



Utah  
Chapter

2017

# CLINICAL VIGNETTE COMPETITION

UTAH ACP RESIDENTS &  
FELLOWS COMMITTEE

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## FALL CLINICAL VIGNETTE PROGRAM | WEDNESDAY, SEPTEMBER 20, 2017

University of Utah | Health Sciences Education Building | Alumni Hall

<b>5:30 PM</b>	<b>DINNER &amp; SOCIAL</b>	
<b>5:50 PM</b>	<b>WELCOME &amp; OPENING REMARKS</b> <i>Residents &amp; Fellows Committee</i>	<b>JUDGES</b> <i>John Christensen, MD</i> <i>Ronak Iqbal, MD</i> <i>Debra Simmons, MD</i>
<b>6:00 PM</b>	<b>PRESENTATIONS</b>	
	<b>TB or Not TB, That is the Question</b> <i>Presented by: Noopur Goyal, MD [R2]</i>	<b>Pg. 07</b>
	<b>Lesions in Lobes: A Connection Between Dyspnea &amp; Dizziness</b> <i>Presented by: William McKean, MD [R]</i>	<b>Pg. 10</b>
	<b>From Hepatitis C to Hypercalcemia: An Unexpected Progression of Disease.</b> <i>Presented by: Christopher Nevala, MD [R2]</i>	<b>Pg. 12</b>
	<b>A Series of Unfortunate Events: Mystery Mass, STEMI, &amp; Extreme Vascular Disease in a Young Man.</b> <i>Presented by: Wendy Rockne [MS3]</i>	<b>Pg. 13</b>
	<b>A Stroke Mimic: Marchiafava-Bignami Disease</b> <i>Presented by: Vanessa Wall [MS3]</i>	<b>Pg. 19</b>
<b>7:15 PM</b>	<b>ANNOUNCE RUNNERS-UP AND 1<sup>ST</sup> PLACE</b>	
<b>7:30 PM</b>	<b>CLOSING COMMENTS</b> <i>Residents &amp; Fellows Committee</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.

- 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.

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**CASE DESCRIPTION:** Pt is a 68 yo Filipino M who presented with rectal bleeding. He endorsed a 50 lb weight loss, night sweats, malaise and intermittent abdominal pain. He had an upper and lower endoscopy which showed extensive ulcers throughout his GI tract (from mouth to rectum). He immigrated to America about 2 years prior to admission.

**Pertinent Physical Exam Findings:** The patient was fully oriented, although he speaks minimal English. He exhibited conjunctival pallor, sclera were anicteric. An ulcer was noted on the buccal mucosa with surrounding erythema. His abdominal exam was benign, he did not exhibit tenderness. FOBT was positive.

**Lab and Imaging Results:** White count was 1.6, Hgb 7.7 and platelet count 65. CMP unremarkable and his INR 1.4. Stool culture and stool PCR were negative. HIV negative, hepatitis panel negative, CMV negative, EBV negative. ESR 5, CRP 0.6. Quant gold was indeterminate. SPEP/UPEP negative. Gastric ulcer biopsy showed reactive gastropathy and congo red stain was negative. Ileal biopsy showed moderate active inflammation. Flow cytometry demonstrated a population of monoclonal CD5 negative, CD10 negative B cells, concerning for lymphoma. CT scan showed significant mediastinal lymphadenopathy so excisional biopsy was performed. The biopsy showed no evidence of malignancy, however, rare AFB organisms were observed and necrotizing granulomatous lymphadenitis.

**Differential Diagnosis:** On admission, differential included IBD (favoring Crohn's disease), malignancy, and infection.

**DISCUSSION:** The patient presented with non-specific symptoms and initial laboratory studies were inconclusive. In fact, he was inaccurately diagnosed with IBD and started on immunosuppression. It took months of extensive work up, and incorrect diagnoses, to finally diagnose him with TB enteritis.

TB enteritis is a very rare disease and presents with symptoms that overlap with IBD. It is imperative to differentiate the two disorders as their treatment is very different. Unfortunately, to diagnose TB you need a positive stain and you should have a high degree of suspicion for it. He had an initial quantiferon gold that was indeterminate which led us to erroneously consider TB less likely. Interestingly, it was re-checked a few months later and turned positive. With that new information, we honed in on tuberculosis. Decision was made to biopsy one of the enlarged lymph nodes and that finally provided the answer. We extrapolated from the lymph node biopsy that AFB organisms were likely in the GI tract as well and diagnosed him with TB enteritis.

**CASE DESCRIPTION:** Patient is a 61 year old male with history of follicular lymphoma (CD10+), grade 3a, first diagnosed in 2016, he received 6 cycles of RCHOP (completed in 2/16/17), and was found to have recurrence in 4/26/17 and started RICE Chemotherapy. He received cycle 1 in 6/15/17 which was complicated by urosepsis. Cycle 2 was delayed until 7/11/16 for cellular recovery. Patient presented to ED on 7/19/17 due to fever of 102F. He denied sweating, headache, sore throat, cough, shortness of breath, chest pain, nausea, vomiting, or abdominal pain. He endorsed anorexia, fatigue and decreased oral intake and mild diarrhea for a few weeks before presentation. Patient was overall asymptomatic his fever but came in as he was told to come to hospital for any fever greater 101.5F.

**Pertinent Physical Exam Findings:** Patient appeared well developed and was resting comfortably. Neck supple, no oral lesions, mucus membranes were dry, lungs clear, heart regular rate and rhythm, no cervical or peripheral lymphadenopathy, neurologically intact, dry skin with diffuse flaking consistent with known diagnosis of ichthyosis.

**Lab and Imaging Results:** K: 2.9, Cl: 117, HCO3 15, PO4: 1.9, Urine Osmoles 294, Beta 2 Microglobulin 165.6, Urine microalbumin/Creatinine 165.6, Mg 1.3, Urine Electrolytes – Cl: 76, Na: 67, K: 25, PO4: 19, Protein 23.2,

**Differential Diagnosis:** for electrolyte abnormalities: Ifosfamide induced Fanconi's syndrome, Gastroenteritis, mucositis, primary renal phosphate wasting, and chronic diarrhea.

**DISCUSSION:** This patient continually required large doses of IV and PO potassium, phosphorus, and magnesium. Further lab work-up revealed findings consistent with proximal tubule pathology. Patient recently on RICE chemo regimen. Ifosfamide is a chemotherapy that can cause Fanconi's syndrome. The patient also had polyuria and polydipsia which are frequently seen in Fanconi's syndrome (urine volumes of 3-4 L per day). Further lab evaluation confirmed the diagnosis.

In patients with hyperchloremic non-gap metabolic acidosis with previous exposure to ifosfamide further work-up is indicated to rule out Fanconi's syndrome. The Fanconi syndrome is characterized by phosphaturia, glucosuria, aminoaciduria, tubular proteinuria and proximal renal tubular acidosis. Lab evaluation for proximal tubule function are: Beta 2 Microglobulin (very sensitive test for tubular dysfunction), glucosuria without hyperglycemia, urine pH, (variable in Fanconi's), phosphaturia and aminoaciduria. The cause of ifosfamide induced tubular dysfunction is not well understood but may be related to renal toxic metabolites. The patient slowly recovered the ability to keep electrolytes stable without IV supplementation and was discharged home with close lab follow up.

## NEVER TOO OLD FOR A DISEASE WITH A WACKY NAME- A CASE OF HENOCH-SCHÖNLEIN PURPURA. | MARIA BORRERO - MS4

**INTRODUCTION:** Patient is a 69 year-old man with a history of hypertension and atrial fibrillation who presented to the Emergency Department with a 1-month history of abdominal pain, diarrhea, fatigue, and lower extremity rash.

**CASE DESCRIPTION:** Patient initially presented with a 6-week history of abdominal pain, diarrhea, lower extremity rash, which resulted in admission to the hospital. At the time, he had a stool culture positive for e.coli, was treated with metronidazole and levofloxacin and discharged home. Shortly after, he presented with worsening symptoms, particularly worsening lower extremity rash and weakness. In the ED, he was given fluids and was admitted to the hospital for concern of ischemic bowel disease vs. vasculitis vs IBD. Nephrology and Dermatology were consulted.

