titer of 1:2560 in 2010. Anti-Smith and anti-double stranded DNA were positive this admission confirming a diagnosis of lupus-associated DAH. Patient had been lost to follow-up in 2010 and his lupus went untreated for 4 years. To complicate the picture a bone marrow biopsy was performed and was consistent with a concurrent myelodysplastic syndrome.

CONCLUSION: Patient responded well to treatment with high-dose methylprednisolone and was discharged on prednisone and hydroxychloroquine. His anemia was likely a combination of hemolysis, MDS and DAH. His concurrent hematologic malignancy is still being worked up. A literature review suggests a possible link between SLE and MDS.
HISTORY: An 85-year-old Polynesian woman with a history of restless legs syndrome presented to the emergency department with 1 day of nausea, vomiting, and headache. Her chronic right leg pain, a dull constant ache, had been worse than usual for the past day as well.

PHYSICAL FINDINGS: Her blood pressure varied from 165/86 to 206/162. On examination, strength, sensation, range of motion, and pulses were preserved in both lower extremities, and they were without swelling or erythema. The examination was otherwise normal, without evidence of focal neurologic deficits. Initial lab workup including CBC, CMP, lipase, and troponin was unremarkable. DVT was ruled out with an ultrasound of the right leg, and CT brain did not show acute intracranial abnormalities.

COURSE: The patient was admitted and treated for hypertensive urgency with labetalol and hydralazine, followed by low dose amlodipine. With a negative CT brain, her headache was attributed to hypertension and treated with ketorolac. The cause for her nausea and vomiting was unclear, and was thought to possibly be related to hypertension or a separate gastroenterological illness. Her leg pain was thought to be an acute worsening of her chronic condition due to nausea and vomiting and was treated with pramipexole. Physical therapy was ordered to "evaluate and treat."

The following day the physical therapist reported that the patient had had significant problems with balance and would not put any weight on her right leg, during her evaluation. She also reported that the patient was not opening her right eye when out in the hallway.

DIFFERENTIAL DIAGNOSIS: posterior fossa ischemic stroke (not visualized adequately with initial CT), vertiginous disorder or osteomuscular abnormality. An MRI of the brain showed a small ischemic stroke involving the right lateral posterior medulla.

TREATMENT: A follow-up complete neurologic exam revealed right eye ptosis, right arm drift, ataxia of the right leg, and decreased pain sensation on the right side of her face. Neurology was consulted and recommended a statin, aspirin, amlodipine, a 30-day event monitor, and physical and occupational therapy.

CONCLUSION: Initial evaluation for cerebral ischemia with CT brain is often insufficient for the evaluation of ischemia to the lateral medulla and other areas supplied by the posterior circulation. Careful neurologic exams should guide further workup, including MRI, in patients with hypertensive urgency. Physical and occupational therapists are critical members of the assessment team, and frequently provide key diagnostic insights as a patient's clinical picture unfolds.
HISTORY: A twenty-year-old Mexican-American female presents to the emergency department with one hour of nausea, vomiting, and abdominal pain. Computed tomography of the abdomen and pelvis reveals a solitary 8 cm cystic mass in the right hepatic lobe. She is started on piperacillin-tazobactam for presumed sepsis from a pyogenic liver abscess and undergoes percutaneous drainage of the cyst by interventional radiology. Within hours of the procedure she develops diffuse hives, fever, tachycardia, and wheezing.

EXAM: She is not ill-appearing. Vital signs are notable only for pulse of 122. Sclera are anicteric. Her abdomen is obese and tender to palpation in the bilateral upper quadrants but without guarding, rebound, or evidence of ascites. She has no skin rash.

LABORATORY: CBC is notable for a WBC count of 18,710 with 77% neutrophils, 14% bands, and 0% eosinophils. Entamoeba histolytica IgG antibody, CA 19-9 and CEA antigens, and blood cultures are negative. Echinococcus antibody is equivocal.

DIFFERENTIAL: Pyogenic and amebic liver abscess are highest on the differential, but Echinococcal hydatid liver cyst, hemangioma, metastatic disease, and simple liver cyst are also considered. Microscopic examination of aspirated cyst fluid confirms the diagnosis of an Echinococcal hydatid liver cyst.

