**2013 Clinical Vignette Competition**

**Part I of II**

**Identification:** 65-year old male with chronic kidney disease, rheumatoid arthritis and newly-diagnosed cirrhosis.

**Chief Complaint:** Decreased level of consciousness, urine and stool incontinence for one day; severe right ankle pain and abdominal distention for two days.

**History:** Patient on chronic methotrexate for rheumatoid arthritis, with poorly-healing non-traumatic right ankle fracture. Recent hospitalization for abdominal pain and distention, requiring large volume therapeutic paracentesis for ascites. Work up revealed multifactorial etiology for his cirrhosis. Methotrexate was discontinued.

**Physical Abnormalities:** Lethargic, responsive to questions. Tachycardic, no murmurs. Tender to palpation all abdominal quadrants, distended and tense with positive fluid wave. Tense edema to bilateral knees. Right ankle exquisitely tender and warm to touch. No asterixis.

**Lab Results:** WBC 1.2, hgb 12.3, platelets 146, Na 128, glucose 36, BUN 45, creatinine 1.92, protein 4.9, albumin 1.8, bilirubin 155, ALT 51, AST 62, urine macro: nitrite and leuk esterase positive; urine micro: 5 WBC/hpf, 1 RBC/hpf, bacteria 1+, PT/INR 19.2/1.7, lactate 8.2, ammonia 17

**Differential Diagnosis:** Urinary tract infection, bacteremia, spontaneous bacterial peritonitis, clostridium difficile infection, septic right ankle joint.

**Case Presentation:** Day before admission, presented to ED for chest and right ankle pain. Discharged with negative workup and scheduled for follow-up with his hepatologist and orthopedist. Patient returned home. Mental status worsened and became incontinent of urine and stool. Returned to ED and was admitted to STICU. Patient underwent diagnostic paracentesis, and blood and urine cultures drawn. Required vasopressors to keep mean arterial pressure (MAP) greater than 60 mmHg and ScVO2 greater than 60%. Patient was given broad-spectrum antibiotics. Right ankle was tapped and sent for culture. Blood, urine and ankle fluid returned positive for Pseudomonas aeruginosa. Peritoneal fluid returned positive for Streptococcus salivarius. Patient did not survive hospitalization.

**Discussion:** Disseminated Pseudomonal infection from urinary tract infection, in immunocompromised patient with end-stage liver and chronic kidney disease, also with spontaneous bacterial peritonitis. Patient was on chronic methotrexate therapy and may have been colonized with Pseudomonas during last hospitalization. Patient developed a urinary tract infection and subsequent bacteremia and septic right ankle joint.

**Conclusion:** Pseudomonas is a known opportunistic gram-negative aerobic bacterium with a predilection for immunocompromised hosts. It is the second most common cause of nosocomial infections, the third most common cause of urinary tract infections and seventh most common cause of bacteremia. Pseudomonas has also become more antibiotic resistant, complicating treatment of these serious infections.

**Part II of II**

**WELCOME 2013 CLINICAL VIGNETTE**

**JULIA CURTIS, MD R-1**

**SEVERE RIGHT ANKLE PAIN ASSOCIATED WITH DECREASED LEVEL OF CONSCIOUSNESS & URINE INCONTINENCE**

**WELCOME 2013 CLINICAL VIGNETTE**

**JULIA CURTIS, MD R-1**

**SEVERE RIGHT ANKLE PAIN ASSOCIATED WITH DECREASED LEVEL OF CONSCIOUSNESS & URINE INCONTINENCE**
Case Presentation: An 18 year old previously healthy female presented to the hospital for persistent vomiting, dehydration, and abnormal LFTs. Vomiting started four days earlier. Mild jaundice was ongoing for two days prior to presentation. She reported pruritus, diffuse abdominal pain, fatigue and malaise for the previous two weeks, subjective fevers, chills, and poor appetite. She reported being sexually active with one male partner. She reported regular menses and her usual period started during her hospital stay. History was otherwise unremarkable.

Objective Abnormalities: Initial temperature was 38.0 °C. She appeared listless with mild scleral icterus. She had right upper quadrant tenderness to deep palpation, without guarding or rebound. Initial lab studies were remarkable for a total bilirubin of 4.8, Alkaline phosphatase of 285, ALT of 364 and AST of 227. CBC abnormalities included a 61% predominance of lymphocytes with a normal WBC count of 8.4 as well as 2+ atypical lymphocytes.

Differential Diagnosis: Considerations for acute vomiting, RUQ pain, and elevated transaminitis in this young female included viral infection including acute viral hepatitis A, B, and C, as well as HIV, CMV, and EBV. Mechanical biliary obstruction, cholecystitis, cholangitis, pancreatitis, autoimmune hepatitis, toxic ingestion including alcohol, alpha-1 antitrypsin deficiency, Wilson’s, or hemochromatosis were also considered. Hyperemesis gravidarum was unlikely given her normal menses and did not account for other findings. Additional testing ordered upon admission included limited RUQ abdominal ultrasound, lipase, EBV, CMV, HIV, and acute viral hepatitis panel. Results were normal except for positive EBV VCA IgG and IgM which is consistent with acute or convalescent infection. She was subsequently diagnosed with an infectious mononucleosis.

Treatment: This patient was diagnosed with an atypical infectious mononucleosis, lacking many of the usual features classically associated with this infection including complaints of sore throat, tonsillar exudate, marked lymphadenopathy, or splenomegaly. This however did not rule out the diagnosis and keeping a broad differential in mind proved helpful in identifying her underlying problem. Treatment was supportive. She was discharged within 24 hours to have outpatient follow-up and her liver function tests to be checked with her PCP to ensure resolution with time.

Conclusion: In a young patient with abnormal LFTs, EBV associated infectious mononucleosis should be a differential consideration, especially with classic features of infectious mononucleosis, but also in cases when classic features are lacking and the diagnosis remains uncertain.
Case Presentation:
A 51 year old male with minimal medical history presented to the University of Utah Emergency Department with complaints of flu-like symptoms. The patient complained of myalgias, fevers up to 101.6 F at home, and headache. He also endorsed a cough productive of yellow-green sputum and decreased appetite. The patient said that he had lost weight over the past year, though he did not know how much. On review of systems, he complained of easier bleeding and bruising. The patient was admitted by night float team at the University of Utah to be worked up by general internal medicine wards team.

Physical Exam Abnormalities:
The patient was febrile to 38.5. Mild bibasilar crackles were present on pulmonary exam. Scattered ecchymoses were present throughout upper and lower extremities. No hepatosplenomegaly was felt on exam.

Lab Results:
WBC 1.01, hemoglobin 8.9 with MCV 104, platelets 79, PMNs 36%, bands 7%, lymphocytes 49%.

Differential Diagnoses:
The patient met SIRS criteria with leukopenia and fever. There was concern for influenza, so a respiratory virus panel was ordered. He also had a neutropenic fever, and was treated with meropenem for possible infection. Blood cultures were drawn. A chest x ray was ordered to evaluate pneumonia, though was unremarkable. HIV, EBV, CMV, and parvovirus testing were ordered. Iron studies, B12, and folate were ordered to evaluate the patient’s anemia. The patient was otherwise healthy, but a potential hematologic malignancy was a concern.

Discussion:
The patient was transferred to the Huntsman Cancer Center for a bone marrow biopsy the day after admission. The bone marrow biopsy showed hypercellular marrow with atypical lymphocytes that were large with cytoplasmic projections. The patient’s flow cytometry showed a CD5 negative, CD10 negative B cell lymphoproliferative disorder. The results were consistent with hairy cell leukemia. This is a very rare, though responsive leukemia that can be treated with cladribine monotherapy. His easy bruising was likely caused by the thrombocytopenia and platelet dysfunction that can occur in hairy cell leukemia. He was diagnosed with influenza B as well. The patient was treated inpatient initially and then discharged for outpatient chemotherapy treatment.

Conclusion:
When otherwise healthy people present with an illness in the setting of pancytopenia, hematologic malignancy should be a consideration as a differential diagnosis.
Case Presentation: The patient is a very healthy 33 year old male who was admitted to Intermountain Medical Center with acute respiratory distress. The patient normally runs in marathons, but after a 10 mile run 5 days prior to presentation, the patient developed myalgias. He developed a sharp and non-productive cough, with fevers up to 103.5 F in the days prior to admission. He went to an Urgent Care where a chest x ray was unremarkable. On the day of presentation, he was breathing quickly and shallowly, so his family brought him to the emergency department.

The patient had traveled to Hawaii with his family 3 months prior to presentation. He also stayed at the family cabin two weeks prior to presentation, which is 10 miles from Capitol Reef. While there he slept on the porch at night with his wife, where he thought a rodent touched him in his sleep. The cabin has well water. He found a dead deer with his wife while hiking and also dug up some bones with his mother while hiking in that area.

Physical Exam: The patient quickly developed respiratory failure within 24 hours of being admitted, and was intubated at the time of examination. He was able to nod yes/no. Crackles were heard at bilateral lung bases. He had scratches on hands, but no insect or animal bites.

Labs:
- WBC 15.4, hemoglobin 13.2, hematocrit 35.9, platelets 49
- Differential: 35% neutrophils, 53% bands, 6% lymphocytes, 5% monocytes
- Na 121, AST 238, ALT 123, lactate 5.1

Respiratory virus panel: negative
S pneumo and Legionella urine antigens: negative
CXR: unremarkable

Differential Diagnoses: As the patient was otherwise healthy, an infectious illness was of concern. The primary team had sent testing for tularemia, Q fever, Borrelia, Yersinia, Leptospira, coccidiodes, RMSF, hantavirus, mycoplasma, Herpes simplex, and PCP.

