



Utah
Chapter

2014

CLINICAL VIGNETTE COMPETITION

UTAH ACP RESIDENTS &
FELLOWS COMMITTEE

Scott C. Woller, MD FACP – Chair
Kencee Graves, MD

SPRING BANQUET & CLINICAL VIGNETTE PROGRAM THURSDAY MAY 22, 2014

University of Utah | Health Sciences Education Building (HSEB) | Room 1730

5:00 PM	WELCOME & OPENING REMARKS <i>Scott C. Woller, MD FACP</i>	JUDGES <i>Dustin Armstrong, MD</i> <i>John Nord, MD</i> <i>Beth Underwood, MD</i>	
5:15 PM	DINNER & SOCIAL		
6:00 PM	PRESENTATIONS		
	Fevers, Weight Loss, and Bumps – Do I Have Cancer, Doc? <i>Presented by: Judy Vu, MD</i>		Pg. 31
	Eosino-What? <i>Presented by: Steve O'Donnell, MD</i>		Pg. 18
	I Have Walked 10,000 Miles and I Will Walk 10,000 More <i>Presented by: Craig Robison, MD</i>		Pg. 26
	Don't Go Chasing Waterfalls <i>Presented by: Stacy Tanner, MD</i>		Pg. 30
	When Lightning Strikes Twice <i>Presented by: Christian Perez, MD</i>		Pg. 20
6:40 PM	ANNOUNCE RUNNERS-UP AND 1ST PLACE		
6:45 PM	CLOSING COMMENTS <i>Scott C. Woller, MD FACP</i>		

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.

- 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.
- 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.
- 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.
- Identify public policy concerns of residents.**
 - Monitor local and national health policy and how it relates to Internal Medicine and residency training.
- Encourage an interest in community service.**
 - Identify ways associates can become involved with community service in Utah.

CONTENTS

The Clue in the Murmur: Post-MI Complications Jason Allen, PGY-1	1
Recheck the potassium, it can't REALLY be that high Heather Balch, MD	2
Faint of Heart Wade Brown, MD	3
I'm Too Young For This Wade Brown, MD	4
A Lofty Diagnosis: Fever & Arthralgia with Hairy Cell Leukemia Meghan Cirulis, MS4	5
Small Fiber Neuropathy in a Patient with Urticarial Vasculitis Jessica Donigan, MD6	
Colonel Mustard, in the library, with the.....Augmentin? Jared Glenn, MS3	7
We better check a UA Marc Granata, MD	8
Human Metapneumovirus: More than Meets the Eye? Jeffrey Halleck, MD	9
Familiarity w/heparin can lead to a false sense of security Verena Haringer, MD	10
Not Your Average Back Pain Jess Harris, MD	11
Painless Jaundice in a Young Female Abdul Haseeb, MD & Sameerah Rehmani, MS	12
Try Some Ciprofloxacin Brad Henriksen, MD	13
Fungus Among Us Jason Jensen, BS & Matthew Feurer, MD	14
She's got a bilirubin that just won't quit! Vance Johnson, MD	15
Just A Little More Tired Than Normal Danette Ko, MD	16
A Rather Interesting Diagnostic Conundrum Lucas Lenci, MD	17
Eosino-WHAT? Steve O'Donnell, MD	18
Rabbits, Falcons and a Fever, Oh My! Andrew Orton, MD	19
When Lightning Strikes Twice Christian Perez, MD	20
Acute Illness in a Patient w/Long-standing Immunosuppression Laura Probst, MD	21
Löfgren's Syndrome Thomas Queen, MD	22
A Sweet Case of Fever and Rash Allen Rassa, MD	23
Huge Bladder Mass and a Huge White Count Joseph Redman, MS	24
Livers and Tumors and Refractory Ammonia, Oh My-eloma Joseph Redman, MS	25
I Have Walked 10,000 Miles and I Will Walk 10,000 More Craig Robison, MD	26
A High-Pressured Situation Natalie Rodden, MD	27
More Than a Fever Austin Schwab, MD	28
Don't Forget the Supplements Jennifer Springer, MD	29
Don't Go Chasing Waterfalls Stacy Tanner, MD	30
Fevers, Weight Loss, and Bumps – Do I have Cancer, Doc? Judy Vu, MD	31
My Headache is Gone; Why Can't I See? Judy Vu, MD	32
Not all that Swells is Arthritis Judy Vu, MD	33
First, do no harm Khine Win, MD	34

CASE PRESENTATION: Mr. S is a 63-year-old male with a history of hypertension, hyperlipidemia, and tobacco use who presented with acute onset of chest pain. Upon his initial presentation he was tachycardic, tachypneic, diaphoretic in notable distress. He was found to have a troponin of 1.09 and an EKG demonstrating ST elevations in II, III, and aVF, suggesting an inferior MI. He was taken to the cardiac catheterization laboratory and found to have a blockage of his distal RCA. A drug-eluting stent was placed and he was transferred to the medical floor for post-MI observation with medical therapy including aspirin, a statin, and Plavix. He was chest pain free after his procedure.

Around 7:00 AM the next day he awoke from sleep with some discomfort in his chest. He was notably tachycardic and tachypneic. He again demonstrated distress. He rapidly went from room air to up to 10L of oxygen by NC. Given his acute hypoxic respiratory failure, a stat ABG, CXR, troponin, and EKG were ordered.

PHYSICAL EXAMINATION: There were diffuse crackles in his lung bases and a loud holo-systolic murmur auscultated throughout the precordium. His breathing was labored. He was clammy to the touch. Breathing was labored. He spoke in short one-two word sentences.

DIFFERENTIAL DIAGNOSIS: Acute heart failure, re-infarction, ventricular septal defect, papillary muscle rupture, ventricular free wall rupture, pericarditis/Dressler's, cardiac arrhythmia, cardiac tamponade, shock

LABORATORY/IMAGING DATA: Troponin 1.09, EKG again with ST elevations in II, III, and aVF.

TREATMENT COURSE: Given EKG findings of new ST elevations despite prior days PCI, a STEMI was activated, he was given Lasix and taken to the catheterization laboratory. He continued to rapidly decompensate. Anesthesia was called for intubation and a PEA arrest occurred. He had a blood gas with profound hypoxia with pH <7 and echocardiogram demonstrating acute papillary muscle rupture resulting in severe mitral regurgitation. He was transferred to the SICU and CT surgery was consulted but given the severity of his illness, profound acidosis, interventions were felt to be without utility and he passed away a few hours later.

CONCLUSION: Despite increased efforts to increase time to tissue re-perfusion and decreased complication rates compared to decades ago, post-cardiac complications remain a possibility and should be considered in a patient with recent MI. Piecing together the clues from the history, physical examination and laboratory studies can help obtain a diagnosis and allow for appropriate interventions to take place.

INTRODUCTION: 18 year old female with complex medical history who developed hyperkalemia

CASE PRESENTATION: 18 year old female with a complex medical history of trisomy 21, congenital heart disease, consisting of AV canal defect, with multiple surgeries, including multiple shunts and stents, and a baseline oxygen saturation of 65% on room air, who was admitted with left sided weakness. She also has a history of hypercoagulability, with previous jugular venous thrombosis, and is on chronic anticoagulation with Coumadin and aspirin, though with erratic INR's. CT on admission showed left midbrain, and right cerebellar infarcts, suggesting a posterior circulation embolus. She was admitted, and was started on a heparin drip, this was continued for 3 days. She was then transitioned to lovenox. 3 days after transition she was found to have a potassium of 6.3 on routine lab work, with normal previous values.

PHYSICAL ABNORMALITIES AND LABS: Exam was notable for Down's facies, left sided facial droop, left arm and leg weakness, significant clubbing and cyanosis of fingers and toes. EKG did not show peaked T-waves. Labs showed: Na-133, K-6.3, HCO3-21, BUN-37, Creatinine-0.66. Aldosterone was normal, Transtubular potassium gradient was 5.1 (less than 5-7 suggestive of hypoaldosteronism).

DIFFERENTIAL DIAGNOSIS: The patient was a very difficult lab draw, so hemolysis initially thought to be the cause of her hyperkalemia. However, multiple repeat values remained elevated. The patient was on an ACE inhibitor and this was thought to be the next likely culprit, the ACEi was stopped and she was placed on a low potassium formula. Also, her BUN had increased, so her fluids were liberalized in case kidney injury was the etiology of high K (creatinine was stable). The potassium did not improve, further labs showed low TTKG, and inappropriately normal aldosterone, which suggested heparin induced hyperkalemia secondary to her lovenox as the most likely etiology.

DISCUSSION: Heparin induced hyperkalemia is thought to be secondary to a direct toxic effect on the zona glomerulosa cells of the adrenal glands, this appears to decrease the angiotension II receptors, leading to decreased aldosterone secretion. Heparin and LMWH have both been found to increase potassium levels. The hyperkalemia is reversible if the offending agent is stopped. However, if this is clinically impossible, then the treatment is fludricortisone, which was started in this patient and her hyperkalemia resolved.

CASE PRESENTATION: 34-year old male with a history of seizures who presented after two episodes of “fainting.” Patient was recently driving to a restaurant when he felt lightheaded, but the feeling resolved spontaneously. However, while at the restaurant, he lost consciousness. The morning of presentation, the patient was talking with a co-worker when he became lightheaded and lost consciousness. Patient reported a history of adult onset seizures for which he was previously treated with Keppra. However, he was taken off this medication about two years ago. Patient reported losing control of his bladder during the most recent episode. Observers reported that the patient was unconscious for 3-6 minutes and was snoring. No shaking, injuries, post-episode confusion, or loss of feces were noted. A brain MRI, EEG, TTE, and cardiac MRI revealed no abnormalities.

LABORATORY RESULTS: No abnormalities.

PERTINENT PHYSICAL EXAM FINDINGS: No abnormalities.

DIFFERENTIAL DIAGNOSIS: Nonsyncopal considerations included seizure disorder, conversion disorder, and narcolepsy. Syncopal considerations included reflex (orthostasis, vasovagal), cardiac (arrhythmia > valvulopathy/ACS), and neurologic (vertebrobasilar TIA, vertebrobasilar steal, ventricular colloid cysts) syncope.

TREATMENT: A careful history and an EKG were obtained. The patient was diagnosed with Brugada Syndrome (Type I). He was fitted with a LifeVest and discharged after scheduling an appointment for placement of an ICD.

CONCLUSION: The differential diagnosis for syncope is broad and includes the conditions listed above. A careful history is essential in evaluating syncope, and, in this case, revealed that the patient’s previous seizure episodes were distinct from his “fainting” spells (i.e., standing, stiffness, falling backwards, shaking for ~3 min, and ~30 minutes of subsequent confusion), and that nearly all of the male members of the patient’s family died of cardiac causes at early ages. Similarly, nearly all patients with syncope should have an EKG. In this case, careful history and pathognomonic EKG findings revealed the cause of this patient’s syncope. Brugada syndrome, a genetic channelopathy affecting myocytes, is an exceptionally rare cause of syncope. The prevalence of Brugada pattern on EKG is 0.012 – 0.4% in the United States, and the incidence of Brugada Syndrome (EKG findings + symptoms) is only ~ 4% year-1 in those with classic EKG findings. Because the syncope from Brugada Syndrome is most often the result of paroxysmal ventricular tachyarrhythmias

that can lead to sudden cardiac arrest, treatment for those affected is placement of an ICD.