TREATMENT/COURSE: Over the initial few days of her hospitalization, she experiences repeated episodes of fever, tachycardia and wheezing, and develops a peripheral eosinophilia. Albendazole 400mg twice daily is initiated. Three weeks after her initial diagnosis, she is taken to the operating room where she undergoes aspiration and instillation of hypertonic saline into the cyst, followed by resection of segments five and six of her liver. Albendazole is continued for six-months. Serial ultrasounds repeated every three months show no evidence of cyst recurrence.

DISCUSSION: Echinococcal hydatid liver cysts are caused by the cestode parasite Echinococcus spp. Echinococcus granulosus and Echinococcus multilocularis account for the majority of human infections. Dogs are the definitive host for Echinococcus. Humans are incidental hosts, and infection begins following ingestion of eggs in dog feces.

If Echinococcus is on the differential for a liver cyst, a pathologist should be present during cyst aspiration to microscopically examine aspirated fluid for parasitic organisms. Positive microscopy necessitates instillation of hypertonic saline into the cyst prior to reaspiration, in order to kill the parasites and prevent abdominal spillage. Spillage can result in anaphylaxis and the need for surgical cystectomy, as occurred in this patient.
**CASE PRESENTATION:** Pt is a 72 year old male with a history of hypertension and numerous skin malignancies who presented with acute worsening of lethargy, fatigue, and intermittent confusion. Over the prior three months he noticed that he had become too tired to get out of bed and continue daily activities. Over the two weeks prior to presentation he didn’t have the energy to get out of bed and walk around the house. He had difficulty with recent memories and often felt confused when talking to friends. He was unable to walk even 50 yards when needed. He had no dyspnea. He did have a 15 pound weight loss over 2 months and history of night sweats that were a chronic issues. He also noted deep muscle and bone pain in his legs and arms for the week prior to presentation. He had significant worsening of bone pain over his spine for the past month and worsening of his chronic back pain. He had noticed a decrease in bowel movements as well over the past 2 weeks.

**PHYSICAL ABNORMALITIES:** Pt was fully oriented though diffusely weak. He has severe pain to deep palpation of his legs, arms, and lower back. He also had moderate tenderness to deep abdominal palpation with no palpable stool.

**LAB RESULTS:** Calcium of 12.8, iCa of 1.75, PTH: 5, 1,25 Vit D 188.0, SPEP/UPEP: negative, PTHrP: negative. Beta 2microglobulin: 4.8, TSH 4.12, flow cytometry: negative

**DIFFERENTIAL DIAGNOSIS:** On admission differential included primary parathyroid disease, malignancy associated hypercalcemia, endocrinopathy, and granulomatous disease.

**DISCUSSION:** This patient had an appropriately suppressed PTH with elevated calcium and concerning symptoms. His 1,25 Vit D level indicated an upregulation of 1 alpha hydroxylase activity. He had no symptoms of sarcoidosis, which is the primary etiology in this setting. He also did not have any risk factors for TB. Marrow biopsy revealed diffuse B cell lymphoma after a month of diagnostic workup that was stage IVB with diffuse retroperitoneal spread.

**CONCLUSION:** In patients with symptomatic hypercalcemia and suppressed PTH with significantly elevated 1,25 Vit D level an aggressive approach must be taken to look for lymphoma even without lymphadenopathy on exam or peripheral symptoms of disease. This process can be quite advanced without significant physical findings and only persistent hypercalcemia cardinal finding.
CASE PRESENTATION: Ms. S is a 19-year old woman who had been having 2 months of polyarthralgias, bloody diarrhea, and palpable purpuric rash. Biopsy of her rash demonstrated IgA and C3 deposition in the vessel wall so that she was diagnosed with HSP vasculitis. Her symptoms were refractory to prednisone taper. Therefore, she was prescribed 25 mg of dapsone TID and continued on steroids. 10 days later, she presented to the ER complaining of worsening pleuritic chest pain and dyspnea. On exam, her O2 sats were 89-91% on room air which did not improve with supplemental oxygen. Respiratory rate was 20. She had scleral icterus with cyanotic lips and nail beds. There were no crackles or wheezing on lung auscultation. CXR and CTPA were normal. Total bilirubin was 6.2 with 5.5 indirect and normal LFTs. ABG demonstrated a pH of 7.49, pCO2 of 30, pO2 of 138 on 4 L n/c, and MetHgb level of 11.9. Her MetHgb level increased on subsequent measurements to a peak of 17.8 on hospital day # 3. Methemoglobin reductase level was within normal limits.