Discussion: The patient was otherwise healthy prior to presentation and presented with fever, respiratory failure requiring intubation, lactic acidosis, thrombocytopenia, hyponatremia in the setting of increased hematocrit on presentation (hct 51.6), and exposure to rodents. He needed pressor support and had an EF on echocardiogram of 35%. After one week of waiting for results, his hantavirus IgM returned elevated.

Conclusion: An otherwise young healthy person who has rapid respiratory failure should have hantavirus as a differential diagnosis.
Case Presentation: Mr. F is a 49-year old gentleman with no significant medical history who was seen in an ED in Park City for back pain after falling on ice. Initial X-ray revealed no abnormalities; however, CT scan revealed a lemon sized mass within his abdomen. Patient was then lost to follow up because he didn’t have insurance. He returned to the ED 2 months after initial CT scan and was prescribed a 5 day course of oxycodone and sent home. Over the next month, his back pain continued to worsen until patient became bed-bound. At the prompting of his family, he returned to the ED a third time due to debilitating back pain along with lower extremity weakness. During his third visit, a repeat CT of his abdomen revealed a compression fracture at L3 along with diffuse intra-abdominal masses with the largest measuring 7x5x7 cm. Subsequent imaging revealed diffuse bony metastases throughout the CT, and L-spine and calvarium. In addition there were several sub-centimeter hepatic lesions.

Physical Exam abnormalities: Initial exam in the ED revealed bilateral lower extremity weakness secondary to his compression fracture along with substantial bilateral inguinal adenopathy. CBC and CMP were essentially normal without any elevation in total protein. Other laboratory results were unrevealing. Upon arrival to the floor, a thorough physical exam revealed a quarter sized, flat, irregular, multi-shaded lesion with discrete tan and brown colored components on the patient’s lateral right heel.

Differential Diagnosis: Initial differential was difficult to compile as imaging and lab testing were not consistent with any specific primary carcinoma, though Multiple Myeloma or sarcoma were the leading initial possibilities. After the skin lesion was found on exam, dermatology performed a skin biopsy with pathology returning for malignant melanoma. CT guided biopsy of intra-abdominal mass was then obtained which showed poorly differentiated malignancy, suspicious for melanoma.

Treatment: At the time of diagnosis, patient was inpatient in the Huntsman Cancer Hospital for pain control and workup. Following diagnosis, he received palliative radiation prior to discharge. He is currently awaiting a clinical trial.

Discussion: In general, metastatic melanoma carries a poor prognosis with median survival of 6-12 months. Systemic therapy is indicated in patients with widely metastatic disease. Treatments include immunotherapy such as ipilimumab or IL-2, ZELBORAF for BRAF mutation, systemic chemotherapy, clinical trials, or palliative radiation.

Conclusion: Though imaging has remarkable diagnostic value, we must never forget that a thorough physical exam may hold the keys to diagnosis.
Identification: A 60 year-old man with a history of atrial fibrillation and Charcot foot.

Chief Complaint: “I found my husband on the floor”

History: Patient was brought in by his wife after she found him on the floor of their bedroom at 0600. Prior to admission, he experienced 2 days of generalized malaise, fevers and chills that also encompassed a day of fasting which was done for religious reasons. He was admitted to an outside hospital with septic shock, group A beta-hemolytic strep bacteremia and acute renal failure and treated with fluid resuscitation, ertapenem and vancomycin. The sepsis improved, but renal insufficiency and altered mentation did not, and he developed worsening thrombocytopenia.

Physical Abnormalities: A delirious, disoriented man who was unable to communicate or follow commands. The left lower extremity had pitting edema and confluent erythema present from the toes to the mid-tibial level. Bilateral charcot foot with chronic appearing wound was present. The exam was otherwise normal.

Lab Results: During his 3 day admission at the outside hospital, platelets decreased from 117 to 83. His creatinine improved from 2.2 to 1.5. INR and aPTT were elevated on admission at 1.5 and 50 respectively and were 1.2 and 54 on transfer. Total bilirubin increased from 1.4 to 2.1, LDH was 1887, and the peripheral smear showed shistocytes. Direct Coombs test was negative. Fibrinogen was elevated at 840. An MRI of the brain was normal.

Differential Diagnosis: TTP-HUS, DIC, vasculitis, SLE

Hospital/Treatment Course: The patient underwent plasma exchange with improvement in hemolysis (as evidenced by the absence of shistocytes, and normalization of LDH and fibrinogen), resolution of thrombocytopenia, and improvement in renal function. He had a total of 6 sessions with marked improvement in mental status.

Discussion/Conclusion: This patient’s condition was felt to be most consistent with TTP/HUS given the renal insufficiency, mental status changes, thrombocytopenia, and evidence of hemolysis. Certainly this is an odd presentation of TTP/HUS as it occurred in the setting of sepsis, and one would expect any microangiopathic hemolytic anemia to be due to DIC in that setting. The TTP/HUS diagnosis was favored over DIC given the elevated fibrinogen level and normalization of the INR. The elevated INR on admission was felt to be due to dabigatrin which the patient regularly took for atrial fibrillation anticoagulation. No obvious etiology of TTP/HUS was identified.
Identification: 22-year-old female.

Chief Complaint: “Shakes and shivers”

History/Case Presentation: 22-year-old Caucasian female with schizoaffective disorder, presents from a psychiatric facility with “shakes and shivers” for one week. Associated symptoms were diaphoresis and agitation. She denies fever, headache, visual changes, nausea, vomiting, bowel/bladder incontinence, loss of consciousness, focal weakness or visual/auditory hallucinations. Home medications included clonazepam, diphenhydramine, benztrapine, lithium, risperidone, trazodone, escitalopram, and methocarbamol. Risperidone dosing was doubled five days prior to admission.

Physical Abnormalities: Upon arrival, vitals revealed a labile systolic blood pressure and tachycardia. The initial interaction with the patient was notable for somnolence (but easily aroused), a flat/constricted affect, a vacant gaze, and slowed response to questions. Decreased 4/5-muscle strength and a coarse rapid tremor in the extremities were accompanied with diffuse rigidity.

Lab Results: Laboratory findings were significant for leukocytosis at 14.6 x 10³/microl (83% neutrophils) and elevated levels of AST 160 U/L and CK 7495 IU/L. The patient’s lithium level was low at 0.3mEq/L. Chest X-ray and CT of the head showed no acute process. EEG findings revealed diffuse slowing consistent with encephalopathy.

Differential Diagnosis: Differentials were meningitis/encephalitis, neurologic conditions (malignant catatonia, agitated delirium, serotonin syndrome), or endocrine conditions (thyrotoxicosis or acute porphyria). Focus was primarily placed on the possibility of neuroleptic malignant or serotonin syndromes, given use of multiple psychotropic medications and recent dosage increase. Patient’s primary sign was rigidity, which pointed prominently toward neuroleptic malignant syndrome (NMS). Serotonin syndrome presents with myoclonus, hyperreflexia, and diarrhea that were less prominent in our patient.

Discussion: NMS is a syndrome of hyperthermia, rigidity, altered consciousness, and autonomic dysfunction. Usual suspects are typical antipsychotics (haloperidol), but atypical antipsychotics (risperidone and olanzapine) may also be culprits. The predisposing factors in our patient included recent dose escalation of risperidone, concomitant use of lithium and anticholinergics, and dehydration. Treatment is based on the Woodbury classification system and begins with withdrawal of the offending agent. Pharmacological therapy is largely unsupported by clinical trials. Possible agents include: dantrolene, bromocriptine, and benzodiazepines. In our patient — all offending medications were stopped and started on bromocriptine, benzodiazepines, and IVF’s. The patient did not demonstrate any substantial improvement and she was started on electroconvulsive therapy (ECT). ECT is generally reserved for refractory and prolonged NMS.

Conclusion: Given the widespread use of neuroleptics, all physicians should be able to recognize and manage NMS. Complications from NMS have shown an increased mortality ranging from 10-30%, emphasizing the need for early diagnosis and treatment. Neuroleptic therapy should be started slowly and at incremental doses to prevent NMS.
Identification: 75 year-old man, no known PMH, presented to ER after syncopal episode.

History: He awoke with headache, dizziness, and diaphoresis that he attributed to hangover from alcohol consumption the night before. While reaching into medicine cabinet for analgesic, his girlfriend witnessed him appearing “unsteady on his feet and passing out”. He recovered spontaneously after 1 minute. He felt lightheaded prior to the episode but denied chest pain or palpitations. He had sweats, chills, and SOB for 1 week but denied fever, cough, nausea or dysuria. He incidentally reported 4/10, right-sided, sharp, constant abdominal pain, present since his fall. He does not receive regular medical care. He is a retired laborer with significant alcohol and smoking histories.

Physical Exam and Laboratory Findings: He was afebrile, normotensive (121/87) and slightly tachycardiac (106). He was alert and oriented and had unremarkable pulmonary, CV (except tachycardia), extremity, and neurological exams. Abdomen was TTP in RUQ and RLQ with involuntary guarding, no palpable masses, and no rebound.

Initial CBC and chemistry panels were normal. Serum lactate was 1.8. UA revealed pyuria and bacteria. ECG: Sinus tachycardia, otherwise normal. CXR normal.

Initial Course: Treated in ER with IVF and IV antibiotics for presumed UTI, but dizziness persisted. Repeat laboratories indicated new leukocytosis, new anemia, and worsening serum lactate (6 from 1.8). He became transiently hypotensive and was admitted to ICU.