CASE PRESENTATION: 28-year-old female with multiple sclerosis and epilepsy transferred from an outside hospital for chronic cough, progressive shortness of breath, lower extremity swelling, 20 pound weight gain, and a cardiac apical wall abnormality noted on TTE. On admission, patient confirmed progression of the above symptoms over the preceding seven months and also described orthopnea, PND, as well as SOB with even minimal activity. The patient reported that she felt these symptoms were the result of a new medication, Tecfidera, which she started around the same time as the onset of symptoms. Patient was admitted on no oxygen, but developed increasing O2 needs that improved with diuresis. Liver and renal function assessments were within normal limits. Patient underwent a TTE, a Cardiac MRI, and, eventually, an L/RHC with biopsy.

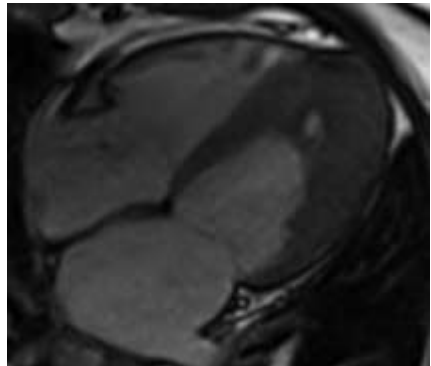
PHYSICAL EXAM ABNORMALITIES: O2 Sat of 91% on RA. JVP at 10-11 cm. Bilateral pulmonary crackles.

LABORATORY VALUES: BNP > 700. WBC of 10.62 with 7% eosinophils.

DIFFERENTIAL DIAGNOSIS: Myocarditis, non-compaction cardiomyopathy, apical thrombus, Löffler's endocarditis/hypereosinophilic cardiomyopathy, or endomyocardial fibrosis. Initial TTE revealed isolated apical hypokinesis and obliteration of the left ventricular by echodense material. Cardiac MRI revealed severe apical hypokinesis and endomyocardial disease with contained thrombus. Endomyocardial biopsy showed mild interstitial fibrosis.

TREATMENT: The patient was diuresed with loop diuretics and eventually started on a beta-blocker. Patient was also started on a prednisone taper and bridged to warfarin for her apical thrombus. A subsequent cardiac MRI showed improvement of LV function and apical hypokinesis, as well as reduction in the size of LV thrombus. Symptoms improved from NYHA IV to NYHA II.

CONCLUSION: Though initially thought to have endomyocardial fibrosis, subsequent dermatohistopathologic evaluation of the endomyocardial biopsies showed positive eosinophil granule protein staining (EMBP1 and EDN) suggestive of hypereosinophilic cardiomyopathy. In hypereosinophilic cardiomyopathy, hypereosinophilia leads to eosinophil penetration and degranulation with resultant acute necrosis, thrombosis, and eventual fibrosis. Fibrotic thickening of portions of the myocardium produces a picture similar to that found in endomyocardial fibrosis. Glucocorticoids are often highly effective in treating hypereosinophilia and resultant hypereosinophilic cardiomyopathy. As this patient was not at risk for helminthic infection, and does not have myeloproliferative hypereosinophilia (FIP1L1/PDGFRA negative), the relationships between phenytoin and/or tecfidera and hypereosinophilia are being investigated.



CASE DESCRIPTION: Mr. L is a 56 year-old man with a history of ulcerative colitis (UC) and remitted hairy cell leukemia who presented to an outside clinic after acute onset of low-grade fever and severe polyarticular, asymmetric arthralgias, predominantly affecting the right ankle. The patient was in recovery from recent cladribine chemotherapy and had required a red blood cell transfusion several days before symptom onset. Home treatment with high-dose acetaminophen had proven ineffective and he was unable to ambulate. Evaluation revealed infiltrates on chest x-ray and he was given levofloxacin and discharged home. His joint pain was not addressed at the visit and subsequently worsened, prompting admission.

Mr. L presented with a temperature of 39.3°C. He had right conjunctival erythema with blurred vision and pain. The pulmonary exam was unremarkable. Abdominal exam revealed new left-upper quadrant tenderness and previously noted splenomegaly. Range of motion in the hips, knees and ankles was limited by pain. Although Mr. L denied having a rash, several raised, tender and erythematous nodules were observed on the volar aspect of the bilateral forearms.

The patient had fever, increased splenic tenderness, bone pain and skin findings concerning for a vasculitic or infiltrative process—all described as symptoms of hairy cell leukemia. Disseminated tuberculosis and systemic fungal infections produce similar constitutional, lung and skin findings. Reactive arthritis was also considered in the setting of active ulcerative colitis and possible ophthalmologic involvement.

Laboratory evaluation demonstrated normocytic anemia with a white cell count of 2.97 k/ μ L. Erythrocyte sedimentation rate and C-reactive protein were elevated at 100 and 26.9, respectively. Creatinine kinase and urinalysis were normal and a bone-marrow biopsy disproved relapsed leukemia. Testing for gonorrhea and chlamydia, HLA-B27 and rheumatoid factor were unrevealing. Skin and blood tests for tuberculosis were negative. Repeat chest x-ray showed reticulonodular opacities in the apices and hilar adenopathy. X-ray of the right ankle was negative for effusion or erosion. Computed tomography (CT) of the chest revealed hilar and mediastinal lymphadenopathy. Skin biopsy demonstrated septal panniculitis.

The patient received high-dose ibuprofen and prednisone with prompt resolution of the arthralgias and skin findings.

CONCLUSION: Lofgren's syndrome is an uncommon but benign presentation of sarcoidosis. Diagnosis is clinical with a pathognomonic triad of symptoms: erythema nodosum, acute polyarthritis (predominantly ankle) and hilar adenopathy. Follow-up with bronchoscopy found CD4:CD8 ratio of 3.63, consistent with pulmonary sarcoidosis. CT chest at four months demonstrated resolution of adenopathy.

CASE PRESENTATION: 55-year-old female with a 3 year history of normocomplementemic urticarial vasculitis who began experiencing dysesthesias and numbness in her distal legs and arms, lips, and jawline over the course of 5 months. At the onset of the patient's neuropathy, she was taking dapsone; however, her neuropathy persisted despite discontinuing the medication. She had increased numbness and dysesthesias when her urticaria would flare, with improvement in the neuropathy when her wheals would clear. Past medical history was also significant for Hashimoto's thyroiditis and hypertension.

PHYSICAL EXAM ABNORMALITIES: Scattered wheals with an unremarkable neurological exam.

DIAGNOSTIC RESULTS: Laboratory testing was within normal limits with the exception of elevated anti-thyroid peroxidase antibodies. Skin biopsy was consistent with urticarial vasculitis. Sural nerve biopsy, electromyography, and nerve conduction studies were normal.

Punch biopsy specimens were later obtained from the left calf and thigh and sent for intraepidermal nerve fiber analysis. Both sites had decreased intraepidermal nerve fiber densities.

DIFFERENTIAL DIAGNOSIS: Diagnosis of small fiber neuropathy associated with urticarial vasculitis. Considered dapsone-related peripheral neuropathy; however, EMG and nerve conductions were normal. Vasculitis-induced nerve ischemia was also considered, but sural nerve biopsy did not show evidence of vasculitis or ischemic damage. Other etiologies considered included diabetes, alcohol use, and HIV infection, but were ruled-out with patient history and laboratory testing.

TREATMENT: Over the course of her disease, the patient had been on several medications to manage her urticarial vasculitis including high dose antihistamines, mycophenolate mofetil, methotrexate, colchicine, omalizumab, prednisone, and dapsone. She showed partial response to dapsone, but was unresponsive or intolerant to the other medications. Both the skin disease and neuropathy were eventually controlled on methotrexate and adalimumab.

DISCUSSION: Small fiber neuropathy (SFN) is both a sensory and autonomic neuropathy. Sensory symptoms most commonly have a length-dependent course and occur in a stocking-glove distribution. There are several causes of small fiber neuropathy with diabetes being the most common. Other causes include other metabolic diseases, drugs, infections, autoimmune diseases, and hereditary diseases. Routine nerve conduction studies and electromyography are normal in SFN because these studies assess large nerve fibers. Skin biopsy has become a major part of the diagnosis of SFN because it can quantify the density of intraepidermal nerve fibers.

CONCLUSION: This case is important and it is the first reported case of SFN in a patient with urticarial vasculitis. Additional studies are warranted to further evaluate a possible association of small fiber neuropathy and urticarial vasculitis.

CHIEF COMPLAINT: Cough

HISTORY: Patient is a 29 year old gentleman with 2 weeks of a mild cough accompanied by a small amount of white or yellow sputum production. 1 week prior to presentation he noticed that his face was slightly swollen which prompted him to visit his PCP where he was prescribed Augmentin. Several days later, he woke up with significantly increased amount of facial swelling with limited ability to open his mouth. He presented to the ED where he was treated for an allergic reaction to the Augmentin. On the day of presentation, our patient reported persistent swelling and 4 episodes of severe epistaxis which prompted his return to the ED. Review of systems confirmed recent headaches with localized pressure behind his eyes, chest discomfort, and mild redness and swelling of the face and neck.

PHYSICAL ABNORMALITIES: Physical examination revealed tachycardia to 120 bpm. Noticeable erythema and swelling of the neck and face with marked cyanosis of bilateral ears. His lungs were clear with no wheezes or crackles. There was no peripheral edema.

DIFFERENTIAL DIAGNOSIS: Superior vena cava syndrome, Bronchogenic adenocarcinoma, Lymphoma, Mediastinal mass, Lung cancer

IMAGING: CT chest: large, anterior mediastinal mass measuring 14.3 x 10.3 .11.5 cm with a large filling defect in the superior vena cava. CT of the neck with contrast: significant thrombosis of the superior vena cava and the left brachiocephalic vein.

LAB RESULTS: CBC: WBC 14.20, Hgb 15.1, Hct 43.9, Plts 425 | Chemistry: within normal limits | Alpha Fetoprotein tumor marker 496 | Beta hCG, serum quantization 794 | LDH 890

PATHOLOGY: FNA: mixed germ cell tumor composed largely of immature teratoma with focal regions suspicious for yolk sac tumor and embryonal carcinoma.

DISCUSSION: Our patient has superior vena cava syndrome secondary to venous thrombosis and a primary mixed germ cell tumor of the anterior mediastinum. Treated with bleomycin, etoposide, and cisplatin chemotherapy with future tumor resection.

A 2010 study showed that of 55 patients diagnosed with primary mediastinal germ cell tumor, 83% complained of vague symptoms such as cough, chest pain, or dyspnea, 14% of whom also had superior vena cava syndrome. The Median 5 year survival time for these patients was 87.9 months, with those receiving two of three treatment modalities (surgery and/or chemotherapy and/or radiation therapy) having the longest median 5 year survival rate of 118.3 months. Poor prognosis was associated with 2 or more metastatic sites, and non-seminomatous tumor histology.