DIFFERENTIAL DIAGNOSIS: Once ABG results were obtained, methemoglobinemia secondary to dapsone use was the suspected diagnosis. Inherited causes of methemoglobinemia were ruled out: HgbM disease when she responded to methemoglobinemia and cytochrome b5 reductase deficiency when methemoglobin reductase level was normal. Chest imaging ruled out pneumonia and PE.

TREATMENT: She was treated with 3 doses of methylene blue. After her last dose, her MetHbg level on ABG was noted to have dropped to 2.80 so that she was discharged on hospital day # 6. She returned 2 days later with recurrent dyspnea, pleuritic chest pain, and cyanosis despite reporting she had not taken any additional dapsone or other agent known to cause methemoglobinemia. MetHbg level had rebounded to 15.8. She was again treated with methylene blue after which her MetHgb level improved to 3.30, and her hypoxia and chest pain resolved.

DISCUSSION: Dapsone is known to be one of the most common causes of methemoglobinemia. Since dapsone undergoes enterohepatic recirculation, it has a long elimination half life of ~30 hours. Therefore, serial levels of methemoglobin must be followed and multiple doses of methylene blue may need to be administered. This case suggests that a normalizing levels of methemoglobin may not necessarily indicate that dapsone has been eliminated due to its unique metabolism.
CASE PRESENTATION: A 73 year old male with a number of well-controlled chronic diseases presented with anuric renal failure and anasarca. He was clinically stable until six months prior to presentation, when he developed progressive lower extremity edema and weight gain. Initial evaluation demonstrated a transudative pleural effusion, which became the focus of evaluation and treatment and ultimately required permanent pleurocentesis catheter placement. Repeated work-ups of his anasarca and pleural effusions, including echocardiography and bronchoscopy, were unremarkable. Pulmonary, cardiac, hepatic, and nephrotic processes were ruled out, with consultants commenting that his anasarca was “mysterious.”

One week following nephrology consultation, the patient went to a local ED for three days of anuria and swelling. He was hypotensive, and transferred to a tertiary center for further evaluation. On initial assessment, the patient did not have clear signs/symptoms of heart failure, and echocardiography was notable only for a dilated and non-collapsible IVC.

FINDINGS: The patient’s blood pressure continued to decline, ultimately requiring three vasopressors. To further evaluate his shock a pulmonary artery catheter was placed, revealing reduced cardiac output and elevated SVR, consistent with cardiogenic shock. Analysis of the hemodynamics also revealed equalization of pressures (CVP, PA diastolic, and PCWP), suggesting a constrictive or restrictive cardiac process. Simultaneous right and left heart catheterization was performed, demonstrating ambiguous results with features of both ventricular concordance and discordance. Cardiac MRI was then performed, revealing interventricular interdependence (diastolic septal bounce), a thickened pericardium with delayed enhancement, and positive myocardial tagging, all of which are consistent with constrictive pericarditis. Further work-up for infectious, toxic, and autoimmune etiologies was negative, and the patient’s history did not reveal a cause for constrictive pericarditis.

DISCUSSION: Determining the cause of shock can be difficult in the absence of clear signs and symptoms. In this case, invasive hemodynamic assessment was critical to making the diagnosis of cardiogenic shock. The equalization of filling pressures suggested a more specific etiology of cardiogenic shock - restrictive cardiomyopathy versus constrictive pericarditis. Delineating between these processes is paramount, given that idiopathic restrictive cardiomyopathy is difficult to treat and carries a reduced prognosis, while constrictive pericarditis can often be effectively treated with pericardiectomy.

In this case, determining between restrictive and constrictive etiologies was challenging, as initial diagnostic testing did not have definitive features of either. Cardiac MRI revealed three key findings, confirming the diagnosis of constrictive pericarditis. The patient underwent pericardiectomy to relieve cardiac constriction and is now doing well, with resolution of his anasarca and renal failure. We hypothesize that his decompensation represented an acute on chronic process, and that constrictive pericarditis was also the cause of his initial symptoms.
CASE PRESENTATION: MS is a 69 year old Argentinian female with a past medical history of Type 2 Diabetes, hypertension, and CAD s/p 5vCABG who presents with 4 days of nausea, vomiting and diarrhea. She lives in Washington state, and is traveling by RV through the Mountain West with her husband. She has been unable to take PO for the past 2 days due to nausea and vomiting. ROS is otherwise negative. This is her 3rd admission for similar symptoms in the past 6 months. She has traveled to the Dominican Republic and Mexico within the past 3 years. Denies recent international travel, food exposures, sick contacts, or antibiotic use. She is admitted for dehydration from presumed infectious diarrhea.