ICU Differential Diagnosis: Primary considerations were infectious, including urosepsis or intraabdominal abscess. Gastric volvulus, bowel obstruction, bowel/mesenteric ischemia, pancreatitis, and PUD were also considered.

ICU Course: He was hemodynamically stable upon ICU arrival. STAT CT of abdomen showed 9.2 x 9.1 x 12.7 cm infrarenal abdominal aortic aneurysm with active hemorrhage into right retroperitoneum (figure). (a ruptured AAA)

During preparation for emergent surgery, he became progressively hypotensive, requiring emergent resuscitation with IVF, PRBC, vasopressor support. He subsequently suffered PEA cardiac arrest. He was never stable for OR transfer and expired after a prolonged resuscitation attempt.

Discussion: This case illustrates an atypical presentation of ruptured AAA. While this patient’s age, gender, and tobacco use history are risk factors for AAA, and syncope and abdominal pain can herald a ruptured AAA, diagnosis was confounded by findings suggesting an infectious process. He lacked the classic finding of pulsatile abdominal mass.

Conclusion: Misdiagnosis of AAA is common, and AAA rupture carries 80-90% mortality. Classic findings are present in only 25-50% of patients, and over 60% of ruptured AAA are initially missed. This case illustrates the need to maintain a wide differential diagnosis and be alert to atypical presentations of emergent conditions.
Case presentation: This 65 year old Caucasian male presented to his primary care physician with 4 months of fevers, night sweats and 30lb weight loss. Initial work up by the PCP revealed thrombocytopenia and monocytosis. Hematology work up included peripheral smear, CT chest, abdomen/pelvis and bone marrow biopsy which were unrevealing except for an elevated LDH. Subsequently he presented with transient episodes of RUE numbness/tingling, expressive aphasia and word finding difficulties and a negative work up for ischemic and embolic causes. He presented to the hospital for work up of persistent and worsening symptoms.

Physical Abnormalities: Fevers up to 38.7. Speech is soft, and mildly dysarthric in quality. MMSE showed mild cognitive impairment with 1/3 word recall and impaired concentration based on inability to spell "world" backwards. Neurological exam otherwise is unremarkable.

Labs/Imaging: Extensive serum and CNS infectious work up was undertaken which was negative for HIV, Hepatitis B, Syphilis, EBV, CMV, TB, Q fever, Cat scratch, Brucella, Tularemia, HSV, Listeria, Toxoplasma, Coccidioides, Histoplasma, Cryptococcus, Aspergillus, Candida. Serum was negative for ANA, lupus acg, anti RO, anti LA, ANCA. CSF showed highly elevated protein but was otherwise normal, without oligoclonal bands. MRI brain showed non-enhancing multifocal periventricular white matter disease indicating no active demyelination. PET scan revealed extensive hypermetabolic activity predominantly involving the bones but also the pericardium, pulmonary parenchyma, soft tissue nodules, thickened fascia within the retroperitoneum, adrenal glands and duodenal bulb.

Differential Diagnosis: In addition to the aforementioned infectious and rheumatologic etiologies investigated above, malignancy; especially lymphoma was high on the differential. Biopsy of hyper metabolic area of femur provided the diagnosis of Diffuse large B-cell lymphoma, NOS (DLBCL). In the absence of CNS involvement, his neurologic symptoms were thought to be paraneoplastic. Other potential differentials are Infectious mononucleosis, Melanoma.

Discussion: R-CHOP therapy was initiated. DLBCL is aggressive and the most common histologic subtype of Non-Hodgkin’s lymphoma. Approximately 60% of patients are in advanced stage (3 or 4) at diagnosis. Our patient is categorized as high risk with a 5 yr overall survival rate of 26%.

Conclusion: Patient was a diagnostic challenge. In the setting of B symptoms, an elevated LDH, and thrombocytopenia suggestive of a hematological cause the initial evaluation was non-diagnostic. Diagnosis was only made after PET scan showed hyper metabolic areas and biopsy, CNS paraneoplastic symptoms are an unusual presentation of lymphoma and the diagnosis can be delayed or missed.
Introduction: A 50-year-old female with osteoporosis and chronic low back pain presents with a one-week history of abdominal pain and fevers.

Case presentation: Ms. T presents with abdominal pain and intermittent fevers, measured as high as 103 degrees. The pain is described as sharp and severe, located in the lower abdominal quadrants and associated with diarrhea. She also describes bodyaches and diffuse joint pain. Initially, she’d experienced right breast pain with greenish discharge, treated by her Primary Care physician with Keflex. Related to worsening abdominal pain, a CT abdomen/pelvis without contrast was completed, demonstrating inflammation surrounding her abdominal aorta and prompting her current presentation.

She has a complicated medical history including a remote history of encephalitis resulting in infrequent seizures (one event or less per year), as well as poorly recalled episode of “kidney and renal failure” resulting in a prolonged ICU admission. Medications include Fosamax and Lortab PRN. Family history is significant for a mother and maternal grandmother with breast cancer. She has a 30 pack-year smoking history and is a current smoker. She’s sexually active with her boyfriend of the last year and denies previous sexually transmitted infections.

On physical exam, she’s afebrile and other vital signs are within normal limits. Examination of the affected joints is normal with the exception of tenderness to palpation among the TMJ, elbows, hands, knees and sacroiliac joints. She also endorses diffuse abdominal tenderness with mild distention and positive rebound. CRP is 23.5 and Hgb is 9.9 mg/dL. CBC doesn’t demonstrate a leukocytosis and urinalysis is inconsistent with infection.

Parenteral Flagyl and Ceftriaxone is started out of concern of infectious diarrhea. Her CT abdomen is reviewed with Radiology; the preliminary read is aortitis. Overnight, Ms. T decompensates with tachycardia, an elevation in respiratory rate and increasing abdominal pain. She’s seen by General and Vascular Surgery. A CT angiogram is performed, though the patient is taken to the OR for replacement of the infrarenal aorta prior to communication of the results of this study.

Ms. T is admitted to the SICU after her operation and observed without complication for nearly one week. She’s discharged on Vancomycin and Meropenem with plans to continue parenteral antibiotics for a total 6-week course.

Discussion: Epidemiologic studies have had difficulty defining the rates of aortitis, which is better characterized in children. The most common etiologies involve large-vessel vasculitides, namely Giant Cell and Takayasu’s arteritis. Infectious aortitis is most commonly related to infection with Staphylococcal or Streptococcal species, as well as Salmonella. Syphilis, typically involving the thoracic aorta, is now exceedingly rare in developed countries. The typical presentation involves back or abdominal pain, with or without fevers. Complications of aortitis include dissection and rupture of the affected region of the blood vessel. While the gold standard for diagnosis was once angiography, CTA and MRA are now the primary means of establishing a diagnosis. Open reconstruction of the aorta is most often indicated, with endovascular repair showing good promise in recent studies.

Our patient is a 42-year-old Japanese woman with Adult Onset Still’s Disease (AOSD) who was in her usual state of health when she began to develop a nonproductive cough, chest tightness, runny nose, and sinus congestion. Her symptoms evolved to include a pink, lacy rash over her trunk and extremities, malaise, fevers, fatigue, and sore throat.

Physical Abnormalities: She was febrile, hypotensive, tachycardic, and tachypneic. Physical exam was notable for generalized weakness, a swollen and edematous upper lip, diffuse crackles with increased work of breathing, and a salmon-colored coalescing macular eruption over her trunk and extremities.

Lab Results: Leukocytosis with bandemia, anemia, and normal platelets were noted. Chest x-ray revealed bilateral interstitial opacities consistent with multifocal pneumonia.

Differential Diagnosis: The presumed diagnosis was septic shock due to atypical pneumonia. Due to immunosuppression from treatment for Still’s Disease, atypical organisms, such as Pneumocystis Jiroveci, were considered. Methotrexate-induced pneumotoxicity was also postulated as a contributing factor, and her scarlatiniform rash and sore throat suggested the possibility of a group A streptococcal infection progressing to pneumonia.

Case Presentation: She received several antibiotics, improved clinically, and was discharged. Then, 6 days later, she collapsed at home and was brought back to the hospital. In the emergency room her hemoglobin was 6.6, her platelets were 67, her AST was 1225, and her LDH was 29,838. Her erythematous rash evolved into Stephens-Johnson Syndrome with...
Case Presentation:
Patient is a 52-year old female s/p colectomy and ileostomy who presented with 3 days of fatigue, confusion, and muscle jerking. The patient reported a history of fatigue and hyperammonemia that began 6 months post-surgery that had been refractory to lactulose and rifaximin. Previous liver biopsy was without evidence of cirrhosis. The patient also endorsed a history of chronic UTIs, which interestingly when treated with antibiotics transiently improved her fatigue and hyperammonemia. She recently completed a course of ciprofloxacin 3 days prior to the onset of her presenting symptoms.

Physical Exam:
On admission to the MICU the patient was encephalopathic with asterexis on exam. Abdominal exam was notable for multiple scars and ileostomy. Labs were notable for WBC of 18.17, 69.3% granulocytes, serum ammonia of 187 and ABG with pH of 7.189, PaCO2 29.1, PaO2 89.6, and HCO3 10.7 with lactate of 0.7. Urinalysis, urine culture, and blood culture were without evidence of infection, despite leukocytosis. Citrulline was low normal at 12 and urine orotic acid was normal. The patient’s branched-chain amino acids threonine, valine, and isoleucine were also decreased. Serum vitamin B12 and methymalonic acid were normal. Fecal fat was increased.