CASE PRESENTATION: The patient is a 53 year-old man with a past medical history significant for type 2 diabetes mellitus, hypertension, and hyperlipidemia, who presented to the hospital ED with an approximately one week history of worsening dyspnea, palpitations with exertion, and generalized swelling. He reported inadvertently losing all of his home medications several weeks prior to presentation but did recently complete a course of amoxicillin for a “respiratory infection.” Additionally, the patient noted an isolated episode of “very bloody urine” four days prior to presentation. In the ED, the patient was noted to be significantly hypertensive with an initial blood pressure of 216/98. He received IV labetalol and furosemide and was admitted to the Medicine Night Float Service for further evaluation, diuresis, and blood pressure control.

PHYSICAL ABNORMALITIES: Initial physical exam per the resident’s H&P revealed “an obese man breathing comfortably in NAD, JVD to the mid neck bilaterally, minimally reduced breath sounds at the bases, and 2-3+ pitting edema in bilateral upper and lower extremities.”

Initial laboratory work up in the ED revealed a mild normocytic anemia with Hgb, Hct, and MCV of 11.1, 31.8, and 87.4, respectively. CMP revealed a total protein of 5.9 and albumin of 2.6, but was otherwise unremarkable with BUN, creatinine, and LFTs all WNL. INR and BNP were WNL and troponins were negative x2.

Initial EKG in the ED was unremarkable and a single view chest radiograph revealed no acute cardiopulmonary process; although, small bilateral effusions and mild bilateral compressive atelectasis were noted on follow up CTPA, which was negative for PE.

DIAGNOSIS AND CONCLUSION: The patient was admitted with hypertensive emergency and presumed heart failure. However, TTE revealed normal LVSF with an EF of ~55-70%. Urinalysis the following morning revealed 22 RBC/hpf and 300 protein. Follow up protein-to-creatinine ratio was 3.9, and the patient was ultimately diagnosed with the nephrotic syndrome with some nephritic features. Nephrology was consulted, work up for glomerulonephritis was negative and the patient ultimately underwent a kidney biopsy, which revealed diffuse diabetic glomerulosclerosis and changes consistent with hypertensive nephrosclerosis.

BRIEF DISCUSSION: The nephrotic syndrome is not altogether uncommon, and should always be considered in the differential for patients with generalized edema, especially those with risk factors, such as diabetes and hypertension. In such cases, a urinalysis can prove invaluable, as it can provide important clues that can help lead the clinician down the road to correct diagnosis.

CASE PRESENTATION: Patient is a 39-year old female with history of IV methamphetamine abuse and latent tuberculosis who presents with 3 days of fevers, rigors, myalgias, dyspnea, wheezing and nonproductive cough. Review of systems revealed one month of blurred vision. Initial exam revealed normal saturations on room air, respiratory distress with accessory muscle use, and diffuse wheezing. After admission her respiratory status worsened and she was transferred to the ICU for concern of inevitable respiratory failure. Her respiratory viral panel tested positive for human metapneumovirus (HMPV). In the ICU she received BiPAP and was started on IV methylprednisolone. On hospital day 4 she developed weakness in her distal lower extremities, complete numbness of her feet, saddle anesthesia, urinary/fecal incontinence, and low back pain. Three hours later her weakness spread proximally to involve her thighs and upper extremities

PHYSICAL ABNORMALITIES: On hospital day 4 she was found to have 2/5 strength with dorsiflexion and plantar flexion and 3+ patellar reflexes with crossed adductor response. Three hours later she was unable to lift her legs off the bed and exhibited 3/5 shoulder abduction and grip strength. Hoffman's sign was positive on the right. Sensation was decreased to pinprick below the C6 dermatome.

DIAGNOSTIC STUDIES: Extensive CSF analysis (including HMPV PCR) was initiated with all results negative for a potential etiology of her symptoms. MRI of the brain and spine were negative for any inflammatory, ischemic, or mechanical lesions.

DIFFERENTIAL DIAGNOSIS: Patient presented with new onset quadriparesis, hyperreflexia and distinct level of sensory loss consistent with a cervical myelopathy. Post-infectious transverse myelitis was the favored diagnosis given her concurrent HMPV infection and improvement of symptoms with steroids. Upper motor neuron findings made Guillain-Barre Syndrome less likely. Other potential etiologies included dural arteriovenous fistula, multiple sclerosis, neuromyelitis optica, Sjögren's syndrome, and paraneoplastic syndrome.

DISCUSSION: The most likely diagnosis was felt to be post-infectious transverse myelitis. Patient responded well to high dose methylprednisolone with improvement in her upper extremity strength and resolution of her hyperreflexia. By time of discharge she continued to have persisting foot drop requiring transfer to inpatient rehabilitation.

CONCLUSION: HMPV is in the family paramyxoviridae. Literature review revealed no case reports of HMPV infection causing transverse myelitis. There was one case report linking HMPV to encephalitis. Other paramyxoviruses (i.e. measles and mumps) are associated with myelopathy and thus this raises the question whether human metapneumovirus is an unrecognized cause of transverse myelitis.

HISTORY: A 78- year old man with a past medical history of BPH presented with productive cough, hematuria and generalized weakness for 3 weeks. He was admitted to the Medicine Team and treated with antibiotics for symptomatic UTI and CAP. Admission labs showed elevated troponin (1.4). The patient was loaded with aspirin but no further treatment was initiated since EKG showed no ST changes, and the patient denied chest pain. TTE showed mildly decreased LV systolic function with apical wall motion abnormalities. A heparin gtt was started the following day since troponins were uptrending (1.9). One hour after starting heparin the patient had an acute increase in his oxygen requirement.

PHYSICAL ABNORMALITIES: Vital signs showed a temperature of 102.3, HR 120/min, BP 150/80, RR 32, SpO2 98% on 15L O2/min via face mask. On physical examination marked expiratory wheezes, decreased airflow, and a generalized pruritic macular rash was noted.

LABORATORY RESULTS: CBC was notable for WBC of 11.85 with 72.8% Neutrophils, platelet count 116,000. Lactic acid was elevated to 3.6. ABG showed pH 7.366, pCO2 29.7, pO2 88.4, HCO3 16.6.

DIFFERENTIAL DIAGNOSIS: CXR showed no evidence of ARDS or pneumothorax. Troponins peaked at the time of the acute event (2.1). Repeat myocardial ischemia seemed unlikely since the patient continued to deny chest pain, EKG was without significant changes to prior and repeat TTE showed no new wall motion abnormalities.

HOSPITAL COURSE: With the suspicion for an anaphylactic reaction the patient received 1mg of epinephrin IV and was transferred to the MICU. His respiratory status quickly returned to baseline. Since the patient had a Wells Score of 6.0 and repeat TTE showed dilated right-sided chambers, severe pulmonary hypertension, and underfilled left-sided chambers, an argatroban gtt was initiated with suspicion for PE. This was discontinued after a negative V/Q scan. Presumed diagnosis was an anaphylactoid reaction to heparin given the time course of the acute event, the findings on physical exam, improvement of TTE findings on repeat study, and the resolution of symptoms shortly after administration of epinephrine. The patient was tested for HIT antibodies which were found to be weakly positive (0.419).

CONCLUSION: There is increasing evidence of anaphylactic and anaphylactoid reactions to heparin. Immediate-type reactions are mostly non-allergic reactions due to contaminants. Administration of IV heparin in patients with circulating HIT antibodies can provoke anaphylactoid reactions that present as 'pseudo-pulmonary embolism' with fever, hypertension and acute respiratory distress.

CHIEF COMPLAINT: “My back hurts”

HISTORY: A 24-year-old female with a history of IV drug abuse presented with 1 week of severe low back pain. She initially attributed this back pain to prolonged bending while painting her toenails, having had similar pains after an injury roughly 4 years ago. This pain, however, was worse in the left sacral area with radiation to the left leg. Approximately 5 days prior to admission, the pain was so severe that she began having difficulty walking. She also endorsed a productive cough for the same period of time, in addition to fevers and chills over the previous 2-3 days. Her last known IV heroin use was 6 days prior to presentation. On the day of admission, she had a fever of 39.3 and CXR findings suggestive of multifocal pneumonia obtained at an urgent care, whereafter she was instructed to present to the ER.

EXAM: Her exam was notable for tachycardia (130’s), tachypnea and hypoxia (91% on 2). Chest exam revealed a III/IV systolic murmur at the left lower sternal border and diffuse bilateral rhonchi. She had no focal tenderness over her back. She had back pain with passive elevation of the left leg. Multiple bruises were noted over her arms and legs .

LABS: CBC showed a leukocytosis with left shift. CTs of her chest and abdomen demonstrated bilateral airspace consolidation with central cavitations and multiple enhancing collections along the left psoas and iliacus muscles from L4 to the groin, consistent with abscesses. Transthoracic echocardiogram illustrated an echodensity on the tricuspid valve with severe tricuspid regurgitation.

DIFFERENTIAL DIAGNOSIS: The presumed diagnosis was Tricuspid valve endocarditis with septic pulmonary embolic and iliopsoas pyomyositis. Blood cultures persistently grew Methicillin sensitive Staph aureus.

TREATMENT: Intravenous Nafcillin was chosen as treatment, however while inpatient she injected heroin through her PICC line, compromising her plan. She was eventually discharged home on 6-week course of oral Rifampin and Linezolid.

DISCUSSION: The treatment of endocarditis in an IV drug abuser is a difficult ethical situation. The risk associated with long-term central access in this patient was identified when she accessed her PICC for Heroin use. Treatment of bacterial endocarditis with an oral regimen is suboptimal, however, there is little data that explores the efficacy of oral antibiotics to treat endocarditis.

IDENTIFICATION: 28-year-old female.

CHIEF COMPLAINT: “Painless jaundice in a young female”

HISTORY/CASE PRESENTATION: The patient presented with a one-week history of jaundice and pruritus. She had no significant past medical history or home medication use. She had a wisdom tooth extraction six weeks prior to the presentation and had completed a seven-day course of amoxicillin. She denied any oral contraceptive or herbal supplement use. She denied any recent illness, sick contacts, or travel. She also developed acute transient right arm numbness with spontaneous recovery approximately one day after presentation.

PHYSICAL EXAM/ LAB RESULTS: The patient was jaundiced on physical exam, but no other abnormalities were observed.

Initial labs showed elevation in total bilirubin 9.6(direct bilirubin 6.9), accompanied by elevations in AST 131, ALT 223, and Alkaline phosphatase 436. Acute viral hepatitis, iron/copper overload, and autoimmune hepatitis work-up were all negative. CA 19-9 levels were within normal limits. MRCP showed intrahepatic biliary dilatation predominantly in the left lobe, extensive retroperitoneal/mesenteric adenopathy, and an incidental finding of bibasilar focal opacities. CT of the chest was completed to further evaluate the incidental pulmonary opacities and was significant extensive subpleural and parenchymal nodules. MRI-brain was obtained to evaluate the right arm numbness and revealed a punctuate focus of high T2/FLAIR signal in the hand knob area of left precentral gyrus.

An ultrasound-guided liver biopsy showed cholestasis without malignant cells. CT-guided biopsy of the lung nodules revealed mucinous adenocarcinoma with TTF-1 and Napsin-A negativity. These features indicated a metastatic nature to these nodules from a likely gastrointestinal primary malignancy. CA-125 was elevated. Subsequent ERCP revealed a malignant appearing stricture at the bifurcation of the hepatic ducts with ductal dilation. Stents were placed in the pancreatic and bilateral hepatic ducts.