PERTINENT EXAM FINDINGS: On exam she is afebrile but hypotensive and tachycardic. Her abdomen non-distended and is not tender to palpation. The rest of her exam is unremarkable.

PERTINENT STUDIES: Initial laboratory evaluation revealed a creatinine 2.0, non-gap acidosis, Potassium 3.2, Calcium 12.9, and glucose 370. An infectious work-up including blood cultures, stool culture, ova and parasites, and E. coli shiga-like toxin are negative. Extensive review of outside records reveals a recent normal colonoscopy and a prior abdominal and pelvic CT scan with a 5.7 x 7.1 cm mass arising from the body of the pancreas. VIP level also found to have been elevated at 690 (normal <75) 2 months prior but never relayed to patient.

DIFFERENTIAL DIAGNOSIS: Previous differential of her pancreatic mass was a pancreatic non-functioning neuroendocrine tumor, and plan had been for surveillance.

TREATMENT AND COURSE: Patient is initially treated with aggressive IV fluids, electrolyte replacement, anti-emetics and anti-diarrheals but symptoms and electrolyte derangements persist. Once VIPoma diagnosed, she is initiated on Octreotide 50 mcg SQ TID, with complete and rapid resolution of her symptoms and lab abnormalities. Once stable, she is discharged and referred to a Surgical Oncologist in her home city for consideration of surgical resection.

CONCLUSION: VIPomas should be considered in the setting of refractory, watery diarrhea with hypokalemia and achloridia. More importantly, providers should be diligent in reviewing a patient’s previous records, even when a diagnosis seems apparent. Furthermore, unresulted labs upon admission should always have plans for follow-up, or a diagnosis may be missed.
INTRODUCTION: This is a 27yo Native American female with a PMH significant for lupus, antiphospholipid syndrome (APS), and seizures.

CASE PRESENTATION: The patient presented to an OSH with 3 days of melena, 3 days of pleuritic chest pain, and 1 day of hematemesis. She was recently told to hold her warfarin for an elevated INR. In the OSH ED she was noted to be hypotensive to 80mmHg systolic, have gastroccult positive emesis, and an elevated troponin. Given her complexity, she was transferred to our facility. We continued to hold her warfarin as there was ongoing concern for GI bleed because her hemoglobin dropped from 10 to 8 while at the OSH. There was also concern for ACS as her troponin peaked around 11. A CTA coronary was negative for atherosclerosis, but showed some apical akinesis as did a cardiac MRI. In the following days her creatinine rose to 1.6 from 1 on admission with nephrotic range proteinuria and platelets dropped from 138k to 50k. Renal biopsy was performed and showed thrombotic microangiopathy (TMA). Simultaneously, she developed a DVT in her right forearm and persistent headaches. An MRI revealed multifocal recent peripheral infarcts involving bilateral cerebral/cerebellar hemispheres.

NOTABLE LABS:
- B2GP 1, IgM Ab >150 (H)
- Cardiolipin Ab IgM 108 (H)
- dRVVT Confirmation Positive
- PTT-D Confirmation Positive

DIFFERENTIAL DIAGNOSIS: Before the diagnosis of TMA on renal biopsy, we considered an autoimmune hemolytic anemia as the patient had a positive Coombs test, but no schistocytes were seen on smear and haptoglobin was not significantly elevated. DIC was also considered, but workup was negative.

TREATMENT: Initially we tried high dose steroids and immunosuppression with mycophenolate and rituximab, but her platelets and kidney function continued to decline. We then proceeded with plasmapheresis (TPE) and a heparin drip bridge back to oral warfarin. The patient improved after 8 rounds of plasmapheresis with stabilization of her creatinine and recovery of her platelets.

DISCUSSION: This is a case of catastrophic antiphospholipid syndrome (CAPS), an acute and complex process that leads to occlusion of small vessels of various organs. While rare, mortality rates approach 50%. CAPS is defined as thromboses in three or more organs developing in less than a week. Our patient had kidney, brain, vascular, and probable cardiac and GI involvement. Treatment consists of anticoagulation, immunosuppression, and TPE if refractory.
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Questions? Contact Brittany Patterson | (801)635-6921 | brittany.patterson@hsc.utah.edu