Differential Diagnosis:
Given the onset of the patient's symptoms 6 months following colectomy and ileostomy and worsening of symptoms following antibiotic cessation it was determined that the most likely cause of hyperammonemia in the patient was small bowel bacterial overgrowth, supported by...

Differential Diagnosis:
Given the onset of the patient's symptoms 6 months following colectomy and ileostomy and worsening of symptoms following antibiotic cessation it was determined that the most likely cause of hyperammonemia in the patient was small bowel bacterial overgrowth, supported by malabsorption and increased fecal fat. Bacterial overgrowth leads to elevation of propionic acid via breakdown of branched chain amino acids in the gut, resulting in decreased levels of isoleucine, valine, and threonine. Propionic acid inhibits the synthesis of N-acetylglutamate, an obligatory activator of carbamoyl phosphate synthetase I (CPS I), resulting in transient inhibition of the urea cycle. This leads to elevated ammonia, low-normal citrulline, and normal urine orotic acid. Furthermore, propionic aciduria leads to metabolic acidosis despite elevated ammonia levels. Vitamin B12 deficiency can also cause methylmalonic aciduria as well as propionic aciduria further worsening inhibition of CPS I.

Treatment & Conclusion:
The patient was treated with intramuscular vitamin B12 to prevent methylmalonic aciduria as well as a 30-day course of Augmentin for small bowel bacterial overgrowth with resolution of her symptoms. This case demonstrates the importance of considering bacterial overgrowth in patients s/p small bowel surgery as an etiology of hyperammonemia and concurrent metabolic acidosis.
CASE PRESENTATION: Patient is a 22 yo woman with past medical history significant for celiac disease who was in otherwise good health until she developed flu-like symptoms along with her boyfriend which persisted and became worse over the course of a three week period despite her boyfriend's symptoms resolving. Two weeks after initial symptoms, she was sent to the health clinic from a lecture by her professor at BYU for looking "yellow." She took homeopathic medications for one week during which her symptoms expanded to darkened urine, nausea, vomiting, and finally epistaxis. She went to an OSH ER where ENT evaluated her and diagnosed her with a deviated nasal septum. Later, her labs indicated that she was in fulminant liver failure with a MELD of 33. While in the ER, she developed hypotension, encephalopathy, and respiratory failure necessitating pressors and intubation.

EXAM ABNORMALITIES: Initial physical exam was concerning for jaundice and evidence of upper extremity posturing. CBC showed a leukocytosis 25, Hgb of 8.0, thrombocytosis of 700. CMP revealed a Sodium of 135, Cr of .56, Glucose of 30, AST of 915, ALT of 464, TBilirubin of 17.4, AP of 107. Hepatitis viral panel was negative. EBV, HTLV, and HIV were negative. Ferritin was 383, A1- Antitrypsin was low at 62, F-Actin was high at 63, Liver-Kidney Microsomal was normal, Ceruloplasmin was 8 (low), free Serum Copper was low at .47.

DIFFERENTIAL DIAGNOSIS: When the initial set of labs returned, there was concern for autoimmune hepatitis v. Wilson's Disease (WD). The patient was too unstable to undergo liver biopsy so work up Wilson's Disease continued and we ordered a 24 hr Urine Copper, which was 1074 ug/dl (typical WD cutoff is >100 ug/dl). Wilson's became our working diagnosis.

TREATMENT: Both transplant surgery and the liver service believed that transplant was the ultimate treatment for the patient's fulminant liver failure in the setting of WD. Unfortunately, the patient developed ARDS and subsequent DAH in the setting of RSV infection. The patient’s poor neurological status secondary to hyperammonemia and prolonged hypoxia also precluded from being added to the transplant list. The patient eventually had a cerebellar herniation and was placed on comfort care where she later expired. Autopsy confirmed the diagnosis of WD.

CONCLUSION: Wilson's Disease is a rare autosomal recessive disease. Fulminant liver failure is most commonly seen in young females, where rapid diagnosis is essential to improving survival.
Case Presentation: A 68-year old male with a history of systemic discoid lupus presented to the hospital with two weeks of non-bloody diarrhea, worsening AMS and diffuse body pain. He had a similar hospitalization last year, infectious work up was negative at that time but symptoms did improve with Prednisone. Since then he has been maintained on Prednisone 5 mg, Hydrochloroquine, Azathioprine and Gabapentin. No recent changes in medications, antibiotics, sick contacts or travel. In the ER, his temperature was 40 C and had a WBC count of 18.1 K/uL with 6% bands.

Physical Exam Abnormalities: He was alert, but not oriented to place or time. Exam was notable for purpura and hyperpigmentation in bilateral lower extremities, swan neck deformities in fingers and sclerodermatous changes in his hands. There was diffuse tenderness in all joints, however, no excessive warmth or erythema.

Differential Diagnosis: DDx included Sepsis versus Connective Tissue Disease Flare. ID and Rheumatology were consulted. ESR and CRP were elevated at 109 and 15.2, respectively. All infectious work up including bacterial and fungal cultures, viral panels, TB testing, C Diff PCR and Brucella and Coxiella serologies were negative. Imaging of the patient, including CXR, CT abdomen and pelvis, US of lower extremities and MRI of the thoracic and lumbar spine were WNL. A lumbar puncture and bone marrow biopsy were ordered and were WNL.

Treatment: The patient remained hospitalized for 9 days. Initially he was treated with empiric IV Primaxin and Prednisone 5 mg Qday. Despite treatment, he continued to spike high grade fevers and had a WBC count as high as 27.7 K/uL. After infectious etiologies were ruled out by hospital day seven, he was started on Prednisone 40 mg and Hydrochloroquine. The last three days of hospitalization the patient remained afebrile and the WBC count dropped to 15.8 K/uL. He was discharged with a ten day supply of 40 mg of Prednisone and was scheduled to see Rheumatology seven days after discharge.

Conclusion: Fever of unknown origin (FUO) is often misused to describe a patient with a fever with no identifiable source. This patient, however, meets the criteria by having an unclear diagnosis after one week of work up in the hospital and multiple temperatures >38.3 C. The most common causes of FUO include infections, noninfectious inflammatory diseases and malignancy, with connective tissue diseases representing 22% of the cases.
Identification/Chief Complaint: 48 year old man without prior medical history presents for work-up of new heart murmur.

History: Patient presented from an urgent care clinic following the instructions, “Tell the doctors you have a new murmur.” In addition, he reported he had been feeling ill for approximately 4 weeks prior to presentation, with malaise and night sweats which were increasing in frequency. He also had shortness of breath which he described as “if something heavy” sits on his chest, but denied weight change, peripheral edema, orthopnea or dyspnea on exertion. He did not have any cough, upper respiratory symptoms, dysuria, or nausea/vomiting. He had no history of intravenous drug use or recent dental procedures.

Physical Abnormalities: Initial exam was notable for low grade temperature of 99.9F, poor dentition and III/V holosystolic murmur heard throughout precordium. No Roth’s spots, Janeway lesions, Osler’s nodes or splinter hemorrhages were noted.

Lab Results: Complete blood count revealed mild leukocytosis with WBC of 10.3 with normal differential, anemia with Hgb 11.6, and peripheral smear revealed Dohle bodies. Chemistry panel was unremarkable, cardiac enzymes were negative, and coagulation studies were normal.

Imaging Results: Chest x-ray revealed LLL consolidation, CTA of chest with LLL consolidation and reactive mediastinal lymphadenopathy.
Identification: 56-year old Caucasian male

Chief Complaint: Involuntary lower extremity shaking

Case Presentation: Patient is a 56-year old Caucasian male who presented after 2-weeks of sudden onset of involuntary bilateral lower extremity tremors, fine motor skill impairment, gait abnormalities requiring the use of an aid, and memory loss. His past medical history is significant for chronic pain following a cycling accident 8-years prior, severe depression with suicidal ideations, anxiety, and insomnia. He has had to take a leave of absence from work. At presentation, he was treated with Gabapentin, Methadone, Oxycodone-Acetaminophen, Mirtazapine, Venlafaxine, Bupropion, Ibuprofen, Diazepam, and Zolpidem.

Physical Abnormalities: Intermittent bilateral lower extremity tremor that was distractible with variable rate and amplitude. Weakness due to the tremors in the proximal upper and lower extremities. Generalized hyperreflexia. Unsteady gait with astasia-abasia.

Lab Results: CMP, CBC, TSH, B12/Folate within normal limits. MRI of the brain, C/T/L spine demonstrated moderate cervical stenosis that was unchanged from prior evaluation.

Differential Diagnosis: Conversion Disorder
Drug-Induced Tremor

Treatment Course: The diagnosis of conversion disorder was made. The patient was seen frequently, initially at one-week intervals. Venlafaxine was discontinued but the patient’s tremors and mood were unchanged. Over time, the patient disclosed a history of sexual abuse as a child. Following this disclosure, the patient’s tremors improved to where there were absent while in his home, but present anytime he left home and continued to be debilitating. His tremor worsened when discussing his experiences. Following four more months of frequent visits with his primary care physician, therapist and psychiatrist, the patient’s tremors and mood were unchanged and failed to demonstrate further improvement. The patient requested inpatient opiate and benzodiazepine detoxification. While undergoing inpatient detoxification, his gait rapidly normalized, his tremors disappeared, and his mood improved significantly. Five months since detoxification the patient is now being treated only with Gabapentin and Bupropion, ambulates without aid, denies any depression, and has returned to work fulltime.