DISCUSSION: The differential diagnosis of jaundice is large. Neoplasms should be considered after the usual viral infections, toxic exposures, alcohol, and inherited disorders are disqualified by history and pertinent labs. In our patient, the presence of asymptomatic lung nodules and neurological symptoms initially led us to the possibility of atypical sarcoidosis. However, the lung nodule biopsy revealed adenocarcinoma. This eventually prompted an ERCP, which led to the discovery of cholangiocarcinoma at the bifurcation of the hepatic ducts. This tumor is also known as Klatskins tumor.

CONCLUSION: The incidence of intrahepatic cholangiocarcinoma is increasing worldwide, Cholangiocarcinoma is a devastating malignancy with a delayed presentation, is notoriously difficult to diagnose, and is associated with a high mortality. A diagnosis of intrahepatic cholangiocarcinoma should be actively considered in patients with a lung or liver mass histologically confirmed to be an adenocarcinoma without an obvious primary source.

PRESENTATION: Patient is a 52-year-old male with recent history of subdural hematoma with neurosurgical resection three weeks prior to developing suprapubic abdominal pain radiating to his back. At an outside clinic the patient was diagnosed with prostatitis and started on ciprofloxacin. The patient's pain progressively worsened and spread to his left leg over the following 4 days until he came to the ED for further evaluation.

The patient had a history of provoked DVT in his left leg and had been on warfarin prior to his recent admission for subdural hematoma when therapy was discontinued for concerns of intracranial hemorrhage. When the patient later presented to the ED with suprapubic and left leg pain, CT angiogram abdomen pelvis showed thrombus extending from IVC filter (placed during previous admission) to iliac and femoral veins. The patient was started on a heparin drip overnight per VTE protocol.

PHYSICAL ABNORMALITIES AND LABS: Physical exam was notable for subprapubic tenderness, diminished pulses, edema, and decreased temperature in bilateral lower extremities. Platelet count was 54,000 at admit and decreased to 27,000 the following morning. UA and micro did not show evidence of urinary tract infection, and pelvic imaging did not show evidence of prostatitis, thus ciprofloxacin was discontinued.

DIFFERENTIAL DIAGNOSIS: The presumed diagnosis at the time of admission was repeat provoked VTE secondary to immobilization. Given the patient's drop in platelet count by 50% after initiation of heparin therapy, other diagnoses including DIC and heparin induced thrombocytopenia (HIT) were considered and the thrombosis service was consulted.

CASE PRESENTATION: With a careful review it was determined that the patient had received 11 doses of heparin during his previous admission. The patient's 4 T's score was 7, indicating a high pretest probability for HIT: platelet decrease 30-50 percent at <1 day with heparin exposure in the previous 30 days, confirmed new thrombosis, and no other causes for thrombocytopenia apparent. HIT ELISA and serotonin release assay confirmed the diagnosis. Heparin was stopped and the patient was started on argatroban drip and later changed to warfarin with fondaparinux bridging.

DISCUSSION: HIT has high morbidity and mortality, thus prompt recognition treatment is critical. As in this case, previous heparin exposure may not be initially evident, but a significant decrease in serum platelets, especially with new thrombosis, should prompt exploration for previous heparin exposures.

CHIEF COMPLAINT: “Sores on my face”

CASE PRESENTATION: Patient is a 31 yr old male who presented to clinic with a large skin lesion on his right cheek. This lesion was first noticed one year ago; additional lesions subsequently developed on his left temple and sacrum. The lesions initially presented as non-painful boils that would rupture then crust over. 10 months prior to these lesions he was hospitalized for unspecified pneumonia. Social history was significant for 10 different sexual partners over the past year. Patient was previously told his findings were consistent with HIV infection.

PHYSICAL EXAM ABNORMALITIES: Raised fungating 4cm lesion on right cheek with an area of central depression/necrosis and serosanguinous fluid drainage. A similar lesion is on the left temple. There is a 2cm lesion on his sacrum. On his left hand he had a 1cm scaling plaque and a similar lesion on his left foot. Lungs were clear to auscultation bilaterally. Cryptococcal, histoplasma, and blastomycosal antigen testing were all negative. A punch biopsy of the sacral lesion was consistent with blastomycosis. HIV testing was negative. Chest CT showed minimal cicatrization atelectasis consistent with a fungal pneumonia. The overall presentation led us to treat for blastomycosis.

TREATMENT: He was started on Amphotericin B IV therapy and transitioned to itraconazole PO. He initially did well, but was readmitted to the hospital after 1 month due to medication induced rhabdomyolysis.

DISCUSSION: Blastomycosis infection is commonly found in the great lakes region of the US, but is still relatively rare in immunocompetent hosts. Lung involvement is seen in up to 91% of cases and is often misdiagnosed as community acquired pneumonia. Additionally, accurate diagnosis is often delayed after multiple rounds of antibiotics. Skin involvement is seen in approximately 18% of cases, however disseminated disease is only typically seen in immunocompromised hosts. Our patient had severely disseminated disease with lung involvement and 5 separate sites of skin involvement, although all testing and history found him to have a fully intact immune system.

CONCLUSION: Although blastomycosis is atypical in the western United States, and disseminated blastomycosis is incredibly rare in an immunocompetent host, it is still an important disease to keep on a differential list when considering an abnormal presentation of pneumonia and/or fungating skin lesions.

IDENTIFICATION: 79 year old female presenting from an outside hospital with a PMHx of asthma, Crohn's Disease, 30 pk/yr smoking history, spasmodic dysphonia, hypertension and a distant history of treated non-Hodgkin's Lymphoma s/p full treatment with chemotherapy and radiation.

CASE PRESENTATION AND DIAGNOSTIC COURSE: The patient presented with symptoms of nausea and vomiting for 3-5 days and an OSH CXR with consolidation. At the time of admission her presenting symptoms were overshadowed by an oxygen requirement of 4L by NC and wheezing and ronchi by physical examination. Further tests revealed that the patient had an elevated white count and positive corona virus by Respiratory Film Array by PCR with a mildly elevated bilirubin all thought to be a part of the patient's septic picture. She was treated presumptively with antibiotics for CAP and possible aspiration pneumonia secondary to recurrent vomiting from a contaminant GI viral process.

Over the next few days the patient improved gradually and a white count that peaked at 18.3 and then declined were reassuring that the patient's infection was resolving. However, follow-up evaluation of the patient's bilirubin was different. Only 72 hours after admission it has climbed to be 3.8 (3.2 conjugated). This eventually peaked at 4.3 and was combined with an alkaline phosphatase, which rose to 736. This combined with nightly fevers despite continued antibiotic therapy.

An investigation of the patient's biliary anatomy by U/S revealed acalculous cholecystitis without biliary dilatation and limited evidence of acute cholecystitis without abdominal pain. Our medical team then turned to a CT Abdomen and Pelvis, which showed no acute abdominal process, and a morphologically normal liver; however, a large right-sided pleural effusion was found at this time. This was tapped and sent for cytology and chemical analysis showing transudative characteristics by Light's Criteria and containing 220 WBC/uL (94% lymphocytes).

Next an MRCP was obtained which showed "hepatomegaly with left hepatic and caudate lobe hypertrophy" with "a few mildly prominent porta hepatitis nodes." The result of this test came back at the same time the results of the flow cytometry of the pleural effusion. The lymphocytes were predominately B-cells, whose "immunophenotype is compatible with non-Hodgkin lymphoma." Further Hepatology and Heme/Onc consultations revealed that the patient's NHL had returned.

CONCLUSION: Lymphoma is known as "the great mimicker", able to present in a variety of ways under a variety of circumstances. This patient's presentation, while containing infectious elements, was an example of one of the many insidious and surreptitious ways in which lymphoma can present.

CHIEF COMPLAINT: Fatigue, fever

HISTORY: The patient is a 36 year old female, with no significant medical history, who had presented with complaints of fatigue for the past two days. She went to an Instacare after she developed fever and chills. She was noted to have an external hemorrhoid. It was presumed that this was the source of her infection and she was prescribed Keflex and discharged home. Later in the day, she was called and told to report to the nearest ED due to abnormal laboratory results, notably a low leukocyte count. Of note, she reported that her past two menstrual cycles have been heavier than normal. She denied any other systemic symptoms such as chest pain, shortness of breath, easy bruising, bleeding elsewhere.

PHYSICAL ABNORMALITIES: Vitals and physical exam were normal except for an external hemorrhoid.

LAB RESULTS: CBC showed pancytopenia. WBC was 0.51, Hb 9.8, Hct 27.2 and platelets 36. Chemistry panel was unremarkable. PTT, PT, fibrinogen were normal. D-dimer was elevated at 10. Peripheral smear showed pancytopenia. Preliminary bone marrow aspirate showed 47% atypical promyelocytes by morphology.

DIFFERENTIAL DIAGNOSIS: Acute promyelocyte leukemia (APL), acute myeloid leukemia (AML), viral infection, autoimmune disorder, myelodysplastic disorder.

DISCUSSION: The patient was empirically treated with ATRA while awaiting final results from the bone marrow biopsy. Bone marrow biopsy showed 45% promyelocytes by flow cytometry. T(15:17) (q22;q12) PML/RAR translocation was detected by FISH. EKG showed a prolonged QRS but echocardiogram was normal. Upon confirmation of the diagnosis of APL, the patient received induction chemotherapy with ATRA and idarubicin. Although arsenic has a better side effect profile, it can cause QT prolongation. Therefore, arsenic was not used due to her prolonged QRS at baseline. She was also started on viral and fungal prophylaxis.

CONCLUSION: Acute promyelocytic leukemia is a specific variant of acute myeloid leukemia (AML). The classic translocation seen is T(15:17) involving the promyelocytic leukemia gene (PML) on chromosome 15 and the retinoic acid receptor-alpha gene on chromosome 17 (RARA). This gene fusion blocks the differentiation of granulocytes in the promyelocyte stage. In patients with suspected APL, empiric treatment with all-trans-retinoid acid (ATRA) should be started. ATRA is unique as chemotherapy drug as it does not directly kill malignant cells. Rather it induces promyelocytic blasts to differentiate into mature neutrophils.

CASE PRESENTATION: 33 yo male of mixed Korean/Italian origin presents with complaints of genital ulcers, perianal fissures, and pain/swelling of left ankle/knee/hands. The patient's initial symptoms started in 2011 when he was having bloody diarrhea and persistent abdominal pain. He had a colonoscopy and biopsy and was diagnosed with Ulcerative Colitis started on prednisone, but then developed some issues with his vision and this was discontinued. Due to recurrent diarrhea, perianal skin tags and fissures Biopsy was done again and diagnosis was changed to Chrons.

Since then, he has had diarrhea which is still occasionally bloody. He has also had significant perirectal pain with bowel movements over last month. Of note, he also had intermittent oral ulcers, blurry vision, and sore throat. He also has noticed a genital ulcer for the last 9 months, which is painful. He also had swelling of his joints, and pain with joint movements of the hands and knees as well as generalized muscle weakness and fatigue over the last month. No F/N/C/Cough/CP/SOB/HA.

PHYSICAL EXAM ABNORMALITIES: Notable for swollen left foot and ankle without erythema, ttp and pain with movement. Swelling of his left index and 5th finger around MCP and DIP joints. 2 aphthous ulcers in soft palate. Genital ulcer on shaft of penis, also an area of eschar which was reportedly pustular in nature initially. Perianal fissure and skin tags. Positive pathology.