**Pertinent Physical Exam Findings:** Patient was alert and oriented. Abdomen was soft, nontender, normoactive bowel sounds, no guarding or rebound tenderness, no abnormal masses palpated. Skin findings significant for multiple 5mm palpable, non-blanching, deep red lesions on bilateral lower extremities, extending from ankles to knees.

**Lab and Imaging Results:** On admission: WBC 11.73, granulocyte % 82.2, HGB 12.2, HCT 39.0, MCV 96.5, platelets 473. Na 137, K 4.4, Cl 105, Cr 1.28 (baseline: 1.0), AST 23, ALT 42. U/A significant for hematuria. IgA 339, ESR 28, ANCA <1:320

**Differential Diagnosis:** Ischemic bowel disease given history of atrial fibrillation, IBD, infectious colitis, vasculitis.

**DISCUSSION:** Patient had CT abdomen/pelvis that showed segmental wall thickening in the ileum and fat stranding with no concern for obstruction or perforation. Patient had biopsy of lower extremity purpura, which was consistent with a leukocytoclastic vasculitis and the diagnosis of Henoch-Schönlein Purpura was made. Supportive care restored kidney function and improved symptoms.

Though HSP is more prevalent in children (approximately 1 per 150 to 200), compared to adults (1 per million) , it is important to acknowledge that it can indeed present in the adult population. Classic symptoms of HSP (diarrhea, renal failure, purpura, arthralgias) should prompt further investigation to diagnose this condition, rather than ruling it out based on the low incidence in the adult population. Studies have shown that renal function is significantly impaired in adults compared to children . Given the long-term sequela of this disease in the adult population, prompt diagnosis is imperative.

**INTRODUCTION:** KM is a 29-year-old, married, retail associate, born in the U.S.

**CASE DESCRIPTION:** KM is a 29-year-old female with a history of mild obesity, Type 2 Diabetes, and oligomenorrhea who presents with infertility. She has never had a primary care physician. She has been trying to produce offspring with her husband, who has confirmed fertility, for the past five years. Two years ago, she miscarried at 7 weeks, but has not become pregnant since. She is not using any form of birth control and reports menstruating 3-4 times a year since menarche, which occurred at age 10-years-old. KM recalls breast development and pubic hair before age seven, which is consistent with premature pubarche. She denies acne but reports hirsutism, which manifested during late adolescence. She met with an endocrinologist six months ago, where she was found to have elevated total and free testosterone and Type 2 Diabetes. She was put on Metformin, which successfully regulated her periods—Despite this, her inability to conceive persists. Also, KM has had multiple pelvic ultrasounds—all negative for polycystic ovary morphology. She has not been diagnosed with PCOS nor has she trialed clomiphene.

**Pertinent Physical Exam Findings:** Blonde facial terminal hair in a masculine pattern and obese abdomen.

**Lab and Imaging Results:** Basal serum 17-hydroxyprogesterone: 210 ng/dL, serum 17-hydroxyprogesterone with ACTH stimulation test: 1589 ng/dL, total testosterone: 79 ng/dL, Sex-hormone binding globulin: 30.5 nmol/L, calculated free testosterone: 2.2 nmol/L, TSH: 0.8 mU/L, HbA1c: 6.8%, beta hCG: negative for pregnancy

**Differential Diagnosis:** Differential diagnosis included PCOS, obesity, idiopathic hyperandrogenism, hypothyroidism, and Nonclassic (late-onset) congenital adrenal hyperplasia (NCCAH) due to 21-hydroxylase deficiency.

**DISCUSSION:** This patient had the classic clinical presentation of PCOS, but lacked the polycystic ovarian morphology associated with this diagnosis. Work-up for hypothyroidism was negative, and a positive result would not have explained the hyperandrogenism. Basal 17-hydroxyprogesterone greater than 200 ng/dL warrants a high dose (250 mcg) ACTH stimulation test, which is the gold standard for the diagnosis of NCCAH. An exaggerated response to ACTH (17-hydroxyprogesterone greater than 1500 ng/dL) confirmed the diagnosis in this patient.

In patients with a history including obesity, hyperandrogenism, oligomenorrhea, and infertility, it is important to consider NCCAH in addition to PCOS. Among whites, NCCAH may be as prevalent as 1 in 100 and is notoriously underdiagnosed. Proper diagnosis is imperative to providing appropriate therapy. Infertility associated with NCCAH is treated with glucocorticoids alone or with clomiphene citrate.

**INTRODUCTION:** Ms. A is a 70-year-old woman with gastric bypass 15 years prior with subsequent chronic severe malnutrition and chronic pain who presented to the ED with severe pain and confusion.

**CASE DESCRIPTION:** Two weeks prior, Ms. A was admitted for unintentional weight loss of 30 pounds over 2 months on an oral feeding trial. A MBS demonstrated prevertebral C5-7 esophageal fistula and CT neck did not show evidence of infection, CT surgery was consulted and felt surgery was neither indicated or safe. She was discharged home with a naso-jejunal feeding tube. She initially did well, until about 1 week prior to admission when she had worsening of chronic back and extremity pain. On the day of admission, she was unable to stand so was brought to the ED.

**Pertinent Physical Exam Findings:** T 38.6 °C, HR 106, BMI 12.9. She was AOx4, minimally conversant, and in significant discomfort. She had diffuse tenderness to palpation along paraspinal muscles and in all extremities without deformities. Cranial nerves were intact, she moved all 4 extremities, had symmetric reflexes, and down-going toes bilaterally. Neck and back pain which was unchanged with movement.

**Lab and Imaging Results:** Na 126, K 3.2, Cl 91, Bicarb 28, BUN 11, Cr 0.28, glucose 143. WBC 22.4 with 85% neutrophils, Hgb 9.9, plts 784, lactate 1.0. UA showed 15 WBCs and 4+ bacteriuria. CXR was clear.

**Differential Diagnosis:** UTI, hypovolemia, hyponatremia, refeeding syndrome, esophageal abscess, pneumonia, intra-abdominal infection, meningitis.

**DISCUSSION:** Ms. A was diagnosed with a UTI and started on ceftriaxone. She had fluctuating mental status and a CT head demonstrated new hydrocephalus on hospital day 1. An LP on hospital day 2 showed WBC 193,000 and glucose 1 mg/dl so antibiotics were broadened for meningitis. At that time, she was AOx4, conversant, with normal CN exam which appeared inconsistent with the LP so an MRI of the neuroaxis was obtained overnight. The following morning, she had acutely worsened mental status. The MRI showed a C6-T1 peri-vertebral abscess, C6-7 epidural abscess, cervical cord edema. She transitioned to comfort care and died early the following morning.

In patients with altered mental status and back pain, meningitis should be considered early. In this specific scenario, esophageal fistula was considered but was not actively pursued since it had been addressed as “nothing to do” at a recent hospitalization. Extra care should be taken to avoid anchoring bias from previous work-ups.



**CASE DESCRIPTION:** Pt is a 67 y/o man with a history of chronic pain and headaches treated with epidural steroid injections and is on daily opioid medications who presented following a trip to Amsterdam with worsening neck pain, headaches, photophobia and confusion over the past 72 hours. The patient endorsed improvement of headaches when lying flat, fevers, and some mild persistent nausea but denied vomitus. His recent medical history is significant for epidural injections two months prior to presentation and radio frequency ablation of the L spine one month after that. The patient denied any recent trauma.

Pertinent Physical Exam Findings: Vitals: 38.3, HR 112, RR 14, BP 104/69, O2 sat 93%

Neuro: the patient was grossly orientated to year and wife but not to location or month. He was mildly dysarthric with speech. Neck pain with flexion. CN's intact. Motor strength: 4/5 throughout upper/lower extremities with intact sensation throughout.

Lab and Imaging Results:

CSF studies: WBC count 2525 w/ 74% neutrophils, Protein 121, Glucose 14.

CSF Gram stain: Gram Positive Cocci

Peripheral WBC count 12.1.

CT brain: was significant for intracranial gas vs fat globules in the CSF. Subsequent MRI confirmed the presence of fat globules in the CSF.

Differential Diagnosis: On admission, the differential diagnosis included chemical meningitis, bacterial meningitis, encephalitis, stroke, drug intoxication, and trauma.