Discussion: This case describes an instance of conversion disorder that was successfully aborted with opiate and benzodiazepine detoxification. The role of drug dependency and polypharmacy in conversion disorder is unclear, but attempting to wean controlled substances might be considered as a treatment option in select patients. Further inquiry is needed in this area.
CASE PRESENTATION: Patient is a 64-year old male admitted to a local hospital with one week history of dyspnea, productive cough, and pleuritic chest pain. He had sepsis at presentation and had multilobar pneumonia and streptococcus pneumoniae bacteremia. He was placed on Ceftriaxone and transferred to our facility for further management. He remained febrile despite appropriate antibiotic therapy. He also developed herpetic lesions in his oral cavity secondary to his critical illness and was pancytopenic. Patient had no medications and an unremarkable social history. The patient’s son had been diagnosed with NHL in his 30s.

Patient’s repeat urine and blood cultures remained negative. 2D-Echo did not reveal cardiac valvular vegetation. A repeat CT chest showed nonspecific pulmonary nodules and reactive lymphadenopathy and no significant pleural effusion or lung abscess. In the last seven years, the patient reported frequent respiratory infections despite receiving the pneumococcal vaccine. Given this, an evaluation for immunocompromising condition was undertaken. His quantitative IgG, IgA and IgM were undetectable. A peripheral leukemia/lymphoma phenotyping showed no evidence of malignant process. Bone marrow biopsy was negative for leukemia/lymphoma or hemophagocytic cells. SPEP showed no monoclonal gammopathy. LDH and B2 microglobulin were normal. HIV by PCR was negative. C3/C4/CH50 testing was normal. The CT chest did not reveal an enlarged thymus gland. Ferritin, fibrinogen and triglycerides were normal. There was no evidence of liver disease or nephrotic syndrome.

PHYSICAL ABNORMALITIES: In general the patient was alert, but dyspneic. There was no sinus tenderness. Nares were patent and non-edematous without discharge. No palpable adenopathy. There were herpetic lesions on tongue and oral mucosa, tachycardia, tachypnea, bibasilar pulmonary crackles and coarse breath sounds R>L, and labored respirations.

DIFFERENTIAL DIAGNOSIS: The leading diagnosis was hypogammaglobulinemia secondary to late-onset CVID. Differential included Multiple Myeloma, Waldenstrom’s Macroglobulinemia, Leukemia/lymphoma, HIV infection, Wegener’s Granulomatosis, Hypoalbuminemia due to nephrotic syndrome, liver disease or protein losing enteropathy, HLH (Hemophagocytic lymphohistiocytosis) and Good syndrome.

TREATMENT: Two rounds of IVIG were administered with a goal to maintain total serum IgG levels >500mg/dL, shortly thereafter the patient deferevesced. Patient remained on Ceftriaxone for two weeks after last positive blood culture.

CONCLUSION: Common variable immune deficiency (CVID) is a primary immunodeficiency with an estimated incidence of 1 in 50,000 people. Patients are susceptible to frequent and recurrent sinus and lung infections secondary to hypogammaglobulinemia, chronic lung disease, autoimmune disease, gastrointestinal and liver disorders, granulomatous infiltration, splenomegaly, and an increased risk of malignancy.
**Case Presentation:**

Patient is a 41 y/o female who presented to hospital after vacationing in Honduras. Days 1-8 - patient exposed to mosquitoes and sand flies. Day 2 - patient presented to Honduran physician with diarrhea and insect bites and received 5 days ciprofloxin and an “itch” injection. Mid-trip patient participated in snorkeling and fresh water fishing. Day 6 - patient bitten on hand by white-faced monkey with no medical attention. Day 7 - patient developed fever, malaise, hand pain, and re-presented to Honduran doctor where she was prescribed dicloxacillin. Day 8 - fevers persisted, joint pain and headaches developed and she returned to USA. Day 11 - patient presented to the ER complaining of fever, abdominal pain, arthralgias and headache. No anti-malarial or vaccines were taken prior to travel.

**Physical Abnormalities:**

Exam was notable for temp 38.5, hypotension, insect bites on legs, teeth marks between 1st and 2nd digits right hand, and abdominal pain with guarding. Labs on second day admission demonstrated WBC of 7.2 with 32% bands, 29% poly, 31% lymphocytes with atypical lymphocytes on smear. Hb 11.8, PLTs, inflammatory markers, chemistry and lfts were normal.

**Differential Diagnosis/Discussion:**

Initially Ms F’s febrile illness was thought most likely due to Dengue, Malaria or typhoid. Exposure to monkey increased her risk for rabies or herpes B virus. Other considerations included Chagas disease, leptospirosis, HSV, and CMV. During hospitalization malarial blood smears were negative, dengue serologies were negative, and blood/stool cultures were negative. CSF analysis and imaging of Abdomen/Pelvis/Brain were unrevealing. Patients clinical course remained…

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**Differential Diagnosis/Discussion:**

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**Conclusion:**

Although CMV is not an uncommon travel related infection, its ability to cause a profound inflammatory and destructive response in an otherwise immuno-competent host is rare. Current guidelines do not give clear direction on when, or if, pharmacotherapy should be initiated for CMV infections in immuno-competent hosts although research would be helpful in treatment strategies for future cases of severe infections.
IDENTIFICATION: 59 y.o. female with fever of unknown etiology

HISTORY: Patient is a 59 y.o. female in who presented to her PCP with over one week of headache, dry cough, fevers. She was started on antibiotics, which were broadened with persisting symptoms. A CT was obtained which suggested a multifocal pneumonia vs. septic emboli and she was admitted to the medicine service. During the CT, she developed shortness of breath which resolved with Benadryl and Solumedrol. Upon admission, her antibiotic coverage was broadened and workup for infectious etiology continued. Despite the extensive antibacterial treatment, she continued to fever above 38.7, and was transferred to IMC.

PHYSICAL ABNORMALITIES: At the time of transfer, she was still having fevers and feeling diaphoretic. Otherwise not ill appearing and in no acute distress. Notably: T 38.7, tachycardic to 110’s, normal BP and room air sat

Normal physical exam except as noted below:
• GEN: diaphoretic, warm to touch
• CARD: tachycardic, no appreciable murmur or rubs
• PULM: coarse throughout, no wheeze,
• SKIN: small scale on palms and R ankle with patch of discolored and desquamated skin

LAB RESULTS: Workup from the initial hospital included:
• CBC w/ WBC of 13. The Differential was lymphocyte predominant. No other atypical cells
• TEE without evidence of vegetation

Admit labs of note: elevated eosinophils on differential

DIFFERENTIAL DIAGNOSIS: Our initial differential included infectious, malignant, rheumatologic and drug reactions. We reviewed the data already obtained, and tested for fungal, viral, mycobacterial and bacterial causes. We sent serum markers for rheumatologic and inflammatory causes. Malignancy was considered with plan to scan if the other studies were negative.

CASE PRESENTATION: We felt it would be safe to hold abx and observe her in the hospital. With the pulmonary findings on CT, a bronchoscopy was the next important diagnostic test. Of note, the last CBC w differential had a high number of eosinophils. The bronchoscopy was performed, and she remained afebrile until Bronch results revealed an eosinophil count of > 30, helping confirm diagnosis of acute eosinophilic pneumonia. Her symptoms resolved within a day of starting steroids.

CONCLUSIONS: Acute Eosinophilic Pneumonia is a rare condition that can present as a fever of unknown etiology. Pulmonary symptoms and imaging abnormalities often result in non-therapeutic antibiotic treatments. The steroids she received after her CT scan likely masked her eosinophilia on the initial CBC w/diff. Willingness to reevaluate is essential in diagnosing and providing appropriate therapy.
Chief Complaint: “Head fog”

History: 65 year-old female heavy smoker with a history of peripheral artery disease, a benign neck mass, and hypertension treated with hydrochlorothiazide that presents with confusion and fatigue feeling increasingly depressed, lethargic and irritable. Patient endorses decreased appetite, with nausea and constipation. Patient reports an intentional 45 pound weight loss in the last 4 months from supplements she has been taking.

Physical Abnormalities: Patient is confused, not oriented to time or event. Patient is afebrile with >20mmHg discrepancy in BP between arms. Patient is tachypnic and requiring 2L of O2. Bilateral carotid bruit. 2/6 systolic murmur. Lungs are clear, abdomen is soft, non-tender hypoactive bowel sounds. Weak pulses. Cranial nerves intact with bilateral ptosis. Hyperreflexive symmetrically.

Labs:
- I onized Calcium: 2.47 Calcium: 18.6
- Phosphorus 2.8, Magnesium 21 Potassium 2.9 BUN 37 Cr 1.4
- PTH 11 ng/mL; PTHrp, TSH, Vit D and A: all wnl
- UPEP/UPEP: non-selective proteinuria without monoclonal spike
- No leukocytosis or anemia.

Differential Diagnosis: Hypercalcemia secondary to malignancy (lung carcinoma with 50-pack year, weight loss, and hypoxia or breast cancer, multiple myeloma or leukemia. Also possible iatrogenic with thiazide, hypervitaminosis D, A or calcium. Considered primary and secondary hyperparathyroidism, milk-alkali syndrome, sarcoidosis, paget’s disease or familial hypocalciuric hypercalcemia.

Case Presentation: 65-year-old female with an extensive past smoking history and hypertension who initially presented with confusion and fatigue and was found to be severely hypercalcemic. An extensive workup was performed with a low PTH and high calcium suspicious for a paraneoplastic process. Negative UPEP/SPEP and no abnormalities on imaging (CT chest, neck, brain and mammogram) other than a small 7 mm nodule in the lung but with negative PTHrp. Vitamin D, A, and urine calcium were not elevated. Patient’s hypercalcemia was felt to be due to her thiazide use in conjunction with exogenous and excessive supplementation with up to 3.8 grams of daily elemental calcium intake. Patient was treated with IV fluids and bisphosphonates. Thiazide and herbal supplementation was discontinued with normalization of labs and symptoms.