DIFFERENTIAL DIAGNOSIS: Chrons flare with extra-intestinal manifestations vs. Behcets disease vs infectious colitis vs. PAN vs. RA.

+ labs: ANA, skin pathology, fecal WBC's, ESR 90, endoscopy c/w diffuse colitis and deep ulcers

- labs: HLA B27, HIV, cANCA/pANCA, Hep panel negative, HSV, CMV, C. Diff negative, CK wnl, uric acid wnl, CBC, BMP largely wnl, G/C PCR.

TREATMENT: After consultations with DERM, OPHTHO, GI, RHEUM, we started prednisone for acute inflammation with transition to Remicade in the future.

CONCLUSION: What a fascinating case. It helped me review unusual vasculitides, Behcets disease, as well as the fascinating topic of extra-intestinal manifestations of IBD. Overall, it was felt that despite meeting the criteria for Behcets disease, the patient was likely presenting with extra-intestinal manifestations of IBD supported by positive path report. This case proved that diagnosis can be difficult and that patients never present according to the textbook. Thankfully treatment for both diseases included immunosuppression, but learning to coordinate care, understand treatment decisions in complex situations was invaluable especially early in my medical career.

CASE PRESENTATION: A 55 year old gentleman presents to the Emergency Department with two days of fever, sore throat, shortness of breath, and dry cough. Past medical history is significant for 35 pack years of smoking, recurrent sinus infections, and allergic rhinitis. His temperature is 38.3o Celsius, Chest X-Ray is unimpressive, and WBC is 23,000 with 34.8% eosinophils on differential. He receives 2 grams of ceftriaxone and is discharged home with a prescription for amoxicillin and a presumptive diagnosis of an upper respiratory infection.

Three days later he returns to the ED. All of his previous symptoms have persisted. For the past day, however, a new rash has developed over his knees and feet that is itchy and painful to the touch.

A Chest CT Angiogram is negative for PE but shows three pulmonary nodules in the right lung that are consistent with granulomas vs. metastatic disease.

PHYSICAL EXAM ABNORMALITIES: Confluent petechiae over the lateral surfaces of both knees, tender to the touch. Petechiae on the dorsal and lateral surfaces of both feet as well as palpable purpura.

PERTINENT LABS: CRP 11.6, ESR 41. The following are negative: Hep B and C, ANA, C3, C4, HIV, EBV, ANCA

SURGICAL PATHOLOGY: A skin biopsy shows dense dermal inflammation with prominent eosinophils and neutrophils with areas of leukocytoclasia and vascular damage.

DIFFERENTIAL DIAGNOSIS: Initially, DRESS or hypersensitivity vasculitis was considered since his rash developed after receiving antibiotics. His eosinophilia, SOB, allergic rhinitis, and fevers, however, were present prior to antibiotic administration. Palpable purpura and skin biopsy revealing eosinophilia and vascular damage combined with pulmonary nodules on CT led us toward a diagnosis of Churg-Strauss syndrome (also known as Eosinophilic Granulomatosis with Polyangiitis) despite negative ANCA's.

TREATMENT: A lung biopsy is planned in the near future for confirmatory diagnosis. He was started on prednisone 40 mg qday with concomitant alendronate, oral PPI, vitamin D, and calcium.

DISCUSSION: Present in 40% to 60% of cases, a positive ANCA is not required to diagnose Churg-Strauss. This patient met four of six diagnostic criteria outlined by the American College of Rheumatology (allergic rhinitis, positive biopsy, eosinophilia, pulmonary nodules). Until the use of glucocorticoids, Churg-Strauss was uniformly fatal. The syndrome can involve any organ system. Immunosuppressants such as cyclophosphamide and aziothioprine are also routinely utilized to achieve remission. Emerging evidence shows a role for monoclonal antibodies and rituximab is being considered for this patient.

CHIEF COMPLAINT: “I keep getting fevers”

HISTORY OF PRESENT ILLNESS: Mr. W. was a 47 year old gentleman with end stage renal disease on dialysis for >10 years who complained of persistent fever. He was from a small community in Montana where he practiced Hawking and falconry. Two months earlier he had been training a new hawk to hunt; after catching a small jack rabbit, the bird had landed on his ungloved hand, sinking its talon through the animal, and into his knuckle. This incident preceded his fevers by 1 week, and his symptoms had since evolved to include cough, painful lymphadenopathy of his arm, and right upper quadrant belly pain. He also noted a 10 lb unintentional weight loss.

PHYSICAL EXAM: Mr. W. appeared tired, thin and cachectic. There were crackles in the base of his right lung, and he was tender in his right upper quadrant. He showed us a small black eschar over the PIP joint of his right first digit where his hawk had sunk its talon. There were tender swollen lymph node chains rising along his right arm and into his right axilla.

LAB AND IMAGING RESULTS: A CT scan of the belly ruled out cholecystitis and abscess. A CTPA ruled out PE, but showed a large, dense irregular infiltrate of the right lower lobe and marked cervical, axillary and brachial lymphadenopathy in his right arm. Serologies for Yersinia, Brucella, and Tuleremia were negative.

DIFFERENTIAL DIAGNOSIS: Malignancy, dialysis-related infection and zoonosis including Tuleremia were all considered. A biopsy of the lung mass showed granulomatous inflammation without organisms or dysplasia, most consistent with pulmonary tuleremia. Repeat serology of F. Tulerensis returned positive.

CONCLUSION: Tuleremia, known colloquially as “Rabbit Fever” or “Pahvant Valley Plague” is a rare but serious infection harbored in animal reservoirs. The clinical syndrome depends on the mode of infection, with inhalation of the organism resulting in lung disease, ingestion resulting in diarrhea and direct inoculation causing local infection. Mr. W. had an unusual case of direct inoculation leading to lymphocutaneous spread progressing to pulmonary infection, likely due to underlying immunodeficiency due to long standing dialysis. Since the Department of Homeland Security lists Tuleremia as a potential bioterrorism weapon, we should all have a cursory familiarity of this disease and its presentation. Mr. W. Did very well on a course of PO Levofloxacin.

CLINICAL CASE: A 27 year-old woman had a four-year history of paroxysmal hypertension. Workup of her postpartum labile hypertension showed elevated plasma normetanepherine (19.3 nmol/L; nl <.89) and 24 hr urinary metanepherines and normetanepherines (2911 μ /dl and 40826 μ g/dl respectively; nl <350 and <650). MRI showed a large adrenal mass. Over an eight week period following delivery, the patient developed hypokalemia, moon facies, buffalo hump, altered mental status, hyperglycemia, and right upper lobe pneumonia due to *M. Catarrhalis*. 120 mEq KCl supplementation per day was needed to maintain her serum K >3.2 mmol/L. 24 hour urinary cortisol was 275 μ g/dl. ACTH rose from 316 pg/ml to 605 pg/ml over a 3-day period. Imaging of the pituitary, chest, and pelvis was normal. CT of the abdomen showed a hypodense intrahepatic lesion interpreted as metastatic pheochromocytoma.

The patient's pneumonia responded to ceftriaxone; blood pressure was controlled with alpha and beta blockade; hypercortisolism was treated with mifepristone and spironolactone. The patient underwent a right adrenalectomy and liver biopsy of the lesion identified on CT. Intraoperative pathology of the adrenal tumor was consistent with a pheochromocytoma; biopsy of the liver CT lesion showed no malignant cells. There was no evidence of retroperitoneal or abdominal metastases. Examination of stained tissue confirmed the intraoperative diagnoses. Immunohistochemical staining for ACTH identified ACTH stain positive cells distributed throughout the adrenal tumor. RET mutations weren't present.

The patient's hypertension, hypokalemia, and altered mental status remitted in the immediate postoperative period. 8 am ACTH and cortisol levels on postoperative day 1, while the patient was receiving dexamethasone 2 mg iv q6h, were <5 pg/ml and 2.8 μ g/dl respectively. The patient was discharged home on the second postoperative day feeling "the best I've felt in four years".

CONCLUSION: This is the first case of a pheochromocytoma present prior to and throughout pregnancy with the new onset of Cushing's syndrome following delivery in which both the fetus and mother survived. Although the patient had symptoms consistent with a pheochromocytoma for four years, hypercortisolism appeared following delivery and had an aggressive course resulting in opportunistic infection, encephalopathy, and life-threatening hypokalemia. Through a multidisciplinary approach between medicine, endocrine, surgery, and anesthesia, the patient recovered. Finally, this case also highlights that reliance on diagnostic parsimony doesn't always lead to a diagnosis as the assumption that the simplest solution is the most likely solution can lead the clinician astray.

CASE REPORT: A 52 year-old white man with a history of psoriatic arthritis presented with complaints of fatigue, non-productive cough and dyspnea on exertion for two months. He received two courses of empiric antibiotics without improvement. He then developed rapid swelling of the neck along with drenching night sweats over a 2-week period. No loss of appetite or weight loss was reported. There was no history of travel or sick contacts. Notably, the patient was on chronic immunosuppressive therapy with weekly oral methotrexate with excellent control of his inflammatory arthritis. He had previously been treated with anti-TNF based therapy (infliximab) between 2007 and 2008.

OBJECTIVE FINDINGS: Patient was febrile with temperature of 38.8 C, tachycardic and tachypneic. Physical examination revealed diffuse, symmetric, non-tender cervical, axillary and inguinal lymphadenopathy. There was no clinical evidence of hepatosplenomegaly. Laboratory studies showed normal white blood cell count and differential, mild macrocytic anemia and normal platelet count. Liver function tests and renal function tests were normal. CT of the chest, abdomen and pelvis revealed mediastinal, hilar, abdominal, and pelvic lymphadenopathy, bilateral pulmonary nodules and mild splenomegaly.

DISCUSSION: Diffuse lymphadenopathy in the presence of fever and night sweats is highly indicative of a lymphoproliferative disorder. Notably, chronic immunosuppressive therapy and prior treatment with anti-TNF based therapy are known to increase this risk. Viral, fungal and mycobacterial infections, particularly reactivation of tuberculosis should also be considered. In this patient, excisional biopsy of the right cervical lymph node showed infiltration with small, polymorphic lymphocytes that stained positive for CD-20 and EBV. There was no definitive co-expression of BCL-2, CD-5, CD-10 or CD-43. Proliferation index was approximately 70%. Cyclin D1 was negative. The findings were suggestive of an iatrogenic immunodeficiency-associated lymphoproliferative disorder, polymorphic type. Serum EBV was also detected and quantified at 46,000 copies. Patient improved significantly with near complete resolution of lymphadenopathy and B-symptoms simply by removing the offending agent.

CONCLUSION: Methotrexate-induced lymphoproliferative disorders have been described in the literature and typically are polymorphic processes driven by disseminated EBV infection. Most patients respond to discontinuation of methotrexate due to the return of cell-mediated immunity. CD-20 monoclonal antibody treatment is effective in patients who fail to respond to discontinuation of immunosuppressive therapy alone. This case highlights the clinical challenges in diagnosing disorders that can affect an immunocompromised host.