**DISCUSSION:** The patients CSF findings and gram stain were consistent with a bacterial meningitis however, given the presence of intracranial fat within the CSF there was suspicion for possible concomitant chemical meningitis. Furthermore, there was concern for possible septic infiltration of fat globules which may act as a bacterial reservoir or foreign body within the CSF. Intracranial fat can be a sequelae of dermoid cyst rupture which, in this patient, may have been related to recent lumbar spine instrumentation.

There are currently no guidelines or published data for the treatment of bacterial meningitis in the setting of fat globules within the CSF. In this case, the patient was initially treated with broad antibiotics which were subsequently narrowed to vancomycin for concerns for staphylococcal meningitis. Follow up LP at 14 days demonstrated resolution of bacterial meningitis with a decrease in inflammatory markers.

**CASE DESCRIPTION:** A 73 year old woman presented to an emergency department for the third time in one week due to abdominal pain. Her family also noted two weeks of worsening confusion and personality changes. Her medical history is notable for systemic lupus erythematosus (SLE) and B-cell non-Hodgkin's lymphoma (NHL), with medication review including hydroxychloroquine with prednisone for SLE and rituximab for NHL. Following admission she developed a fever of 38.4C, worsening transaminitis, and her cognition deteriorated to name-only orientation. A lumbar puncture was performed with administration of appropriate antimicrobials. Intracranial imaging ruled out acute infarction or hemorrhage, but MRI was notable for non-specific bilateral subcortical hyperintensities. Upper endoscopy to further assess her abdominal pain revealed gastritis with ulceration. She remained encephalopathic, her abdominal discomfort persisted, and she developed a vesicular rash on the fourth day of admission.

**Pertinent Physical Exam Findings:** Her cutaneous exam was normal on initial evaluation but became significant for small clear vesicles on mildly erythematous bases scattered across her face, torso, back, and extremities. Her neurologic exam was unremarkable other than tangential and disorganized thought content with intermittent violent outbursts. Abdominal exam was significant only for mild epigastric tenderness.

**Lab and Imaging Results:** Cerebrospinal fluid studies were bland with no malignant cells and negative viral PCR. Varicella zoster virus (VZV) PCR was positive from fluid obtained from two separate cutaneous vesicles. Gastric biopsy revealed findings suggestive of viral cytopathic effect and Cowdry type A inclusions. Transaminases reached peaks of aspartate aminotransferase 279 u/L and alanine aminotransferase 174 u/L.

**Differential Diagnosis:** Unifying differential diagnosis includes disseminated VZV infection, SLE flare with cerebritis and bullous rash, or progression of NHL.

**DISCUSSION:** Following consultation with infectious disease colleagues this patient was diagnosed with a disseminated VZV infection deemed secondary to her chronic immunosuppression. Viral dissemination was demonstrated by dispersed cutaneous vesicles rather than a dermatomal distribution. Her encephalopathy was likely due to VZV induced vasculitis rather than encephalitis given negative cerebrospinal fluid VZV PCR. MRI findings are nonspecific but consistent with this diagnosis. Gastric biopsy with evidence of herpesvirus replication suggests VZV dissemination to her gastrointestinal tract. This likely underlies her abdominal discomfort and transaminitis as her liver function tests normalized with acyclovir therapy. The patient was discharged on a three-week course of IV acyclovir and was improving at the time of discharge.

Common infectious diseases can yield perplexing manifestations in immunocompromised hosts, requiring broad differential diagnoses and thorough clinical evaluation.

**CASE DESCRIPTION:** 82 year old Cambodian male with a history of hypertension and diabetes, presents with a six month history of fevers, chills, sweats, and weight loss. He notes painful axillary lymphedema that had developed over the last three months. A CT abdomen/pelvis showed widespread metastatic disease of the mesentery, omenta, and retroperitoneal lymph nodes, with concern for disseminated lymphoma. Given consolation of symptoms, further workup included negative HIV, hepatitis panel, EBV, and ANA. Surprisingly, patient had a positive RPR and Quantiferon Gold. Patient denied known TB exposure or treatment, and his last visit to Cambodia was twenty years ago. He further denied recollection of chancre, rash, and diagnosis or treatment for syphilis in the past. This became a curious case of disseminated lymphedema with work up to evaluate etiology of lymphoma versus tuberculosis, with concurrent syphilis findings.

**Pertinent Physical Exam Findings:** Patient was an exceptionally cachectic male, with temporal wasting. He had large cervical, axillary, and inguinal lymphadenopathy bilaterally, painful to palpation. Axillary lymphadenopathy had grossly purulent drainage.

**Lab and Imaging Results:** Positive RPR, FTA, Quantiferon TB Gold, with >10 IU/ml Quantiferon NIL, and Positive Mycoplasma TB PCR from lymph node biopsy. CSF fluid negative for TB or RPR. Flow cytometry of blood consistent with reactive and/or infectious process, without evidence of myeloid or lymphoproliferative disorder. PET CT consistent with hypermetabolic adenopathy seen in the neck, chest, abdomen and pelvis. CT Chest without evidence of pulmonary TB.

**Differential Diagnosis:** Initial concern for disseminated lymphoma vs extra pulmonary tuberculosis, with concurrent positive syphilis findings.

**DISCUSSION:** This patient initially presented with diffuse findings of lymphadenopathy, concerning for lymphoma. However, given his symptoms, Quantiferon Gold was evaluated and found to be positive. Subsequent TB PCR confirmed diagnosis from lymph node biopsy, with flow cytometry negative for malignancy.

Patients with fevers, sweats, weight loss, and significant lymphadenopathy should be considered for TB workup in the setting of known risk factors including immune suppression, immigration status, and extremes of age. TB lymphadenitis is the most common presentation of extra pulmonary TB, and can be diagnosed with FNA of lymph node or excision biopsy. Clinical history plays an important role in consideration for TB workup as extra pulmonary presentation can mimic many other disease processes including malignancy and autoimmune disease.

**INTRODUCTION:** Mr. B is a 19 yo M without past medical history who presents with hemoptysis.

**CASE DESCRIPTION:** 2 weeks ago, he developed nasal congestion, generalized malaise, and diffuse myalgias. 1-week ago, he developed hemoptysis. He describes the hemoptysis as frank, not scant. Today, he also reports fevers to 101.0 F, weight loss of 10 pounds over the past 2 weeks, and dyspnea. He is a college athlete on scholarship to play baseball. He denies any international travel. He also denies tobacco or illicit drug abuse.

Pertinent Physical Exam Findings: VS: T- afebrile, HR- 102, RR- 25, SpO2- 89% on 4L

HEENT: aphthous ulcer on tongue, dried blood in oropharynx

Pulmonary: diffuse, fine expiratory crackles present bilaterally

Lab and Imaging Results: CBC: WBC- 20.8, HGB- 9.7, Plts- 468

BMP: Na- 136, K- 4.1, Cl- 102, HCO3- 25, BUN- 14, creat- 0.98

UA: large blood, > 30 RBCs, 1+ protein, 18 WBCs

Initial CXR showed diffuse alveolar opacities. Broad infectious and rheumatologic laboratory evaluation was ordered. C-ANCA was positive at 1:640, while the rest of his work-up was unremarkable. He was clinically diagnosed with GPA and started on high-dose corticosteroids and rituximab. He was discharged home. As an outpatient, a renal biopsy was consistent with ANCA-associated glomerulonephritis.

Differential Diagnosis: Necrotizing pneumonia, tuberculosis, lung cancer, anti-glomerular basement membrane disease (Goodpasture's), granulomatosis with polyangiitis (GPA), systemic lupus erythematosus (SLE)

**DISCUSSION:** Frank hemoptysis is uncommon in young adults and generates a broad differential. Thorough history can refine the broad differential, yet final diagnosis usually requires laboratory evaluation, chest imaging, and/or bronchoscopy. The most common causes of hemoptysis in developed countries are bronchiectasis, lung tumors (mets vs. primary), and fungal infection. GPA is a rare diagnosis affecting 8-10 patients/million with a mean age of 55. GPA is diagnosed by the presence of 2 of the 4 following clinical criteria: nasal or oral inflammation, abnormal chest radiography, abnormal urinary sediment, or granulomatous inflammation on arterial biopsy. A biopsy is usually obtained to confirm the diagnosis since GPA treatments have many potential adverse events.

Frank hemoptysis in a previously healthy, young adult generates an interesting differential. GPA is a rare but potentially debilitating cause of hemoptysis. The triad of hemoptysis, hematuria, and oral/nasal inflammation should raise suspicion for GPA.