Conclusion: Calcium is a critical ion involved in cellular transport, membrane function and bone metabolism. Severe hypercalcemia can lead to fatigue, constipation, mental status changes, coma and cardiac arrest. Elevated calcium levels can be secondary to chronic renal failure, endocrine aberrations in PTH axis, or a result of medications such as thiazide or lithium. They can also reveal underlying malignancy. Over-the-counter supplementation is often overlooked as both the cause and complication of disease.
**Case Presentation:**
The patient is a 31 year old female who presented to the pulmonary clinic at the Veterans Medical Center for evaluation of chronic, unrelenting cough with sputum production. Patient reported that over the past 10 months she had repeat visits to her primary physician as well as urgent care for productive cough with fevers. She had at least 3 different courses of antibiotics for treatment including a “z-pack” without significant improvement. She was diagnosed with asthma by in-office spirometry 6 months prior and started on albuterol and mometasone without improvement. She denied fevers or night sweats but had a 17 pound unintentional weight loss over the course. She continued to have productive cough with “foul” sputum at presentation. She notes a “positive TB skin test” while stationed in Korea with the Army for which she was treated with “a single pill” for 6 months with a follow-up chest radiograph which she assumes was negative.

**Physical Abnormalities:**
In general, she appeared uncomfortable and coughing produced sputum during exam. Room air oxygen saturation was 95%. She had no palpable lymphadenopathy or splenomegaly. Moist apical crackles were auscultated bilaterally with tactile fremitus noted mostly on the left. No wheezing was noted. PFT performed prior to presentation illustrated moderate airway obstruction without bronchodilator response and mild reduction in DLCO. A CT of the chest illustrated multiple cavitary nodules in the left lung and bilateral apices as well as extensive tree-in-bud opacities.

**Differential Diagnosis:**
Infectious considerations with cavitary lesions on CT include fungal infections such as histoplasmosis or coccidiomycosis. Atypical bacterial infections with tuberculosis, nocardia and actinomyces or septic emboli from endocarditis could also be considered. Other non-infectious etiologies could be vasculitis or sarcoidosis. However, with her history of previous TB exposure, a reactivation of tuberculosis was most likely. Acid staining of sputum cultures were positive for tuberculosis.

**Discussion:**
After 12 weeks of rifampin, isoniazid, pyrazinamide and ethambutol, clearance of her sputum was noted. A nine month course of isoniazid and rifampin is in progress. At her infectious disease follow-up visit she was noted to be improving in regards to her weight loss, sputum production and cough.

**Conclusion:**
This case highlights findings by Mancuso et al where long-term, overseas deployment in TB endemic areas carries an increased risk of active TB infection. In a patient with chronic, productive cough and weight loss with history of overseas deployment, active TB should be a consideration.
The patient is a 77 year old female with heart failure with preserved ejection fraction, atrial fibrillation on warfarin, diabetes, and hypertension who presented with acute onset of nausea, vomiting, vertigo, and one episode of diarrhea. She was recently hospitalized for heart failure exacerbation and recently increased her oral hyperglycemic agents resulting in low fasting glucose, presumably causing her increased somnolence. In the ED, a CT abdomen showed cholelithiasis and a left lower lobe consolidation, CT brain without contrast showed no acute intracranial abnormality. Chest x-ray showed cardiomegaly but no evidence of consolidation.

**Physical Abnormalities:** Somnolent but arousable, generally weak, but no focal deficits. No Nystagmus. Lungs had few fine crackles in left base, no fremitus. Cardiovascular: irregularly irregular. Abdomen: +BS, soft, non-tender

**Lab Results:** WBC 17.7, no bands, Sodium 141, Bicarbonate 26, BUN/Cr 32/1.3, Lactate 1.8, LFTs, lipase, troponin and BNP were normal. INR 1.3

**Differential Diagnosis:** Presumed diagnosis was Left lower lobe Pneumonia (likely secondary to aspiration) and acute renal failure. Bowel ischemia was considered with her cardiac history and atrial fibrillation, but since she did not have any abdominal pain it was unlikely. CT abdomen and labs helped rule out other acute abdominal or cardiac illnesses. Her somnolence and weakness were presumed to be secondary to her acute illness.

**Case Presentation:** The next morning the patient developed truncal weakness and left arm chorea. MRI brain and neck showed evidence of acute left cerebellar stroke with mass effect against the posterior aspect of the left brain stem and mild effacement of the lateral aspect of the fourth ventricle. Neurology transferred her to the Neuro ICU. She eventually required a craniectomy to relieve the pressure.

**Discussion/Conclusion:** If we re-organized the patient’s one liner: 77 year old female with multiple co-morbidities including atrial fibrillation, diabetes and hypertension who presented with acute onset of vomiting, vertigo and a subtherapeutic INR. Now it becomes obvious that the differential should have included stroke or complications secondary to thrombosis. Imaging lead us astray in this setting. The consolidation on CT of the abdomen was not evident on CXR, which was dismissed as different modalities and sensitivities. However, we should have taken a step back to think: What are we missing? Pneumonia did not encompass all of her symptoms. We dismissed somnolence and weakness in an elderly woman, until the focal deficits became evident.
Chief Complaint: 59 year-old otherwise healthy male presents with fevers, chills, night sweats and cough of four weeks duration.

History: The patient lives in rural Wyoming in close proximity to livestock and wildlife. He travels extensively for his consulting job as a structural engineer, including to Korea, Jamaica, Kazakhstan and nearly every state in the country.

His symptoms started acutely, and were accompanied by shortness of breath. He was initially evaluated at an urgent care and diagnosed with pneumonia, for which levofloxacin, clarithromycin and prednisone were prescribed. He takes no other medications.

Fourteen days into his illness he continued to experience biquotidian fevers exceeding 40°C, soaking sweats, severe arthralgia, and a worsening non-productive cough without hemoptysis. He developed painful right inguinal nodules that remitted spontaneously. After three weeks of persistent symptoms he was referred to Salt Lake City.

Physical Abnormalities: The patient was febrile at 38.9°C, tachycardic but normotensive. He did not appear toxic, was communicative and had no meningismus. Bilateral anterior cervical lymphadenopathy was palpable. Coarse breath sounds were present but no murmurs were audible. The right inguinal nodules were barely palpable.

Lab Results: CBC, basic chemistry, urinalysis and liver function tests were all normal. Chest x-ray demonstrated bilateral “buckshot” consolidation. CT Chest showed multiple, bilateral nodules with mediastinal lymphadenopathy.

Differential Diagnosis: The initial differential was broad, and included tuberculosis, endemic mycoses, cryptococcosis, psittacosis, Q-fever, legionellosis, HIV complicated by pneumocystis pneumonia, hypersensitivity pneumonitis, cryptocogenic organizing pneumonia, lymphoma, sarcoid and granulomatosis with polyangiitis.

Additional Work-up: Blood cultures, multiple serological tests, bronchoscopy with BAL and CT-guided lymph node biopsy were performed.

Additional History: The patient reported that he is an explosive demolition expert, and that 10 days prior to symptom onset he had demolished a 100-year old smoke stack in western Pennsylvania that was filled with debris, dead birds and excrement.

Video of the demolition: http://youtube/wgF5VmLpzOc

Diagnosis: Urine and plasma antigen tests for Histoplasma capsulatum were positive. The patient was successfully treated with oral itraconazole.

Conclusion: Histoplasma capsulatum is a soil-based dimorphic fungus endemic to the Midwest and Southeast. It is associated with soil contaminated by avian or bat guano, where more than 10^5 particles may be present per gram of soil. Infection occurs after inhalation, and is most commonly a self-limited influenza-like illness in immune-competent hosts. Disseminated disease is more common in immune-compromised patients, especially HIV, but infrequently occurs in otherwise healthy individuals. Treatment with itraconazole for persistent mild-moderate disease, or amphotericin B for progressive disease, CNS disease, and in immune-compromised hosts is generally effective.
OPENING REMARKS

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VIGNETTE COMPETITION JUDGES
Leslie Lenert, MD
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'VERTIGO'
Down the Rabbit (or Ear) Hole

Matthew E. Feurer, MD, MS
Utah Chapter American College of Physicians
Annual Clinical Vignette Competition
May 16th, 2013
CASE PRESENTATION

HISTORY

CC: Dizziness

HPI: 89 yo male veteran

Long standing hx of vertigo, COPD, CAD, afib

and previous bilateral mastoidectomies

Presents with a two day history of progressive, worsening dizziness.

*Room spinning* and difficulty focusing vision, difficulty with ambulation and inability to walk more than a few steps

*Exacerbated by standing up, head movements or bending over.*

Recently given prednisone by home health for "lung tightness" which made dizziness worse. Denied syncope or lightheadedness.

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**Physical Exam**

- GEN: Lying in bed, NAD
- HEENT: PERRLA, bilateral hearing aids, no nystagmus.
- NEURO: no focal deficits. poor balance, positive Romberg and a left foot drop.

- EKG with first-degree AV block unchanged from prior.