CASE PRESENTATION: A 56 year old female with a five year history of pulmonary sarcoidosis presented to the ER complaining of painful leg skin lesions. She reported that she first noticed some right thumb pain five days prior, but did not recall any trauma. The next day she developed pain in her right shin then her left shin. The pain continued to worsen and on the day of presentation she noticed one red skin lesion on her right leg and multiple lesions on her left leg. She complained of subjective fevers. She went to her PCP on the day of presentation, he was concerned that she could possibly have sepsis from endocarditis and referred her to the ED. Her sarcoidosis was treated with Prednisone, Cytoxan, Remicade and CellCept. It has been noted that patient has had some cytopenias related to these medications. In the ER, her temperature was 38.7 C, HR 110 bpm, and had a WBC count of 1.5 K/uL.

PHYSICAL EXAM ABNORMALITIES: Exam was notable for multiple violaceous, exquisitely tender to palpation subcutaneous nodules on her bilateral lower extremities from proximal thighs to distal shins, 2-10 cm in size. They were warm to the touch. She had some tenderness to palpation at the base of the right first metacarpal joint but no skin findings on right hand or thumb. No ulcerations or erosions seen on exam. Cardiac auscultation was WNL with no murmurs.

DIFFERENTIAL DIAGNOSIS: DDx included erythema nodosum versus nodular vasculitis versus erythema induratum versus polyarteritis nodosa versus erysipelas versus lymphoma versus deep fungal infection. Endocarditis was thought to be less likely.

HOSPITAL COURSE: Endocarditis was ruled out by TTE on admission; lower extremities were WNL on Doppler US. CXR was WNL. Dermatology was consulted, skin biopsy was performed. It was believed the patient had Löfgren's syndrome with skin findings consistent with erythema nodosum. She remained hemodynamically stable and was sent home on a high dose prednisone taper. Per chart review, biopsy results were consistent with erythema nodosum.

CONCLUSION: Löfgren's syndrome is a subtype of acute sarcoidosis characterized by hilar lymphadenopathy, erythema nodosum, arthritis and fever. This patient presented with erythema nodosum, fevers and arthritis. Although hilar lymphadenopathy was not seen on CXR, she does have a history of pulmonary sarcoidosis. Löfgren's syndrome is usually associated with a good prognosis. The patient was sent home with high dose steroids and has continued to improve on follow up.

IDENTIFICATION: An 82 year old man with a past medical history of DM, CAD and GERD presented to the hospital with fever and diffuse erythematous nontender and nonpruritic skin lesions on his face, back, abdomen, thighs and perineum. These initially appeared on his back but later spread to the other areas. He had concomitant new exertional shortness of breath. He denied chest pain, leg swelling, orthopnea, headaches, nausea, vomiting, dysphagia, change in appetite or diarrhea.

Three weeks prior to this the patient had been treated for a ground level fall and MRSA bacteremia that was attributed to cellulitis around the site of a peripheral IV. He had been discharged on vancomycin which he was receiving at home through a PICC line. His blood cultures during the prior admission had cleared immediately after the start of antibiotics. Transesophageal echocardiogram was normal.

The patient denied any history of alcohol, tobacco or illicit drug use. He was a military veteran, had never been married and lived in a monastery as a Trappist monk.

PHYSICAL ABNORMALITIES: The patient was febrile to 38.3C. Diffuse small erythematous macules were present on the abdomen, back and chest. There was a round raised crusted lesion with an erythematous base on the upper left thigh and above the right upper lip. All lesions were nontender. Lung exam was notable for diffuse expiratory wheezing in all lung fields, more prominent on the right side. Extremities showed trace pitting edema and findings consistent with osteoarthritis.

LAB RESULTS: CBC was notable for pancytopenia (hemoglobin level 7.3 g/dL, WBC 2,470 per microliter and platelet count 40,000 per microliter). ESR and CRP were elevated.

DIFFERENTIAL DIAGNOSIS: Sweet's syndrome, erythema nodosum, bacterial, fungal or viral dermatitis, a drug reaction to Vancomycin such as linear IgA bullous dermatosis, a granulomatous process.

TREATMENT: Skin biopsy showed diffuse neutrophilic infiltration in the dermis without vasculitis, consistent with Sweet's Syndrome. The patient was started on intravenous corticosteroids and vancomycin was discontinued. CT of the chest, abdomen and pelvis showed a large tumor in the distal esophagus with invasion into local tissue. The patient deferred further workup or biopsy and chose comfort-only measures.

DISCUSSION AND CONCLUSION: Sweet's syndrome, (acute neutrophilic dermatosis) is an uncommon inflammatory disorder characterized by the abrupt appearance of erythematous papules often accompanied by high fevers and elevated inflammatory markers. It can be idiopathic (Classic Sweet's syndrome) or associated with drugs or malignancies as in this case.

ABSTRACT: A 44-year-old quadriplegic woman presented with fevers, hematuria, and a leukemoid reaction of $>70,000$ cells/mL. Workup for infectious etiologies was negative. A large bladder mass with bilateral lymphadenopathy was discovered by pelvic CT with biopsy revealing a dedifferentiated urothelioma. The cancer was deemed inoperable. The patient was subsequently found to have an elevated GCSF of 182.9 pg/ml (normal range 0.0 – 39.1 pg/ml), consistent with a GCSF paraneoplastic syndrome driving her leukemoid reaction. The patient's mental status became acutely altered in conjunction with poor lung function, likely consequences of leukostasis. Administration of IV Solumedrol temporarily alleviated these symptoms, enabling her to participate in her end-of-life planning. Unfortunately, the patient developed lung and bladder infections while on steroid therapy and died.

IDENTIFICATION / CHIEF COMPLAINT: A 44-year-old quadriplegic woman presented with a 6-monther history of intermittent fevers and hematuria.

HISTORY: The patient had no reported history of urinary tract infections until approximately 6 months prior to presentation at which time she began experiencing intermittent fevers and hematuria. During this time she had been hospitalized repeatedly for anemia and presumed urosepsis (no outside records available).

PHYSICAL ABNORMALITIES: The patient remained communicative and cooperative until hospital day 4, at which time she experienced a sudden decline in her mental and respiratory status. The patient was found to be agitated, disoriented, and though able to follow simple commands was incapable of meaningful communication. Strength, sensation, and reflexes were all unchanged from her baseline disability.

LAB RESULTS: Markedly elevated white count, left shifted, mature granulocytes. Elevated GCSF of 182.9 pg/ml (normal range 0.0 – 39.1 pg/ml)

DIFFERENTIAL DIAGNOSIS: Leukemoid reactions have been associated with DKA, retroperitoneal hemorrhage, medications (e.g., sulfa, glucocorticoids, GCSF), specific infections (Clostridium difficile, tuberculosis, pertussis), and neoplasms including both sarcomas and carcinomas.

DISCUSSION: This report demonstrates the capacity of dedifferentiated urothelial tumors to produce markedly elevated levels of GCSF spurring leukemoid reactions, complicated in this case by steroid-responsive leukostasis. Leukostasis leads to microischemia and hemorrhage in the lungs and brain, resulting in respiratory distress and confusion. Chest x-ray often shows bilateral pleural effusions, as seen in our patient, and there is evidence that steroids reduce leukostasis and improve outcomes even in myelogenous leukemias. It is also worth noting that the differentiation syndrome following ATRA therapy in APML patients is thought by many to be the result of a differentiation-induced leukocytosis with subsequent leukostasis. Interestingly, differentiation syndrome is also treated successfully with dexamethasone therapy.

ABSTRACT: Hyperammonemia has been reported in a growing number of multiple myeloma cases and has been associated with advanced disease. I report a case of encephalopathy secondary to hyperammonemia as the presenting feature of a patient found to have multiple myeloma. The patient had no evidence of liver disease and was initially treated on suspicion of adult-onset urea cycle disorder. Ammonia levels were refractory to conventional medical therapies including lactulose, rifaximin, phenylbutyrate, sodium benzoate, arginine and methyl-THF. An abdominal CT incidentally revealed infiltrative disease of the spinal column concerning for lymphoma. Biopsy confirmed multiple myeloma.

IDENTIFICATION / CHIEF COMPLAINT: A 73-year-old man presented after 2 weeks of steadily worsening confusion, decreased attention span, and short-term memory loss.

HISTORY: Over the last 6 months he had lost 20 pounds. Two months prior he had been seen for a similar episode of slow-onset confusion complicated by expressive aphasia. A head CT and MRI ruled out hemorrhagic or ischemic stroke. The patient was incidentally discovered to have an ammonia level of 113 $\mu\text{mol/L}$.

PHYSICAL ABNORMALITIES: He had normal vital signs and was conversant but disoriented without focal deficits or meningismus. He had no jaundice, scleral icterus, hepatosplenomegaly, nor any stigmata of advanced liver disease. An essential tremor was present but no asterix was noted.

LAB RESULTS: Ammonia of 120 $\mu\text{mol/L}$, a white count of 4,730/mm³, sodium 144 mEq/L, potassium 3.8 mEq/L, chloride 113 mEq/L, bicarbonate 19 mEq/L, blood urea nitrogen 19 mg/dL, creatinine 0.92 mg/dL, glucose 106 mg/dL, and total calcium of 9.5 mg/dL. Alkaline phosphatase was 107 IU/L, aspartate aminotransferase (AST) 17 IU/L, alanine aminotransferase (ALT) 12 IU/L, total protein 6.9 g/dL, total bilirubin 0.8 mg/dL, albumin 4.2 g/dL.

DIFFERENTIAL DIAGNOSIS: Originally worked up for liver disease (e.g., hepatitis, hemochromatosis, Wilson's), and adult-onset urea cycle disorders (e.g., OTC deficiency). Imaging narrowed the differential to hyperammonemia-associated infiltrative cancers, like lymphoma or multiple myeloma.

DISCUSSION: Our patient lacked the majority of typical myeloma findings. His renal function, calcium and albumin were all normal, his imaging lacked lytic lesions, and his anemia, though present, was milder than is typically seen (usually <10 g/dL). Hyperammonemia was his only presenting feature. Though AMS in multiple myeloma is most commonly attributed to uremia, hypercalcemia, or hyperviscosity there is direct evidence that hyperammonemia is an independent cause of AMS. Effective myeloma treatment rapidly improves ammonia levels and concurrently restores sensorium, providing a direct link between the malignancy, hyperammonemia, and encephalopathy.

CASE PRESENTATION: The patient is a 49 year old bisexual Caucasian male who is a long-distance walker who in the past 4 years has traversed the United States twice from Los Angeles to New York and recently completed a trek from Mexico to Alaska. During his journeys, he would sleep under bridges, in bat caves, redwood trees, and along desert roads.

He initially presented complaining of headache, transient loss of vision, left arm numbness and oral pain after a dental procedure for dental carries. The patient complained of 40lbs weight loss over the past 7 months, but stated that this was usual for him during the course of his treks.

Initial stroke workup revealed complete opacification of the right maxillary sinus with cortical breakthrough along the inferior maxillary floor with concern for a neoplastic or fungal etiology. Incidentally, multiple cystic left upper lobe cavitory lesions were detected, suggestive of a fungal etiology. The patient was admitted and underwent lung biopsy and lumbar puncture.

PHYSICAL EXAM: Ophthalmologic exam revealed bilateral suprachoroidal hemorrhages and decreased peripheral vision bilaterally. There was significant oral thrush with erythema on the right hard palate and tenderness over the right maxillary sinus. Considerable cervical lymphadenopathy was present. Lungs were clear to auscultation. Neurologic exam was non-focal.