**CASE DESCRIPTION:** A 55 year old man with diabetes, schizophrenia and cognitive impairment presented to the Emergency Department with abdominal pain, and feeling weak and “different.” His family reported one month of fatigue, poor appetite and weight loss. There was no history of kidney disease. He denied NSAID use, skin rash, or joint pain. The patient was an active smoker with 14 pack years. His home medications included metformin, oxcarbazepine, risperidone, and fluphenazine. He had no drug allergies.

**Pertinent Physical Exam Findings:** On physical exam, the patient was tachycardic to 128 beats per minute and tachypneic with respiratory rate of 28. There were rhonchi heard bilaterally. The abdomen was distended and soft, but nontender and without hepatomegaly. The patient was noted to have ascites.

**Lab and Imaging Results:** Labs showed a leukocytosis to 20,000 (96% neutrophils), creatinine of 17 mg/dL and BUN of 207. Potassium was 7.3 mmol/L and his anion gap was 29. Transaminases were normal. The patient was admitted to the MICU, and his labs improved following placement of a foley catheter. Urinalysis was positive for leukocyte esterase, 63 WBC, and bacteria, although cultures were negative. CT Abdomen/Pelvis was done and showed complex retroperitoneal fluid collections, a large right perinephric fluid collection, marked left renal pelvis urothelial thickening, urinary bladder thickening and moderate ascites. Paracentesis was done, showing 28,000 WBCs, with serum albumin-ascites gradient (SAAG)  $>1.1$ . Ascites cytology did not show malignancy, and secondary bacterial peritonitis was diagnosed.

**Differential Diagnosis:** Differential diagnoses considered included cirrhosis, inflammatory bowel disease, intraabdominal malignancy and peritoneal carcinomatosis.

**DISCUSSION:** This case demonstrates the importance of a considering a single diagnosis to explain many abnormalities. The ascites fluid was determined to be urine, based on the presence of creatinine and a right renal cyst on imaging. Pyelogram revealed rupture of the left renal pelvis, with extravasation of contrast into the retroperitoneum. It was determined that the urine was secondarily infected, causing sepsis. The right renal cyst was likely insignificant. The cause of urinary obstruction was determined to be benign prostatic hypertrophy.

While nontraumatic renal pelvis rupture is rare, it is important to consider this as a diagnosis for ascites in a patient with risk factors for urinary tract obstruction. The SAAG is used to determine the cause of ascites, and SAAG  $>1.1$  is typically diagnostic of ascites due to portal hypertension, though studies likely did not include patients with uroperitoneum.

**INTRODUCTION:** 22 y/o previously healthy female presents with dyspnea

**CASE DESCRIPTION:** This patient is a 22 y/o previously healthy woman with 2 months of arthralgias, facial rash, fevers, and a dry cough. She presented with new onset dyspnea and deep inspirational infrasternal chest pain. Workup was diagnostic of Lupus and APS in addition to biopsy-confirmed Lupus nephritis, Libman-sacks endocarditis, and subacute cerebral infarcts. She was started on immunosuppressant therapy and anticoagulation with Coumadin and aspirin for triple positive APS and cerebral infarcts. Four days later she developed worsening cough, hemoptysis and respiratory distress. Anticoagulation was held but bronchoscopy showed no evidence of hemorrhage. She initially improved with diuretics and continued holding of anticoagulation but then was sent to the MICU for acute respiratory decompensation.

**Pertinent Physical Exam Findings:** BP 147/102, HR 116, RR 41 with 98% (2-4L O<sub>2</sub>). Lungs were clear bilaterally, and a 3/6 holosystolic murmur was best heard at LLSB. Livedo reticularis was present on feet bilaterally.

**Lab and Imaging Results:** WBC 20.66, HGB 6.5, INR 3.0, creatinine of 2.72, procalcitonin 0.24 , BNP 744, D dimer 1051; dsDNA (1:320), +lupus anti-coagulant/+Beta-2 glycoprotein/+cardiolipin, ANA 1:2560 and low complement levels; TTE: moderate to severe mitral valve regurgitation; CXR: basilar R>L consolidation; initial BAL negative; CT chest w/o contrast: alveolar edema and/or hemorrhage in the setting of SLE and hemoptysis. Possible superimposed infection versus hemorrhage; VQ scan: Low probability of PE

**Differential Diagnosis:** PE, pneumonia, DAH 2/2 vasculitis from lupus, pulmonary edema 2/2 mitral valve regurgitation.

**DISCUSSION:** Initial work-up included a negative VQ-scan and negative infectious studies. Given the initial bronchoscopy showed no evidence of blood, pulmonary edema 2/2 worsening mitral valve regurgitation was thought to be contributing. However, she did not improve with additional aggressive diuresis and a new echo showed no worsening mitral valve pathology. For her continued decompensation she underwent a second bronchoscopy which showed DAH.

DAH should be kept on the differential diagnosis when considering respiratory distress in a patient with Lupus. In addition, risks and benefits of anticoagulation have to be assessed carefully. Other than aggressive lupus treatment, there is no optimal solution for this patient as a lack of anticoagulation puts her at risk for PE and additional strokes, whereas anticoagulation may lead to additional life-threatening diffuse alveolar hemorrhage. She ultimately underwent aggressive plasmapheresis and daily cyclophosphamide, was intubated and then developed VTEs and was put back on anticoagulation.

**CASE DESCRIPTION:** The patient is a 44-year-old gentleman with a history of familial hyperchylomicronemia, recurrent pancreatitis, type 2 diabetes mellitus, and hyperlipidemia who presented to the ED with epigastric pain and retching. 24 hours prior to admission, the patient began feeling a sharp pain in his left upper quadrant and epigastrium that soon radiated to his substernal area as well as his back. This pain increased in severity until approximately 4 hours prior to presentation to the ED when the patient began experiencing nausea and repeated episodes of dry heaving. He described the pain as similar in character and progression to previous bouts of pancreatitis.

**Pertinent Physical Exam Findings:** The patient was visibly uncomfortable, but did not appear to be in severe distress. His upper abdomen— including the left upper quadrant, epigastrium, and right upper quadrant— was diffusely tender. The abdomen was soft and non-distended and no rebound tenderness, guarding, or hepatosplenomegaly was appreciated. Bowel sounds were normoactive.

**Lab and Imaging Results:** Glucose 173 Na 135 K 3.8 Cl 103 CO<sub>2</sub> 14 Cholesterol 496 HDL 17 Direct LDL 74 PH 7.419 PCO<sub>2</sub> 36.3 HCO<sub>3</sub> 23 Anion Gap 18 Lipase 2748 Alk Phos 74 AST 13 ALT 15

**Differential Diagnosis:** The patient was quickly diagnosed with acute on chronic pancreatitis. The differential for his gap acidosis initially included euglycemic diabetic ketoacidosis, lactic acidosis, and salicylate poisoning.

**DISCUSSION:** Upon further questioning, the patient admitted that he had run out of his prescribed fenofibrate over 2 months prior to admission and rather than refilling it, had stopped taking it. Subsequent labs revealed a serum triglyceride level of 2193. In the absence of other identifiable etiologies, it is believed that the patient's hypertriglyceridemia was the cause of his anion gap metabolic acidosis.

Severe hypertriglyceridemia, commonly seen in patients with familial hyperlipidemias, type 2 diabetes mellitus, and poor diet, is a rare cause of anion gap metabolic acidosis. It should be suspected when more typical etiologies have been ruled out or when complications of hypertriglyceridemia, such as acute pancreatitis, are evident. Resolution of the anion gap acidosis is achieved by normalizing the serum triglyceride level.

**INTRODUCTION:** Budd Chiari syndrome is defined as venous outflow obstruction of the liver, which usually occurs as a consequence of thrombosis of the hepatic veins. Primary Budd-Chiari syndrome, which is caused by obstruction due to a predominantly venous process, is a

**CASE DESCRIPTION:** A 31 year-old male with history of recurrent venous thromboembolic disease due to anti-phospholipid antibody syndrome (APLS) currently on anticoagulation with warfarin presented with 2 days of rapidly progressing scrotal swelling, lower extremity edema, nausea and vomiting. Pertinent labs on presentation include: international normalized ratio (INR) of 2.8, albumin 2.6 g/dL, total bilirubin 0.5 mg/dL, alkaline phosphatase of 68 IU/L, aspartate aminotransferase (AST) of 90 IU/L and alanine aminotransferase (ALT) of 36 IU/L.