- *Ear canals were not initially examined at admission as*
  1) An otoscope was not readily available, and
  2) Hearing aids were in place.
  3) He denied ear pain or hearing loss.
Differential Diagnosis

Dizziness

- Dizziness accounts for an estimated 5 percent of primary care clinic visits.
- The patient history can generally classify dizziness into one of four categories

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
<th>% of patients with dizziness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertigo</td>
<td>False sense of motion, possibly spinning sensation</td>
<td>45 to 54</td>
</tr>
<tr>
<td>Disequilibrium</td>
<td>Off-balance or wobbly</td>
<td>Up to 16</td>
</tr>
<tr>
<td>Presyncope</td>
<td>Feeling of losing consciousness or blacking out</td>
<td>Up to 14</td>
</tr>
<tr>
<td>Lightheadedness</td>
<td>Vague symptoms, possibly feeling disconnected with the environment</td>
<td>Approx 10</td>
</tr>
</tbody>
</table>

Causes of Vertigo

Peripheral causes (80% of cases)

- Benign paroxysmal positional vertigo
- Vestibular neuritis
- Meniere disease
- Herpes zoster oticus
- Labyrinthine concussion
- Perilymphatic fistula
- Acoustic neuroma
- Aminoglycoside toxicity
- Otitis media

Central causes

- Migrainous vertigo
- Brainstem ischemia
- Cerebellar infarction and hemorrhage
- Chiari malformation
- Multiple sclerosis
- Episodic ataxia type 2

Because they generally are not helpful diagnostically, laboratory testing and radiography are not routinely indicated in the work-up of patients with dizziness when no other neurologic abnormalities are present.
Making a Diagnosis

• Given his prior history of vertigo, ER physician suggested this was a worsening of his baseline symptoms induced by prednisone in the setting of polypharmacy.

Bias in decision making
  – Availability bias – thinking that a similar recent presentation is happening in the present situation.
  – Diagnosis momentum – accepting a previous diagnosis without sufficient skepticism.

• Being skeptical, further chart review revealed patient had multiple clinic visits for a chronic ear infection. Although uncommon, otitis media has been associated with vertigo.

• Returning with an otoscope …

What’s that in your ear??

Curiouser and curioser!
Case Highlights

• Vertigo and dizziness are considered among the most common of symptoms in medicine. Conditions associated with vertigo can be diagnosed by careful history and physical exam alone without the need for extensive testing.

• Diagnostic errors can arise through cognitive errors, especially those associated with failures in perception and biases. This type of error can be reduced by stepping back from the immediate problem to examine and reflect on the thinking process.

References

A Little Swelling Goes a Long Way

Natalie Rodden, PGY 1
ACP Clinical Vignette 5/16/2013

“My, what swollen legs you have!”

History of Present Illness:

64-year-old Hispanic female with a history of hypertension, hyperlipidemia, GERD and hypothyroidism referred to internal medicine clinic from podiatry for further evaluation of lower extremity swelling.
HPI Continued...

- Presented with numbness and burning pain in her arms and legs, worse distally, for which she was taking ibuprofen.
- Swelling in her legs was progressively worsening over the last several months.
- No prior history of renal, hepatic, or cardiac disease.
- Denied dyspnea upon exertion, orthopnea, paroxysmal nocturnal dyspnea or abdominal pain.

History

MEDICAL: hypertension, GERD, HLD, hypothyroidism, degenerative joint disease, depression

SURGICAL: None


FAMILY: Denies cancers, heart or kidney disease

MEDICATIONS: lisinopril, HCTZ, acetaminophen, levothyroxine, duloxetine, celebrex, ibuprofen

REVIEW OF SYSTEMS: Chronic mild lower back pain. Decreased energy, depressed mood. Urine had a foamy appearance.
**Physical Examination**

**VITAL SIGNS:** Temperature 36, blood pressure 130/87, heart rate 67, respiration 16, Pulse ox 96%, BMI 35, weight 81.7 kg (77 kg 3 months prior)

**GENERAL:** Pleasant elderly lady who is teary, in mild distress, alert and oriented x3.

**HEENT:** Extraocular muscles are intact. Pupils are equally round and reactive to light and accommodation. Clear oropharynx, moist mucous membranes.

**NECK:** Supple, with no lymphadenopathy no JVD, no thyromegaly.

**CARDIOVASCULAR:** Regular rate and rhythm. Normal S1 and S2. No murmurs, rubs, or gallops.

**PULMONARY:** Clear to auscultation bilaterally. No crackles or wheezes.

**ABDOMEN:** Soft, nontender, nondistended, positive bowel sounds, obese, no organomegaly. No hepatojugular reflux.

**EXTREMITIES:** 2+ pulses bilaterally, 2+ pitting edema in lower extremities bilaterally to the mid-thigh

**SKIN:** Warm, no rashes or lesions.

**NEUROLOGIC:** Cranial nerves II-XII are grossly intact. No focal deficits, normal gait, arm and leg tenderness to palpation distally. Normal sensation to light touch, pinprick, and vibration.

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**Initial Laboratory Data**

**CBC:** WBC 6.2 Hgb 13.6 Hct 38.8 Plts 331

**BMP:** Na 126, K 4.8, Cl 97, CO2 28, BUN 13, Crt 0.66

**LFTs:** TP 3.7, Albumin 1.5, Tbili <0.1, AST 33, ALT 33, Alk Phos 90

**U/A:** 100+ protein, 7 WBC, 1 eosinophil, specific gravity 1.006

Urine spot total protein:creatinine ratio: 12

**Lipid panel:** TC 512/TG 148/HDL 71/LDL 411
Return Visit, 1 Week Later

History of "foamy urine," heavy proteinuria, hypoalbuminemia, edema, hyperlipidemia...NEPHROTIC SYNDROME!

• Recommended fluid restricted diet, NSAID avoidance, and nephrology referral.

Further lab studies ordered:
• ASO negative
• C₃, C₄ not suppressed
• ANA, anti-dsDNA ab negative
• C-ANCA negative
• anti-GBM negative
• Hep B, Hep C serologies negative
• SPEP: 2 monoclonal spikes with kappa (IgG heavy chain) and 2 faint monoclonal lambda light chains (beta and gamma). Total gammaglobulins appeared decreased.

• UPEP: without spikes but immunofixation showed monoclonal IgG lambda light chains.

Renal Biopsy

H & E Stain x 400

E M
Congo red positive amyloid deposits in the glomeruli and vessels, consistent with lambda light chain AL type

Bone Marrow Biopsy

- Bone marrow biopsy showed normocellular trilineage marrow with mild plasmacytosis (few monoclonal lambda plasma cells).

- Skeletal survey demonstrated no destructive or lucent lesion.
Amyloidosis

- Pathologic tissue deposition of a protein with an altered structure that polymerizes into an insoluble form.
- AL amyloid ("primary") = most common form. Precursor protein is monoclonal light chain, usually lambda. Monoclonal plasma cell proliferative disorder.
- Up to 20% with AL have coexisting multiple myeloma or other lymphoproliferative disease.
- Epidemiology: 6-10/million/year. Median age at diagnosis: 64. Male 65-70% cases.

Clinical Presentation

- nephrotic range proteinuria
- edema
- hepatosplenomegaly
- heart failure (restrictive cardiomyopathy)
- carpal tunnel syndrome
- peripheral neuropathy
Diagnosis

- Histologic confirmation is required. Fat pad or kidney biopsy.
- Kidney biopsy
  - Light microscopy -- amorphous material effacing varying proportions of glomeruli, vessels, or interstitium.
  - Polarized microscopy -- green birefringence on Congo red stain.
  - Electron microscopy -- amyloid fibrils
- Evaluation required for underlying multiple myeloma

Treatment

- Autologous hematopoietic stem cell transplantation (HCT)
- Standard treatment for those eligible is high dose (myeloablative) melphalan chemotherapy followed by HCT.
- Not HCT candidate: low-dose melphalan and dexamethasone
- Newer agents bortezomib and lenalidomide are being evaluated in clinical trials
Criteria for Transplant

- Age ≤ 70
- Trop < 0.06
- NT-proBNP < 5000
- CrtCl ≥ 30
- ECOG ≤ 2
- NYHA functional status I or II
- No more than 2 organs significantly involved (liver, heart, kidney, autonomic nerves)
- No large pleural effusions
- No dependence on oxygen therapy

Prognosis with Treatment

Cibeira et al, Blood 2011

- 421 patients with AL amyloid treated with high-dose melphalan and HCT
- 81 patients died within 1st year (overall 1 year mortality 19%); of 340 remaining, 43% had complete response (normalization of the free light chain levels, negative SPEP, UPEP) and 78% had an organ response
- 52% of those without complete response still had organ function improvement
- Those with a complete response: median overall survival was 13.2 years
- If ineligible for treatment: median survival: 4 months from time of evaluation
Our patient today

- Met criteria for HCT.
- Currently receiving myeloablative chemotherapy.
- Continues diuresis on furosemide and metolazone.

Resources

- Gertz MA. How to manage primary amyloidosis. Leukemia 2012; 26:191.
- MKSAP 16 Nephrology. American College of Physicians 2012; 54-55
Thank you!
Questions??