DIFFERENTIAL DIAGNOSIS: A broad differential was initially considered including a neoplastic process as well as an infectious etiology given his imaging results. Given his wide geographic exposure, Coccidioidomycosis, Histoplasmosis, and Blastomycosis were considered. Given his thrush, HIV and opportunistic infections with CNS manifestations, such as Cryptococcus (including *Cryptococcus gattii*), Aspergillus, Nocardia, Rhodococcus, and Actinomyces were also considered. Mycobacteria was considered less likely given no history of incarceration, military service, or known TB exposures.

LAB RESULTS: Chemistry panel was unremarkable. CBC was notable for hemoglobin of 10.8 g/dL. Serum cryptococcus antigen was positive. Lung biopsy culture was positive for Cryptococcus. HIV was positive by Western Blot. Absolute CD4 count was 69. Lumbar puncture revealed WBC of 4, protein of 30, and glucose of 58.

DISCUSSION: The patient was diagnosed with AIDS with disseminated cryptococcal infection including pneumonia, choroiditis, and probable meningitis (the patient was on therapy at time of lumbar puncture). He was started on Amphotericin and subsequently HAART therapy.

CONCLUSION: HIV/AIDS should be strongly considered in patients presenting with evidence of disseminated infections suggestive of immunocompromasation.

CASE PRESENTATION: This is the case of a 27-year-old pregnant white female with a history of resistant hypertension and prior idiopathic cardiomyopathy. Followed in cardiology clinic, she was on maximum-dose hydrochlorothiazide, amlodipine, carvedilol, hydralazine, and labetalol. She was admitted for urgent delivery at 37 weeks secondary to blood pressures greater than 220/100 and concern for preeclampsia. Post-partum she continued to have elevated pressures despite medical therapy and medicine consultation was sought to further evaluate secondary causes. The patient denied headaches, visual changes, stroke-like symptoms, chest pain, diaphoresis, nor change in urination. Endorsed significant anxiety.

PHYSICAL EXAM: Vital signs: Afebrile, HR 100-120, BP 150s-250s/80s-150s, RR 20, 97% room air. Anxious, post-partum female with a rounded face. Pupils equal and reactive, extraocular muscles and visual fields intact. Moist mucous membranes. Neck demonstrates no jugular venous distension and no bruits. Heart sounds hyperdynamic and with a soft flow murmur. Lungs clear. Soft, non-tender abdomen without bruits. Extremities with strong and equal pulses, no edema. No neurologic deficits. No rashes or striae.

DIFFERENTIAL DIAGNOSIS: Her young age and resistant and severe hypertension prompted work up for secondary causes including fibromuscular dysplasia, primary aldosteronism, renal artery stenosis, obstructive sleep apnea, pheochromocytoma, and Cushing's syndrome.

LABORATORY ABNORMALITIES AND IMAGING: Labs revealed a CBC with leukocytosis of 21, hemoglobin of 11 and platelet count of 459. Comprehensive metabolic panel was normal other than transaminitis (ALT 234, AST 157, Alk phos 151). Urinalysis was normal and urine toxicology screen was negative. The patient had a normal aldosterone/renin ratio but was noted to have elevated 24-hour urine protein and free cortisol. Plasma and urine metanephrines as well as 24-hour urine catecholamines were severely elevated. Abdominal imaging demonstrated a right adrenal mass with mottled enhancement measuring 6 x 5 x 4cm.

DIAGNOSIS AND TREATMENT: This patient was found to have a pheochromocytoma. Surgery was consulted who recommended improving hypertension control for 4-6 weeks prior to surgery. Phenoxybenzamine was initiated and after an extended stay of medication titration, she was discharged also on terazosin, clonidine, diltiazem, methyldopa, and propranolol.

Subsequent work up demonstrated elevated salivary cortisol and ACTH levels. Patient became Cushingoid and had persistent hypertension. She eventually had resection of a right adrenal mass, which by pathology report noted neoplastic cells with markedly nuclear atypia. Stains were positive for chromogranin, S-100 and negative for CKA1-3, supporting pheochromocytoma. Pathology also noted adrenal cortical hyperplasia, considering her to have an adrenal cortical adenoma (likely with ectopic ACTH secretion) without malignant features.

RESOURCES: Lenders JW et al. Pheochromocytoma. *Lancet*. 2005;366(9486):665-675. | Young WF Jr. Adrenal causes of hypertension: pheochromocytoma and primary aldosteronism. *Rev. Endocr Metab Disord* 2007;8:309.

CHIEF COMPLAINT: Fever, weakness, lightheaded

HISTORY: W.R. is a 73 year-old man who emigrated from Syria 6 months ago. He was in his normal state of health until five days ago when his temperature reached 101 degrees Fahrenheit. Around this time, he also developed a sore throat and generalized weakness. His son-in-law is an internist and prescribed him a course of Augmentin. After taking the antibiotic for several days the fever resolved, but his sore throat and weakness persisted. Upon experiencing significant lightheadedness he was brought to the hospital for further evaluation.

Upon arrival patient was asymptomatic and an extensive review of systems was unrevealing.

PHYSICAL ABNORMALITIES: Patient was afebrile and orthostatics were normal. A thorough physical exam resulted in no detectable abnormalities.

LAB RESULTS: CMP showed no electrolyte abnormalities and proper liver and kidney function. CBC revealed pancytopenia with white blood cell count of 2.3, hemoglobin concentration of 6.4, and platelet count of 50. Differential exhibited an absolute neutrophil count of 0.3 and atypical lymphocytes.

DIFFERENTIAL DIAGNOSIS:

1. Aplastic anemia
2. Myelodysplastic syndrome
3. Leukemia
4. Folate/Vitamin B12 deficiency

CASE PRESENTATION: Patient underwent multiple laboratory studies to rule out aplastic anemia. Acute hepatitis panel, parvovirus, HIV, EBV, and CMV were undetectable. Metabolic studies including TSH, folate, and vitamin B12 were all within normal limits. Peripheral blood smear revealed absolute neutrophilia with blasts. After consulting hematology/oncology, a bone marrow biopsy was performed. Bone marrow aspirate showed findings consistent with acute myeloid leukemia (AML) and myelodysplasia-related changes. Treatment for AML consists of induction chemotherapy with daunorubicin and cytarabine. However, due to the cost of the procedure and the desire to be near his family, Mr. W.R. elected to return to his home in Syria without further treatment.

DISCUSSION: By the time patients with AML exhibit symptoms, the prognosis is already bleak. Symptoms of AML stem from bone marrow failure resulting in fever from absolute neutropenia with or without documentation of infection; fatigue, weakness, and dizziness from anemia; and easy bleeding and bruising from thrombocytopenia. Peripheral blood smears reveal blasts and bone marrow biopsies provide the definitive diagnosis. Induction chemotherapy has a 65% response rate, though relapse is a common struggle for the patient and oncologist. Clinical trials are currently investigating chemotherapy regimens, stem cell transplant, and immunotherapy to better treat those diagnosed with AML.

CASE PRESENTATION: A 65 year old female with past medical history significant for hypertension, recent surgical removal of benign throat polyp, peripheral arterial disease and greater than 100 pack-year smoking history presented with one week of weakness and confusion. Home medications included Cilostazol, Hydrochlorothiazide, Losartan, Amlodipine and several supplements. Patient was somnolent but arousable, remainder of exam was unremarkable. Labs were significant for hemoglobin 16.3, Potassium 2.6, BUN 41, Cr 1.39, calcium 18.9, ionized calcium 2.47, and albumin 3.9. EKG showed first degree heart block with diffuse t-wave flattening. CT head showed non-specific atrophy.

DIFFERENTIAL: The differential for encephalopathy and weakness is broad and includes neurologic, metabolic, infectious, and medications/toxins as possible etiologies. Hypercalcemia was thought to be the cause of this patient's symptoms.

HOSPITAL COURSE: Patient was treated with aggressive IV fluid resuscitation, potassium was repleted and HCTZ was held. Patient also received one time dose of Zoledronic Acid. Calcium quickly trended down and mental status cleared.

WORK-UP: Review of records revealed hypercalcemia dating as far back as 6 years ago with calcium of 10.3; no prior work-up was performed due to lack of insurance. The cause of her hypercalcemia was investigated during her hospital stay. PTH was 11 (concerning for malignancy on nomogram), normal PTH-related peptide, vitamin A and D levels, thyroid function tests, SPEP, UPEP and 24-hour urine calcium were also unremarkable. Malignancy work-up was negative. CT chest showed a 7 mm pulmonary nodule of unknown significance. Further questioning revealed patient was a sales rep for Herbalife and used almost all of their products. Review of supplements revealed patient was taking at least 2,800 mg of calcium per day in addition to dietary sources.

CONCLUSION: The presenting symptoms of hypercalcemia are often vague, the well-known mnemonic "stones, bones, moans and groans" highlights the most common symptoms. Common causes of hypercalcemia include hyperparathyroidism, malignancy, familial hypocalcemia hypercalcemia, medications, milk alkali syndrome, high vitamin A or D, granulomatous disease and increased bone turn over. This patient had an elevated PTH and significant smoking history as well as the finding of a lung nodule raising concern for malignancy; however, she had a negative PTH-rp, making her excessive supplement usage the most likely cause of the hypercalcemia. Patient was encouraged to limit her total daily calcium intake from all sources to the FDA recommendation of 1,200 mg/day. It is important to not forget about the supplements.

CASE PRESENTATION: Mr. L is a 42 y/o previously healthy male who presented to the ED with a five day history of fevers, chills and severe muscle aches in his legs and back. He also described intermittent headaches with photophobia, nausea and poor appetite. Ten days prior to the onset of his symptoms, he returned from Costa Rica where he spent a week with his family hiking and swimming in waterfalls and fresh water lakes. He did not receive pre-travel vaccinations or malaria prophylaxis. He was bit multiple times by mosquitos and thought he might have had a spider bite.

On admission, Mr. L had a temperature of 40.3 Celsius, a heart rate of 103 and appeared ill. He had conjunctival injection, mild diffuse abdominal tenderness and a fine macular rash over his lower extremities. His laboratory data had the following abnormalities: BUN 30, creatinine 4.04, total bilirubin 2.5, AST 186, WBC 10.0 with 19 bands. He had a chest x-ray, urinalysis and blood cultures that were negative.

DIFFERENTIAL DIAGNOSIS: Our differential was very broad initially, but in reviewing the details of his presentation, exposures and incubation period we were concerned about Dengue fever, Hanta virus, Chikungunya fever, Rocky Mountain spotted fever, leptospirosis and malaria. His initial antibody testing and blood smears were all negative, however a repeat leptospira IgM antibody assay done on day 5 of his hospitalization later confirmed that he had leptospirosis. His renal failure, fevers and headaches all resolved within 5 days of initiating doxycycline.

TREATMENT: Treatment for leptospirosis is 14 days of 100 mg BID of doxycycline. IV penicillin may be used in severe cases. It is important to start doxycycline empirically if leptospirosis is considered in the diagnosis because of its potential for multi-organ failure.