Hepatic ultrasound of the abdomen with Doppler demonstrated no visible flow in the hepatic veins with hepatofugal portal vein flow, splenomegaly and numerous portosystemic collaterals. Interventional Radiology (IR) was consulted to place a stent from middle hepatic vein to inferior vena cava. In the subsequent days, the patient was continued on warfarin however his swelling did not improve, and repeat ultrasound was essentially unchanged. IR proceeded to perform venoplasty and thrombectomy of the thrombosed hepatic vein stent. Following this procedure the patient's scrotal and lower extremity edema improved significantly. The patient was discharged on lovenox for treatment of his Budd-Chiari syndrome secondary to his underlying APLS.

**DISCUSSION:** Budd-Chiari syndrome is a rare but important case of liver failure, and has numerous etiologies, including APLS. We demonstrate a rare presentation of this disease in a young male who presented with acute onset scrotal swelling. The mainstay of treatment for APLS is anti-coagulation with warfarin. However, our patient was on warfarin prior to presentation, although it is unclear whether his coagulopathy was related to vitamin K antagonist administration or liver failure related to Budd-Chiari syndrome. Novel anticoagulants have not yet been studied on patients with APLS or cirrhosis, so our patient was discharged to remain on low molecular weight heparin.



**CASE DESCRIPTION:** Patient is a 76 yo male with a history of interstitial lung disease, pulmonary embolism, squamous/basal cell carcinoma of the skin, and chronic dizziness who presented for syncope and acute visual changes. Approximately one month prior, the patient had seen his PCP after hospitalization for pneumonia; chest imaging showed a suspicious RML/RLL consolidation with hilar lymphadenopathy. These findings were subsequently determined to be hypermetabolic on PET/CT. For several weeks afterward he developed a persistent cough/dyspnea and headache, and began to see “flashing lights.” This was followed by falls and syncope; outpatient brain MRI revealed numerous ring-enhancing lesions and prompted a referral for hospitalization. On intake, he described an unintentional weight loss of ~40 pounds over 6 months, night sweats, and fevers measuring to 101 F.

**Pertinent Physical Exam Findings:** Hemodynamically stable on presentation; alert and fully oriented without visible distress. Cranial nerves grossly intact except for upper left visual field deficit. No focal musculoskeletal weakness. Regular rate/rhythm of heart without murmurs/rubs/gallops. Bibasilar rales on auscultation, but otherwise good air movement within all fields and no increased work of breathing. No palpable lymphadenopathy or skin rashes.

**Lab and Imaging Results:** Sodium: 137, potassium: 4.0, BUN: 12, creatinine: 0.89, WBC: 16.8, granulocytes: 80.7%, Hgb: 13.1, platelets: 305, HIV-1,2 Ab/Ag: negative, Histoplasma Ab/Ag: negative, Coccidioides IgG/IgM: negative, QuantiFERON-TB: negative, flow cytometry: negative, ANA: negative.

**Differential Diagnosis:** Initial differential included metastatic lung cancer, disseminated tuberculosis, endemic fungal infection, cryptococcosis, nocardiosis, actinomycosis, and toxoplasmosis. Underlying rheumatologic disease was also considered.

**DISCUSSION:** Patient with suspicious pulmonary consolidation presenting with vision changes and syncope, found to have new ring-enhancing lesions on brain MRI. Patient deemed immunocompetent, without risk factors for tuberculosis. Despite concern for metastatic disease, endobronchial biopsy was negative for malignancy and demonstrated only chronic inflammation; empiric antibiotic therapy was subsequently started. Bronchoalveolar lavage returned positive for *Nocardia beijingensis*, and antibiotics were narrowed to ceftriaxone and TMP/SMX.

While the differential for pulmonary consolidation is broad, concomitant cerebral ring-enhancing lesions narrow the possibilities considerably. In such cases, clinical symptoms from infection and malignancy often overlap, and careful diagnostic consideration must be given to both. Furthermore, chronic lung disease can predispose otherwise immunocompetent hosts to opportunistic infections. In the case of systemic nocardiosis, symptoms can be nonspecific and mimic exacerbations of longstanding pulmonary disease; CNS dissemination may be clinically silent until vasogenic edema produces mass effect.

**CASE DESCRIPTION:** 56 yo male with HTN, COPD, tobacco abuse, who presented as a transfer from OSH, where he initially presented with chest pain. This started the afternoon of the presentation, and was described as typical angina. OSH ED course complicated by VF arrest, which resolved quickly with defibrillation, and no chest compressions. After being found to have a STEMI, he was given lytics, ASA, clopidogrel, started on heparin drip, and transferred to UUMC for catheterization. He underwent PCI to LAD, and around 8pm, was admitted to CVICU for post-STEMI management. He was hemodynamically stable, and his chest pain had improved significantly.

Around 1am, he suddenly developed recurrence of severe chest pain, similar in character to his presenting angina.

Pertinent Physical Exam Findings: Vitals stable. In distress due to chest pain, improved after PCI. RRR, normal S1/S2, no M/R/G. No JVD, no edema, no crackles. After recurrence of chest pain: Vitals: HR 130s. SBP 120s. Tachycardic, regular. Harsh 3/6 holosystolic murmur, loudest at LLSB and apex. JVD, crackles, edema now present.

Lab and Imaging Results: Initial troponin: >440; BNP: 106 -> 1642 3 days later; Oxygen saturations: RA-73%, RV-90%, PA-88%, systemic-95%

EKG: Initially, ST elevations in V1-4, depressions inferiorly. Improved after PCI, but worsened with recurrence of his chest pain. Similar worsening noted on telemetry at that time. Echo notable for EF 52%, 7x8mm VSD.

Differential Diagnosis: In-stent thrombosis, coronary or aortic dissection, coronary vasospasm, pericarditis, septal or free wall rupture.

**DISCUSSION:** The initial concern was for in-stent thrombosis, which would present with similar chest pain and EKG changes. However, after auscultation of new murmur, VSD became most likely. Bedside echo prior to catheterization showed left-to-right flow. Repeat angiography confirmed that stent was patent, and a saturation run showed a large increase in oxygen saturation from RA to RV, consistent with left-to-right shunt. VSD was confirmed on formal TTE in the morning. He was medically managed in the CVICU while his clopidogrel washed out, after which he was taken to the OR for surgical VSD repair.

VSDs have become rare as ACS treatment has progressed. When present, they are classically a later complication of large MI. However, this case shows that VSD should be in the differential of clinical decompensation at any stage post-MI. This case also highlights the continued importance of the physical exam in clinical assessment and decision making.

**CASE DESCRIPTION:** Patient is a 66 year old male with a history of PVD, COPD, active tobacco use, and non-obstructive coronary artery disease who presented with left sided substernal chest pain of approximately one year in duration with worsened chest pain of last five hours. He reported worsened left sided, sub-sternal chest pain, not associated with shortness of breath that started with increased physical activity. The chest pain is similar in quality but longer in duration, to the chest pain that he has experienced over the last year. He normally develops chest pain walking around the house and completely it resolves with rest. The chest pain resolved with administration of nitroglycerin on presentation.

Pertinent Physical Exam Findings: BP 131/65, HR 80, RR 18, SpO2 95% on RA. Patient was in no acute distress. Cardiac exam was regular rate and rhythm without murmurs, rubs or gallops. Pulmonary exam was notable for prolonged expiration but no wheezing or respiratory distress.

Lab and Imaging Results: Serial troponins were 0.00, 0.02 and 0.02. ECG: Incomplete R bundle branch block, with non-specific ST and T wave abnormalities. TTE: normal EF without wall motion abnormalities. Coronary angiogram: non-obstructive CAD with mid-LAD myocardial bridge with complete luminal obliteration in systole.