Calcium doesn’t do a body good

Adam Tinklepaugh, MD

November 9, 2012
University of Utah
Salt Lake City, UT
Case

CC: Painful rash

HPI:
- 58 y/o M c/o 1-2 month h/o dark, painful rash on R leg, R foot, R hand, and penis
- Denied blistering
- Denied prior topical treatment
- R foot swelling, skin peeling, and clear drainage 1 week prior to presentation

PMH: DM II (retinopathy, neuropathy), ESRD on HD, PVD

PSH: LLE amputation

Med: ASA, amlodipine, lisinopril, hydralazine, cephalexin

All: NKDA

FH: Denies

SH: From Tijuana, Mexico. H/o EtOH and cocaine abuse
Physical exam

T  36.5   HR 88   BP 154/73   RR 17   P. Ox 99% 2L NC

GEN: Chronically ill appearing
HEENT: WNL
CV: RRR with systolic murmur correlating with his fistula
RESP: CTAB
ABD: Soft, NTND, NABS.
EXT: Nonpalpable dorsalis pedis pulse.
NEURO: A/O x4, CN 2-12 intact

Cutaneous exam

[Images of skin lesions]
Cutaneous exam

Representative

Cutaneous exam

Medscape® www.medscape.com

Representative
Labs

141 95 21 93
3.7 36 2.9 6.8

7.6 7.8 310 5.3

25.0

PTH 203
Vit. D 24

Differential diagnosis

- Vasculitis
- Cellulitis
- Cryoglobulinemia
- Nephrogenic systemic fibrosis
- “Pseudovasculitis”
  - Purpura fulminans
  - Coumadin necrosis
  - Antiphospholipid antibody syndrome
  - Cardiac myxoma
  - Cholesterol embolization
  - Radiation arteritis
H&E – deep dermis x400

H&E x100
Epidemiology

- First described by Selye, et al. (*Endocrinology*, 1962)
- 4% prevalence in hemodialysis patients (*Surgery*, 1997)
- Increasing incidence attributed to clinical recognition and awareness
- Metastatic calcification more common than calciphylaxis in ESRD
Associations

– ESRD:
  • Females on dialysis
  • BMI >30
  • Hyperphosphatemia
  • Protein C/S deficiency, antiphospholipid syndrome
  • Systemic corticosteroids

– Non-ESRD:
  • Hyperparathyroidism
  • Excess vitamin D administration
  • Vascular calcification inhibitor deficiencies
  • Autoimmune disorders
  • Warfarin → atypical calciphylaxis
Therapy

• **Supportive care**
  – Stop offending agent, wound care, antibiotics, pain control

• Correct electrolyte/hormone abnormalities
  – calcium, phosphate → sevelamer, lanthanum
  – PTH → Cinacalcet, parathyroidectomy

• Surgical debridement/amputation
  – Increases the risk of sepsis and mortality

• Sodium thiosulfate

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Therapy

• Bisphosphonates → pamidronate, etidronate

• Prednisone

• Revascularization

• Hyperbaric oxygen therapy
Prognosis

- Ulceration at presentation → mortality >80%
- One-year survival rate 45.8%
  - Infection is the primary cause of the high mortality
- Penile calciphylaxis 6 month mortality 69%

References

"When you hear hoof-beats, why think of zebras?"

5/16/2013
Khine Win, PGY1

You're a perfectly healthy horse* except for those stripes. But I wouldn't worry about the stripes too much. We see this sometimes. You just need to diet and exercise. If that doesn't work, try these antidepressants.

*Medical school mantra: "When you hear hoof beats, think horses, not zebras." — Dr. Theodore Woodward
Chief Complaint

- Bilateral feet swelling and pain.

History of present illness

- 31-year-old man presented with 3 weeks history of worsening bilateral feet swelling and pain.

- Initially diagnosed with diabetic neuropathy in the outpatient clinic and was sent home with pain medications.

- Presented to the ED with acutely worsening swelling, redness and pain in his feet. The pain was localized to the feet, worse with weight bearing.

- Associated with chills and nausea.
Past medical history

- Poorly controlled diabetes (Hb A1c 11.6)
- Recurrent MRSA cellulitis/abscesses
- Hypertension
- Hyperlipidemia
- Chronic low back pain
- Depression,
- Anxiety

Home medications

- Oxycodone 15 mg t.i.d
- Lisinopril 40 mg daily
- Simvastatin 20 mg qHS
- Metformin 1000 mg b.i.d.
- Glyburide 5 mg daily
- Lantus 15 units qHS
- Gabapentin 600 mg qHS
Pertinent Physical Exam

- Vitals: T 37.2, BP 124/59, HR 115, RR 20
- Left ankle - erythema and swelling. Very tender to palpation and any ROM.
- Right ankle - has some erythema and swelling, less than the left ankle. Also very tender to touch and movement.
- Both knees have limited ROM due to pain with minimal effort.
- Good dorsalis pedis pulses on both feet and full motor and sensory function.

Pertinent Admission Labs

- WBC 30.6
- CRP 15.4
- ESR 70
Differential Diagnosis

- Infectious arthritis, due to history of poorly-controlled diabetes and previous MRSA infection.
- Rheumatoid arthritis, reactive arthritis, Gout or pseudogout, vasculitis (polyarteritis nodosum, cryoglobulinemia), hepatitis C, parvovirus, lyme disease and sarcoidosis.

Arthrocentesis

- Aspirated fluid from knee and ankle showed minimal WBCs, negative for crystals.
- Gram stain did not show any organisms.
- Routine and AFB cultures from aspirated fluid were also negative.
Labs

- HLA-B27 (Reactive arthritis)
- Gonococcal and chlamydia PCR
- Rheumatoid factor and Cyclic Citrulline Peptide Ab
- Uric acid, serum

Perihilar lymphadenopathy

Source: Semin Respir Crit Care Med © 2003 Thieme Medical Publishers
Non-caseating granuloma in mediastinal node biopsy

![Image](http://www.flickr.com/photos/pulmonary_pathology/6201646890/sizes/l/in/photostream/)

Management

- Initially treated high dose of ibuprofen and oxycodone for pain.
- Prednisone was not started during the hospitalization due to his poorly-controlled diabetes.
- Seen in rheumatology clinic after discharge.
- His symptoms were resolved later with prednisone.
Sarcoid arthropathy

- Prevalence of sarcoidosis was 4.4%.\(^1\)
- Bilateral ankle arthritis (90%)
- Oligo-articular (87%)
- Symmetrical (76%).
- Arthritis for less than two months, symmetrical ankle arthritis, and age less than 40 years had a high sensitivity and specificity for sarcoid arthritis.\(^1\)

References

Of Mice and Men: A Story of Sin Nombre

Tian Zhang MD/PhD
PGY2

Outline

- Case Presentation
- Pathogenesis
- Natural History
- Conclusion
DAY 1

Case

- 26 year old young lady from Wyoming, otherwise healthy

- Presented to outside hospital with 1 week history of:
  dry cough, short of breath
  nausea, emesis without blood
  small volume, ‘green mushy’ diarrhea
  headache
  fevers, chills
  dizziness
  transient blurry vision

- Review of systems:
  rash, arthralgia, synovitis, lumps/bumps
  dysuria, chest pain, night sweats, weight loss

- Found to have WBC 62,000/uL, no diff, transfer to HCH

DAY 1

Case

VS: T36.9, HR118, BP92/56, RR16, O2sat 91% on 6L via NC

General: thin, lethargic, wide eyed young woman, anxious
HEENT: perrl, eomi, sclera white, no exudate
Heart: regular, rapid, no murmur
Lung: mild crackles at bases b/l
Abd: soft, nontender, no splenomegaly
Ext: not edematous
Nodes: no cervical, supraclavicular, axillary, brachial, inguinal adenopathy
Skin: no rash, petechiae, ecchymosis
Neuro: cnii-xii grossly intact, no focal deficits
### DAY 1

**Case**

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<th>B 17%</th>
<th>L 10%</th>
<th>M 4%</th>
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</table>

UA: 30 protein, -glucose/ketone/bili/blood/nitrites/wbc

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**Peripheral Smear**
Peripheral Smear
Case

Urine pneumococcal, legionella antigen
Mycoplasma IgG, IgM normal levels
HIV1/2 serology
Bronchoscopy/BAL
  RSV pcr
  adenovirus DFA
  human metapneumo virus
  influenza A/B
  parainfluenza DFA
  respiratory culture normal flora
Blood Cx
Fungal Cx
Case...back to basics

Environmental: lives in trailer near construction site, very dusty, moldy
Zoonosis: Cows, chickens, sheep, horses, dogs
Travel: Louisiana, Texas, Nevada

Additional testing sent: Leptospirosis
Hanta virus
Coxiella burnetti
Mycoplasma
Aspergillosis
Coccidioides
Histoplasmosis

DAY 3

Hanta Virus IgM IgG positive
With specificity for Sin Nombre Virus

Hantavirus Outbreaks
8-28-2012: Rare Illness Has Killed 2 Lodgers at Yosemite

Bunyaviridae: Hantavirus

Puumala - Old World Virus
HFRS: hemorrhagic fever with renal syndrome
Voles as carriers
Asia, Europe

Sin Nombre - New World Virus
HCPS: hantavirus cardiopulmonary syndrome
Deer mouse as carriers
North America
The Natural History of Sin Nombre Virus Infection

- Incubation: 2-3 weeks

- Prodrome/febrile phase:
  fevers, chills, myalgias, rhinorrhea, pharyngitis, coryza

- Cardiopulmonary phase

- Shock: coagulopathy, hemorrhage, pulmonary edema,

- Arrhythmias, Oliguric/diuretic phase

- Convalescent phase: reached within 2 - 7 days **DAY 4-5**
Laboratory Findings

- Increased LDH, transaminitis, leukocytosis
- Immunoblasts, >10%
- Presence of myelocytes
- Absence of toxic granules
- Rapid thrombocytopenia
- Hemoconcentration

96% sensitivity  99% specificity

Diagnosis

- Serology (PCR)

Treatment

- Supportive

CONCLUSION

- Avoid anchoring diagnosis
- Value of a careful History and Physical
- Sin Nombre: Early recognition, triage
- Medicine is NEVER BORING!
References:
CLOSING REMARKS

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