CONCLUSION: Leptospirosis is a treatable but potentially lethal disease. One should consider leptospirosis in individuals returning from the tropics who have been exposed to fresh water, especially during rainy seasons. Leptospirosis is a spirochete and commonly thought of as being present in rodent urine, but can also come from cattle, pigs, horses and dogs. Two phases of disease can occur with leptospirosis, a bacteremic phase and an immunologic phase, which occurs later after exposure, also known as Weil's disease. The immunologic phase can result in ARDS, acute liver failure, acute renal failure and aseptic meningitis and has a fatality rate of 5-10%.

CASE PRESENTATION: Patient is a previously healthy 23-year-old Caucasian female who presented to the ED with about 3 weeks of fevers as high as 104F and unremitting generalized headaches. She first noticed left eye swelling, redness, and mild blurry vision – diagnosed as conjunctivitis. Pain then spread to her cheeks and jaw over the ensuing three days and she was subsequently treated with a 14-day course of Keflex and Bactrim for sinusitis. Though eye symptoms resolved, she noticed worsening malaise, neck pain, fevers, chills, anorexia, 10-pound weight loss, and an episode of syncope. Although work-up was negative for meningitis in the ED, she was empirically treated with ceftriaxone, clindamycin, and vancomycin. CT scan showed bilateral enlarged cervical lymph nodes with central necrosis. ENT was consulted for lymph node biopsy.

PHYSICAL EXAM: Patient's VS are normal. She is sleepy but interactive. HEENT exam showed mild OP injection. Neck exam is supple and demonstrates multiple tender, enlarged lymph nodes in the anterior and posterior cervical chain – largest of 2 cm in diameter on the R posterior side. Skin has a lacy reticular rash on her chest and arms. The rest of her exam is benign.

LABS: CBC showed mild anemia with several atypical lymphocytes, thrombocytopenia. CMP demonstrated mildly elevated liver enzymes. LDH 763, TSH 3.3. Monospot, CMV titers, hepatitis panel, HIV, toxoplasma titers, rapid strep are all negative. EBV 542. Blood and CSF cultures are negative.

DIFFERENTIAL/DISCUSSION: Given the patient's initial exam and lab findings, she had a presumed diagnosis of supportive lymphadenitis due to infectious etiology – likely mononucleosis. Pathology of five lymph nodes demonstrated striking karyorrheis and extensive necrosis. There was a rim of surrounding C68+ histiocytes and T-cells, eosinophils, CD8+ and CD4+ T-cells. Differential based on pathology included Kikuchi-Fujimoto Disease, lupus lymphadenitis, drug-induced lymphadenitis, and infection. Review of the patient's medical records almost three years later reveals no recurrence of lymphadenopathy, nor further presentations for infectious, rheumatologic, or malignant conditions.

CONCLUSION: Kikuchi-Fujimoto disease is a self-limited condition and a diagnosis of exclusion. It is characterized by benign lymphadenopathy, fevers, and systemic symptoms – affecting young adults, mostly of Asian descent. Although the etiology is still unknown, viral and autoimmune mechanisms are thought to be triggers. Treatment is supportive and symptoms generally resolve spontaneously within 4 months. Physician awareness of this disorder may help prevent misdiagnosis and inappropriate treatment.

CASE PRESENTATION: Patient is a previously healthy 26-year-old Kenyan male referred to MTRH from an outside hospital for headache. It was described as global, throbbing and progressive of two weeks duration, recently associated with photophobia and dizziness. Patient had subjective, intermittent fevers at the onset, with several days of new generalized weakness and inability to sit up or walk. On admission, he had new neck pain/stiffness and blurry vision. He had no preceding traumas, seizures, LOC, URI symptoms, skin lesions, nausea, vomiting, or diarrhea. PMH and family history was not significant for chronic diseases, immunosuppression or malignancies. On arrival, he received ceftriaxone and dexamethasone prior to obtaining any diagnostic studies.

PHYSICAL EXAM: VS were within normal limits. He appeared ill but with a GCS of 15. HEENT exam demonstrated mydriasis and anisocoria. He had nuchal rigidity. Neuro exam demonstrated 5/5 strength, no sensory deficits, and a negative Kernig sign. His back had moderate kyphoscoliosis. The heart, lung, and abdomen exams were normal.

DIFFERENTIAL DIAGNOSIS: Differential was initially wide. Infectious processes included viral meningitis, intracranial abscess, acute HIV seroconversion, disseminated TB, advanced-stage syphilis, and toxoplasmosis. Neurologic processes included complex migraine headaches, dural venous thrombosis, TIA, and subarachnoid hemorrhage. Intracranial malignancy included astrocytoma, medulloblastoma, ependymoma. Given his ethnicity, neurosarcoidosis was also considered.

LABS: Initial labs showed an unremarkable CBC and mildly elevated alkaline phosphatase on CMP. LP showed increased opening pressure and cloudy CSF with 360 WBCs, 4 RBCs, protein 13, glucose 47, negative Gram stain. India Ink test positive. CRAG positive.

DISCUSSION: Cryptococcus meningitis was previously thought to be seen only in immunocompromised individuals; although this is true for the *C neoformans* species, a different strain can infect healthy adults as well. An outbreak of *Cryptococcus gattii* meningitis occurred in the Pacific Northwest and California between 2004-2011. Before this time, it was found mostly in the tropics/subtropics; it now infects an estimated 25 healthy, immunocompetent individuals living in the Pacific Northwest yearly, and can also infect domestic animals. The incubation period is much longer than that of the *C neoformans* species, averaging 6-7 months.

CONCLUSION: Infection with *C gattii* is more likely to progress to the brain than *C neoformans*, is more difficult to treat, and is associated with high morbidity from neurologic sequelae. There are no preventive or precautionary strategies that can be undertaken to avoid becoming infected. Rapid diagnosis and treatment are essential to preventing progression of neurologic disease.

CASE PRESENTATION: Patient is a 10-week pregnant 29-year-old female who presented to podiatry clinic with 2 weeks of bilateral ankle swelling and pain. Symptoms initially started in her left ankle; she was seen in the ED and prescribed amoxicillin and naproxen. Despite taking these medications, symptoms progressed to her right ankle and knees, with subjective fevers. In the preceding two days, she has been unable to ambulate, requiring a wheelchair due to pain. She recently traveled to Georgia and Yellowstone, but denied insect bites. She denied any recent rashes other than redness around the ankles. No malaise, chills, anorexia, weight loss, injuries, or recent illnesses. Initial labs were concerning for an ESR of 58 and CRP of 87.

PAST MEDICAL HISTORY: Significant for Chlamydia, 4 prior normal pregnancies, and patellofemoral pain syndrome. Family history does not include any rheumatologic disease.

PHYSICAL EXAM: VS are normal. She is in moderate distress from pain. Extremity exam shows swollen, erythematous ankles bilaterally – more pronounced at the lateral aspects and exquisitely tender to mild palpation. Wrists and knees demonstrate no effusions or erythema, but notable pain as well. Skin shows lacy rash on her back, with erythematous and tender nodules on bilateral shins. Strength is normal and the rest of her exam is benign.

LABS: CBC showed mild anemia. CMP unremarkable. ASO 189. GC, RPR, HCV, HIV, Lyme EIA, parvovirus, Coccidioides antibodies, Histoplasma, PPD are all negative. ANA and RF negative. ACE level 59. CXR shows bilateral prominent and nodular hila.

DIFFERENTIAL/DISCUSSION: Sarcoidosis is often considered a pulmonary disease, but it can affect any organ with diverse manifestations. It is a diagnosis that usually requires biopsy showing non-caseating granulomas after exclusion of other causes of granulomatous inflammation. This patient had a classical presentation of Lofgren Syndrome: fevers, ankle swelling, erythema nodosum, and bilateral hilar lymphadenopathy, thus precluding the need for biopsy.

CONCLUSION: Lofgren Syndrome is self-limited, with excellent prognosis in patients with HLA phenotype DRB1*03. Treatment includes NSAIDs, with spontaneous remission in more than 90% of cases within 2 years. This patient was started on prednisone given her functional limitation, rather than symptomatic lung disease. Prospective RCTs show that systemic steroids are associated with only a 10% improvement in FVC after 5 years. Initiation of steroids is also associated with higher risk of recurrence and long-term steroid dependence. Physicians should try to with-hold treatment for 3-6 months, if possible, to assess for spontaneous resolution.

CASE PRESENTATION: GW is a 23 year-old man from South Sudan, presented with hemoptysis. His chest X ray in emergency department showed a left upper lobe lesion. His sputum smear showed 4+ acid-fast bacilli, later identified as *Mycobacterium tuberculosis*. He was admitted to the tuberculosis isolation unit and started the treatment for active pulmonary tuberculosis with the four-drug regimen, isoniazid, rifampin, pyrazinamide and ethambutol.

On day 3 of hospitalization, we have decided to increase the dose of rifampin to 1600 mg daily. A new order was placed to start the new dose in the morning. The nurse misread the order. The patient was given an extra 1600 mg of rifampin at the wrong time. A few minutes later, he became dizzy and unsteady. The nurse apologized to the patient about the error. When the patient mentioned his new symptoms, the patient was told that the doctor wouldn't come and see him at night. However, neither the attending physician nor I received any calls from the nurse. The nurse did not report the medication error in the patient's medical record. Next day, the patient mentioned about the error and his new symptoms. He was quite upset about the whole situation and refused to take any more TB medications.

DISCUSSION: It went wrong in many levels. The patient was locked in TB isolation room, which limited the communication between the patient and his providers. His TB medications were readily available in the floor pharmacy cabinet, so there was no safety net usually provided by the pharmacy. I failed to communicate directly with the nursing about the medication change. The nurse failed to report the medication error and the patient's symptoms to the physician. Worst of all, the patient was told that the doctor refused to come and see the patient after he became sick. We have failed to practice "First, do no harm." We lost the patient's trust. We have apologized and asked him to trust us again. We have promised him that we would do our best to prevent this in the future. Patient finally agreed to take his medications again.

It was a terrifying and frustrating experience even though it did not lead to disastrous consequences. Now, I value more about the essential communication skills and interactions among the health care team members to provide safety and highest quality care for our patients.



ACPSM

American College of Physicians
Leading Internal Medicine, Improving Lives

Utah
Chapter

UTAH CHAPTER LEADER COUNCIL & COMMITTEES

GOVERNOR

Robert Pendleton, MD FACP

VICE CHAIRMAN

Douglas Smith, MD FACP

TREASURY/FINANCE

Anthony Musci, MD FACP

EARLY CAREER PHYSICIANS COMMITTEE

Megan Engelen, DO FACP - Chair

Devin Horton, MD

INTERNAL MEDICINE INTEREST GROUP & STUDENT COMMITTEE

Katie Martin, MD - Chair

Erik Riessen, MD FACP

Jordan Koncinsky, MSII

David Smyth, MSII

MEMBERSHIP & RETENTION COMMITTEE

Michael Galindo, MD FACP - Chair

Thomas Caine, MD FACP

Corwin Edwards, MD FACP

Susan Terry, MD FACP

RESIDENTS & FELLOWS COMMITTEE

Scott Woller, MD FACP - Chair

Kencee Graves, MD

SCIENTIFIC PROGRAM MEETING COMMITTEE

Nate Allred, MD – Co-chair

Karli Edholm, MD – Co-chair

Kristen Ries, MD MACP