Differential Diagnosis: Acute Coronary Syndrome with unstable atherosclerotic plaque rupture, dissection, coronary artery spasm, myocardial bridge, supply/demand mismatch or stable angina

**DISCUSSION:** The patient had acute coronary syndrome/ unstable angina secondary to a significant left anterior descending myocardial bridge with complete obstruction of blood flow during systole. His acutely worsened chest pain over baseline with normal serial troponins differentiates unstable angina from NSTEMI and stable angina. Atherosclerotic plaque rupture, the most frequent cause of ACS, was not visualized on coronary angiogram, despite his known history of coronary artery disease. He had no other non-cardiac hemodynamic factors to suggest a type 2, supply/demand mismatch cause of myocardial infarction. He had no evidence of a double coronary artery lumen to suggest coronary artery dissection. He did not have a history consistent with a recent ingestion or drug use to suggest coronary artery spasm.

Myocardial Bridge is a relatively common congenital anomaly where the epicardial coronary artery takes an intramuscular course under the bridge of overlying myocardium, and although generally asymptomatic, myocardial bridging can have multiple clinical consequences that range from angina to sudden cardiac death.

**CASE DESCRIPTION:** A 63 year old female smoker with a history of compensated cirrhosis secondary to hepatitis C virus (HCV)

She presented with a 3 week history of progressively worsening confusion, fatigue, constipation and pain below her right rib cage.

Pertinent Physical Exam Findings: Physical exam on admission was unremarkable except for mild right upper quadrant abdominal tenderness to palpation and mild dysarthria.

Lab and Imaging Results: Initial laboratory work up revealed severe hypercalcemia with a corrected Ca of 16.3 mg/dl as well as mildly elevated LFTs (total bilirubin 3.5 mg/dl, alkaline phosphatase 277 U/L, and AST 156 U/L). A CT scan of the chest/abdomen/pelvis revealed a heterogenous filling defect within the hepatic vasculature concerning for hepatocellular carcinoma (HCC). MRI of the abdomen confirmed the presence of a large HCC with extensive tumor thrombus. Imaging revealed no evidence of an intrathoracic malignancy and showed no evidence of bony metastatic disease. Further workup revealed an elevated parathyroid hormone-related protein (PTHrP) (19.1 pmol/L) with low intact parathyroid hormone (PTH), low 25 hydroxy vitamin D and normal 1,25 dihydroxy vitamin D suggesting hypercalcemia of malignancy as the cause for her severe symptomatic hypercalcemia.

Differential Diagnosis: This patient was treated with IV fluids, calcitonin, and a bisphosphonate with improvement of her serum calcium levels to 10.5 mg/dL. Unfortunately, given the size of her tumor and associated tumor thrombus the patient was not a candidate for surgical res

**DISCUSSION:** Hypercalcemia is a relatively common presentation of certain malignancies including head and neck, breast, lung, esophageal, renal, ovarian and endometrial cancers. The finding of a large HCC in this case was unexpected, as hypercalcemia secondary to HCC is rare. To the best of our knowledge, the prevalence of PTHrP mediated paraneoplastic hypercalcemia associated with HCC has not been documented in the United States. However in regions of Asia, where there is a higher prevalence of HCV and HCC, studies estimate that between 4% and 8% of HCCs are associated with paraneoplastic hypercalcemia.

When evaluating patients with suspected paraneoplastic hypercalcemia, the differential diagnosis must look beyond the most commonly associated malignancies, especially if risk factors for other malignancies are present such as cirrhosis secondary to HCV in this patient. Lastly, as was also demonstrated by this case, paraneoplastic hypercalcemia with HCC is often associated with higher tumor burden and overall poor prognosis.

**INTRODUCTION:** A 55-year-old man with type 2 diabetes mellitus, gastroparesis, fatigue, weight loss and daily marijuana use presented to the ED with a 5-day history of acute on chronic vomiting.

**CASE DESCRIPTION:** The patient describes a 5-day history of nausea and bilious, non-bloody emesis occurring up to 15 times daily. Notably, the patient has been hospitalized for nausea and vomiting four times in the six months. Previous episodes have been refractory to conservative treatment with ondansetron, promethazine and metoclopramide. Per patient’s chart, these episodes have been attributed to a combination of gastroparesis and cannabinoid use. The patient’s diabetes is poorly controlled with a recent HgbA1c of 12% and he endorses daily use of marijuana over the past two years. On this hospitalization, patient also endorsed increasing fatigue, malaise, night sweats and a 20-lb. weight loss over the past year. Patient specifically denied fever, chills, chest pain, shortness of breath, diarrhea, melena, hematochezia or hematemesis.

**Pertinent Physical Exam Findings:** Patient is afebrile and hemodynamically stable. Awake and interactive, appears older than stated age, mild distress due to discomfort. Dry mucous membranes, skin tenting and conjunctival injection noted. Remainder of physical exam is grossly normal.

**Lab and Imaging Results:** Test: Result (Reference Range) WBC: 20.6 K/uL (3.6 – 10.6 K/uL); Lymphocytes: 13.9 K/mcL (1.2 – 3.1 K/mcL); Atypical Lymphocytes: 3+ (Negative); Immature Granulocytes: 1.95 K/mcL (0.0 – 0.03 K/mcL). Flow Cytometry Report: Bone marrow biopsy sample. Flow cytometry (see figures 1 and 2) showed two distinct aberrant blast populations (B/Myeloid) consistent with Mixed-Phenotype Acute Leukemia (MPAL).

**Differential Diagnosis:** Cannabinoid Hyperemesis Syndrome; Gastroparesis secondary to Type II Diabetes Mellitus; Acute Lymphocytic vs Myelocytic Leukemia

**DISCUSSION:** In this case, our patient had multiple explanations for his recurring nausea and vomiting including gastroparesis and cannabinoid hyperemesis syndrome. A close analysis of the patient’s CBC + differential revealed an unexpected leukocytosis which expanded our differential diagnosis. It was the often-overlooked WBC differential that brought to our attention the curious presence of both atypical lymphocytes and immature granulocytes. Flow cytometry (see figures 1 and 2) was ordered and revealed Mixed-Phenotype Acute Leukemia (MPAL) with t(9;22)(q34.1;q11.2); BCR-ABL1, a rare cancer of the blood which accounts for only 2-5% of all leukemia. The patient was transferred to the Hematology service for bone marrow transplant and chemotherapy.

In patients with vague, progressive and refractory symptoms it is important to create a broad differential diagnosis taking into account all aspects of the patient’s work up. Previous diagnoses in a patient’s chart may provide clues, but should be approached cautiously to avoid skew the physician’s clinical acumen.

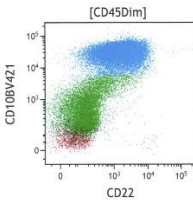


Figure 1. Myeloblasts in green, lymphoblasts in blue.

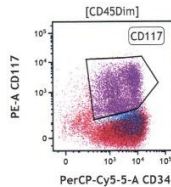


Figure 2. Myeloblasts in purple, lymphoblasts in blue.

**INTRODUCTION:** Mr. SLP is a 51 yo M w/ PMH significant for Hep C, referred here today for joint pain.

**CASE DESCRIPTION:** Mr. Peterson has a long history of hand pain starting several years ago. He reports having multiple hand trauma events in the past including various hand fractures and trauma. He states he has had this similar pain for years now, but recently has gotten worse prompting his referral. He states his pain is constant, worse at night after using his hands all day as a painter, relatively controlled by ibuprofen. He indicates his L pointer finger as the worse with limited ROM due to pain limiting his ability to work. He reports episodes of intense pain, lasting variable amounts of time, during which he has to take ibuprofen and use cold/hot packs in order for any relief.

**Pertinent Physical Exam Findings:** Mr. SLP exhibited significant tenderness around many joints, specifically the R shoulder, b/l wrists, b/l hands (b/l MCPs, L hand index finger) with significant deformation in L index finger. Otherwise, his physical exam was normal.

**Lab and Imaging Results:** CRP: 1.2, ESR 2, RF negative, anti-ccp negative, AST 56, ALT 94, HepB sAg: Neg, HepB cAb: Neg, HepC: 1,400,000 IU/mL, Chlamydia & gonorrhea: neg, A1C of 6.4, Ferritin: 3,000, Serum Fe: 222

Hand XR-Erosive and productive changes involving the bilateral hands, centered about the metacarpals, MCPs and proximal phalanges with relative preservation of the radiocarpal and intercarpal articulations. Findings are most suggestive of CPPD arthropathy.

**Differential Diagnosis:** Differential included osteoarthritis, sero-negative RA, Calcium Pyrophosphate Deposition, Hep C related arthropathy, and Hemochromatosis arthropathy.

**DISCUSSION:** This patient had many possible reasons for his severe hand pain, especially considering his diagnosis of untreated Hepatitis C, history of trauma, and XR suggesting CPPD. With elevated inflammatory markers, negative rheumatoid factors, further exploration was warranted, especially with atypical joint involvement for CPPD and a relatively new diagnosis of diabetes with an elevated A1C. Iron labs showed significantly elevated Ferritin, slightly elevated serum iron, concerning for Hemochromatosis. HFE genetic testing showed a positive C282Y gene.

In patients with undifferentiated polyarthritis, there should be a concerted effort to understand the different presenting complaints for various systemic illnesses and not forget to test the differential diagnosis instead of treating the most convenient or common diagnosis. This is especially true in order to treat for diagnoses of exclusion, such as a hepatitis arthropathy.

**CASE DESCRIPTION:** A 39 year-old healthy male presented with new HFrEF, bilateral DVTs, and PE following recent excision of a retroperitoneal mass. He complained of 6 months of productive cough, fevers, night sweats, and dyspnea on exertion. CT revealed a large cyst compressing the left ureter with resultant hydronephrosis, prompting surgical excision of a fibrotic retroperitoneal mass.

Post-op day 1, the patient developed AKI. EKG showed anterolateral lead ST-elevation, but PCI was deferred because of the AKI. An echocardiogram on POD #3 showed LEF < 30%; coronary catheterization was attempted but unsuccessful.

The patient was then transferred here, where angiogram showed three complete occlusions (LAD, LCX, and OM1) with abrupt cut-offs, concerning for embolic phenomenon. Multiple PCI attempts were unsuccessful. He was also noted to have bilateral distal DVTS and a subsegmental PE.

Pertinent Physical Exam Findings: Alert, ill-appearing, anxious. Tachycardic. Bilateral 2+ LE pitting edema

Lab and Imaging Results: Unremarkable: ANA, APS, homocysteine, ANCA, GBM, RF, HIV, SPEP, UPEP, serum IgG4, hepatitis panel, blood cultures. Iron 34, TIBC 166, Ferritin 6,054. Retroperitoneal Mass Pathology: storiform fibrosis with dense lymphoplasmacytic infiltration rich in IgG4-positive plasma cells

Differential Diagnosis: On admission, differential diagnosis included CAD, surgical provocation, inherited hypercoagulable state, paradoxical embolism, and malignancy.

**DISCUSSION:** Although this patient likely had pre-existing CAD, the extent of coronary disease could not be attributed to CAD alone. Extensive tests for inherited hypercoagulable states, underlying malignancy, autoimmunity, and infection were unrevealing, but pathology of the excised retroperitoneal mass was diagnostic of IgG4-related disease (IgG4-RD).

Re-examination of chest CT discovered unexplained nodules in a bronchovascular distribution, a pattern of lung involvement previously described in IgG4-RD. This prompted consideration of disease spread beyond the retroperitoneum, and literature review revealed case reports of STEMI secondary to IgG4-RD coronary artery involvement.

Refractive tachycardia precluded the patient from coronary CTA evaluation, but aggressive glucocorticoid and azathioprine therapy in conjunction with anticoagulation therapy resulted in steady improvement.

In patients with unexplained retroperitoneal fibrosis, IgG4-RD should be considered, as this may account for many cases of presumed idiopathic retroperitoneal fibrosis. Diagnosis is made by pathologic evaluation for characteristic features, as serum IgG4 levels are elevated in only 2/3 of patients with active disease. A high index of suspicion for systemic involvement must be maintained, as multiple organs may be affected. Though rare, coronary involvement with massive intraluminal disease has been reported, so early diagnosis and treatment is imperative to prevent potentially devastating embolic/thrombotic events.

**CASE DESCRIPTION:** A 42 YOF with recently diagnosed dermatomyositis (DM) presented with worsening weakness, dysphagia, and difficulty breathing. Eight weeks earlier she had developed a non-pruritic, erythematous rash on her hands, elbows, and face. She was treated with topical steroids for suspected psoriasis, but then developed open sores on her hands. Biopsy of a lesion was consistent with DM, and she was prescribed methotrexate.

Three weeks ago the patient began to have fevers, diffuse arthralgias/myalgias, and increasing difficulty standing from a seated position and climbing stairs. She then developed dysphagia and progressive dyspnea. Upon presentation, she had not been able to swallow solid foods, walk without assistance, or breathe at rest without significant difficulty for two days. She endorsed a 20-lb weight loss, episodic mouth sores, and worsening hair loss.

**Pertinent Physical Exam Findings:** Responses oriented but slowed. Dysphonic. Increased WOB. Coarse inspiratory crackles in BLL. Ulcerated Gottron's papules on MCPs/PIPs. Palmar crease hyperpigmentation/ulceration. UE/LE diffusely TTP. Diffuse arthralgia. Muscle weakness, proximal > distal. ROM limited by pain. Reflexes 2+ and symmetric. 3-minute episode of coughing/choking provoked by mild exertion.

**Lab and Imaging Results:** WBCs 4.78, hemoglobin 12.3, ALP 129, CK 52, LDH 566, AST 400, ALT 353, aldolase 17.9. Myositis extended panel unremarkable.

**Differential Diagnosis:** On admission, differential diagnosis included myasthenia gravis, aspiration pneumonitis, methotrexate toxicity, dermatomyositis, and malignancy

**DISCUSSION:** Initially this patient was thought to have aspiration pneumonitis secondary to dermatomyositis-related dysphagia, corroborated by consistent CXR findings. However, she was afebrile and without leukocytosis. Furthermore, the severity and rapid progression of her symptoms seemed more consistent with a highly virulent process such as myasthenia gravis, a particularly severe form of dermatomyositis, or methotrexate-induced toxicity. A high-resolution chest CT showed pathology consistent with dermatomyositis-associated ILD. The patient was started on methylprednisolone and IVIG with progression to rituximab; her condition rapidly improved with treatment.

In patients with known/suspected dermatomyositis and rapidly progressive respiratory difficulty, the possibility of dermatomyositis-related ILD must be thoroughly investigated, even in the absence of high CK levels. In particular, Melanoma Differentiation-Associated gene 5 (MDA-5) dermatomyositis (which can present with normal CK levels, elevated liver enzymes, and no other autoantibodies) must be considered, as it has a greater risk for ILD with rapid progression and high mortality. Patients must be closely examined for the unique clinical manifestations of MDA-5 DM (ulcerated Gottron's papules with palmar involvement, alopecia, oral ulcers, and arthritis), as autoantibody testing is not widely available and rapid initiation of treatment is vital to patient outcomes.



**CASE DESCRIPTION:** The patient is a 59-year-old male with a history of hypertension, diabetes, sleep apnea, and metastatic sacral chordoma who presented to the rheumatology clinic reporting that the fingertips of his left fourth and fifth fingers had become mildly tender, progressively swollen, firm, and had developed a red-purple discoloration over the last 3 months. He had no other associated symptoms and denied any recent trauma, known insect bites, or chemical exposures, and had an unremarkable travel history.

His primary care doctor told him that although he did not have a history of gout, he likely had gouty tophi based on the gross appearance of his lesions and the distal phalanx erosions noted on xray. He was started on colchicine and when his symptoms did not improve, a referral was made to the rheumatology clinic.

**Pertinent Physical Exam Findings:** It was hard to miss his extremely swollen, bulbous, red-purple left 4th and 5th fingertips. The lesions felt very firm and immobile, and were limited to the palmar surfaces. The overlying skin was intact. With the exception of a large surgical scar over his lower back, there were no other abnormalities found on exam.

**Lab and Imaging Results:** CBC/CMP/ESR/CRP unremarkable, Uric acid 6.2mg/dl. Left hand XR showed smooth erosions of the distal fourth and fifth phalanges.

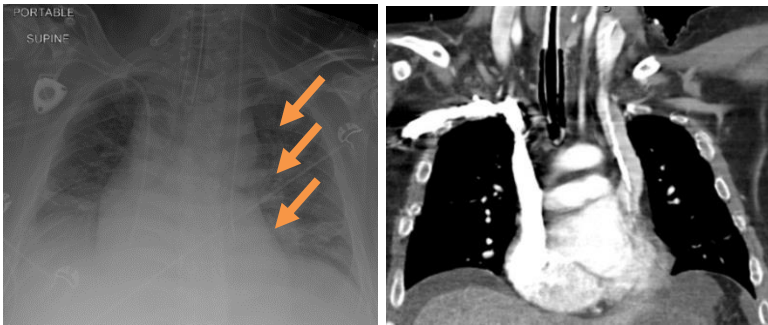
**Differential Diagnosis:** Multicentric reticulohistiocytosis, tumors/metastatic disease, calcinosis, hypertrophic osteoarthropathy, tophi, pernio, emboli.

**DISCUSSION:** While gout can lead to erosive arthritis, his swollen, bulbous, red-purple fingers were not consistent with the characteristic yellow-white firm depositions normally seen in this disease. Furthermore, it would be unusual for tophi of this size to develop over such a short timeline. While an inflammatory condition could present in this manner, a more worrisome condition that more commonly presents this way is metastatic disease. A biopsy was ordered at the conclusion of this visit.

The biopsy returned with results consistent with a chordoma. This patient had been treated in the past for a metastatic chordoma (lungs) originating in the sacrum. Based on the results of our literature search, a case of metastatic chordoma to the fingertips has not yet been reported, making this an exceedingly rare presentation of an uncommon form of cancer.

**CASE DESCRIPTION:** The patient is a 33yo male with a history of Diamond-Blackfan anemia who presented with back pain, abdominal pain, and fevers for about 2 days. A CT of the abdomen showed diffuse stranding of the small bowel without evidence of obstruction or perforation. He was given ceftriaxone and metronidazole, but quickly decompensated on the floor and was transferred to the medical ICU. Despite adequate fluid resuscitation, he progressed to septic shock necessitating placement of a central line for pressors. The left internal jugular vein was selected to preserve the right side in case the need for hemodialysis catheter placement arose. A triple lumen central line was placed using standard procedure. During placement, the wire was confirmed to be in the left internal jugular vein in both cross and longitudinal section on ultrasound. There was good blood return through all ports and no immediate complications. A CXR was ordered to confirm placement.

**Lab and Imaging Results:** VBG from central line: pH 7.20, pCO2 44.8, pO2 53.9, O2hgb 78%



The initial CXR was worrisome for incorrect placement as the line failed to cross midline. A CT chest with contrast was obtained and demonstrated a persistent left superior vena cava (PLSVC) with the left internal jugular central venous catheter terminating at the left superior cavoatrial junction.

**Differential Diagnosis:**

**DISCUSSION:** This is a case of central line placement into a PLSVC. Embryologically, PLSVC is a persistent remnant of the vein of Marshall. This vein fails to regress as development continues resulting in venous blood returning to the right atrium via the connection of the PLSVC with the coronary sinus. In the general population the incidence of PLSVC is 0.3-0.5%. The incidence of PLSVC in Diamond-Blackfan anemia is not known, though these patients do have a 15% incidence of congenital cardiac defects. The initial CXR was worrisome as the patient did not have a prior diagnosis of PLSVC. The differential for a central line that tracks down the left mediastinal border includes placement in the descending aorta, internal thoracic vein, superior intercostal vein, pericardiophrenic vein, pleura, pericardium, or mediastinum. Before confirmatory imaging was obtained, a blood gas from the line at least confirmed venous placement. One can also transduce a CVP from the line to ensure venous placement. Ultimately, cross sectional imaging is required to document this anomaly. There is no contraindication for use of a central line in PLSVC.

**CASE DESCRIPTION:** 46 year old female with a 25 year history of alcohol abuse was found by her husband after falling off the couch. She was very confused, unable to move any of her limbs, and could not talk. She was immediately sent by life-flight to a larger hospital where she was started on a banana bag, high-dose thiamine (500 mg 3 times daily), and CIWA protocol (lorazepam taper) due to her history of alcoholism.

**Pertinent Physical Exam Findings:** Patient noted to be severely malnourished, with altered mental status, dysarthria, bilateral upper and lower extremity weakness, more prominent on the left, bilateral ankle contractures, diffuse hyperreflexia, and decreased sensation to light touch and pain past the mid-calf.

**Lab and Imaging Results:** Thiamine levels were not checked initially, but post-infusion levels were 752 pg/mL (normal range, 345-1485). Liver enzymes showed AST was 60 unit/L (normal range, 9-40), ALP was 124 unit/L (normal range, 40-120), and a normal ALT. Platelets were 144 K/mc, normal range (150-400).

An MRI of the brain without contrast revealed diffuse thinning of the corpus callosum with mild T1 hypointensity in the mid layers with moderate global atrophy and mild scattered foci of T2 signal hyperintensity in the supratentorial white matter and pons. No infarct or hemorrhage was noted. Moderate global atrophy was seen in the ventricles.

**Differential Diagnosis:** Stroke, Wernicke's encephalopathy, and Marchiafava-Bignami Disease.

**DISCUSSION:** Work-up for stroke was negative and MRI findings were consistent with Marchiafava-Bignami Disease. Her elevated AST, ALP, and thrombocytopenia were likely secondary to chronic alcoholism. Patient received high-dose thiamine for 1 week and then 250 mg once daily for 5 days. Patient was also given a short course of high dose steroids to reduce intracerebral swelling. Patient was discharged to an acute neuro rehabilitation center. At follow-up, the patient was noted to have regained her strength and mobility, improved cognition and speech, gained 20 pounds, and quit drinking.

Marchiafava-Bignami Disease is a very rare outcome of chronic alcoholism and malnutrition, characterized by corpus callosum degeneration. Even though the patient's presentation was very compelling for a stroke, incidental findings on MRI revealed demyelination and atrophy of the corpus callosum. As Marchiafava-Bignami is primarily a radiologic diagnosis, it is important to consider rare "stroke mimics" and their diagnostic methods and treatments in chronic alcoholics who present with stroke-like findings. Immediate high-dose thiamine and steroid infusion likely prevented worsening of the patient's encephalopathy and eventually led to a good outcome for this patient.

**CASE DESCRIPTION:** 64-year-old man with history of Crohn's disease, hypertension, aortic insufficiency and CHF who presents with dyspnea, admitted to Cardiology with concern for HF exacerbation. On review of systems, the patient also reports joint pain, specifically involving the right wrist and left foot. He was diuresed with IV Lasix with improvement of dyspnea and was on room air by hospital day 2. However, he subsequently developed persistent fevers. Upon further questioning, he reports a possible insect bite on the right wrist during a recent hiking trip. He also reports an unprotected sexual encounter 10 days prior to presentation.

**Pertinent Physical Exam Findings:** Patient appears non-toxic. No oral lesions. Cardiac exam normal, with no murmurs. JVP is not elevated. Lungs are clear to auscultation. Left forefoot shows a 3x4cm area of swelling and erythema, with normal range of motion of the ankle. 1cm pustule with surrounding erythema on the flexor surface of the right wrist. ROM limited in R wrist, with pain over tendon sheath with movement, but normal strength and sensation. 0.5cm painless scab with erythematous base over the right elbow. Diffuse papular rash over the mid-back. No rashes, lesions or abnormal discharge from the penis.

**Lab and Imaging Results:** WBC 11.52 with 76.3% granulocytes. ESR 50, CRP 10.8. Left foot X-ray with no acute osseous abnormality, moderate subtalar joint OA, and mild soft tissue swelling. MRI of the left foot without drainable fluid collection and no evidence of osteomyelitis. Urine GC/CT testing negative. RPR non-reactive. HIV negative. Blood cultures initially reported as GPC in 1 out of 2 bottles; report later revised to Gram negative diplococci, final speciation positive for *Neisseria gonorrhoea*.

**Differential Diagnosis:** Initially, concern for cellulitis of the left foot vs. osteomyelitis. Given patient's recent unprotected sexual encounter in the context of polyarthralgia, we suspected septic arthritis due to gonorrhoea or other STDs such as syphilis or HIV. Given history

**DISCUSSION:** Patient was empirically started on Ancef. When blood cultures grew Gram negative diplococci, patient was transitioned to ceftriaxone as well as one dose of azithromycin. Fevers resolved and joint pains improved. Patient was discharged after blood culture clearance, with follow-up in ID clinic.

Overall, clinical picture is most consistent with disseminated gonococcal infection with syndrome of dermatitis, tenosynovitis and polyarthralgia. This was likely unrelated to his initial chief complaint of dyspnea