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**Wisconsin  
Chapter**

**2016  
Abstracts**

**Clinical Vignette and  
Research Competition**

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# Case Based Vignettes

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## HYPERTENSION IN A PREGNANT WOMAN

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**Case Description:** This case report describes a 25-year-old G3P0020 female at 36 weeks and 2 days gestation presenting with severe hypertension. She had a 2-year history of hypertension treated with labetalol. She was noted to be hypertensive from the 140s-170s systolic at multiple office visits starting at 11 weeks. She developed episodes of dizziness, sweating and tachycardia during her second trimester. Holter monitor showed sinus tachycardia and transthoracic echocardiogram was normal. Testing returned positive for elevated urine normetanephrine, 24-hour norepinephrine and dopamine levels, and normal metanephrine, 24-hour epinephrine and VMA levels. 24-hour urine protein excretion was also elevated. Of note, she has a positive family history for paraganglioma in her mother. She was started on phenoxybenzamine. Given persistent severe hypertension at 36 weeks, she was admitted to the ICU. MRI abdomen was concerning for extra-adrenal paraganglioma. Nicardipine drip was started, phenoxybenzamine was uptitrated and propranolol was added on hospital day 2. She was delivered by Caesarean section at 37 weeks and 1-day gestational age. This patient remains inpatient at the time of preparation of this report.

**Discussion:** Catecholamine-secreting tumors are a rare but life-threatening cause of hypertension particularly during pregnancy. Pheochromocytomas are intra-adrenal masses, while paragangliomas are extra-adrenal. Diagnosis is made by measurement of urinary and/or plasma fractionated metanephrines and catecholamines; family history may also be of assistance. Strict blood pressure control is essential, particularly in pregnancy, via obtaining alpha blockade before beta blockade. Untreated, this condition causes maternal and fetal mortality rates of 8 and 17 percent, respectively. Timing of surgical intervention is more controversial. In this case delivery by Caesarean section was pursued prior to tumor resection.

## RAPID DISSEMINATION OF BLASTOMYCOSIS IN LATE PREGNANCY

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**Introduction:** Blastomycosis is widely prevalent in the lungs of those living in the Great Lakes region and is widely known to disseminate in the immunocompromised, such as those with AIDS or solid organ transplant recipients. However, in rare cases, a latent infection is activated by the partially immunocompromised state of pregnancy. Given the life-threatening nature of this disease complication, early diagnosis is critical.

**Case:** A 38-year-old female at 35 weeks pregnancy presented to a local ED with 2 weeks of right knee pain, a cutaneous left thigh lesion, and new-onset dyspnea. MRI of knee revealed large effusion with findings consistent with tibial osteomyelitis while CT chest revealed bilateral infiltrates. The patient was taken to the operating room for urgent C-section and I&D of both the right knee and left thigh cutaneous lesion. Post-operatively, she failed to respond to antibacterial therapy and developed severe ARDS. She was transferred to a tertiary care center, where she underwent bronchoscopy and was diagnosed with pulmonary blastomycosis. Retrospective review of original tibial biopsy confirmed the presence of osteomyelitis secondary to blastomycosis, confirming suspicions of dissemination. She later experienced seizures thought to be due to central nervous system infection with blastomycosis, as MRI findings were consistent with such a diagnosis. She was treated with Amphotericin and Voriconazole with subsequent improvement. She was discharged with plan to complete 8 weeks of Amphotericin and 1 year of Voriconazole.

**Discussion:** This case presents an uncommon primary manifestation of blastomycosis in a pregnant patient. The peripartum state of immunosuppression can lead to rapid dissemination and ARDS. Blastomycosis must be considered in the differential of both native joint osteomyelitis and disseminated ARDS among immunocompromised patients in the Great Lakes region.

## NEW ONSET PSYCHOSIS IN YOUNG MAN

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**Introduction:** Anti-NMDAR Encephalitis is a neurologic syndrome with prominent psychiatric manifestations. This autoimmune encephalitis, is often paraneoplastic, post viral, or idiopathic, and leads to limbic encephalitis and frontal lobe dysfunction that can mimic a primary psychiatric disorder. Diagnosis requires a high level of clinical suspicion in order to guide prompt recognition and initiation of appropriate therapy.

**Case:** We discuss a 23 y/o high functioning graduate student with no significant past medical or psychiatric history who presented to the ED after a diarrheal illness with progressive agitation, psychosis, euphoria and suicidal ideation. Patient was initially triaged to psychiatry and discharged on olanzapine. He returned to the ED following a suicide attempt. Urine drug screen was negative; LP demonstrated >100 WBC with lymphocyte predominance with subsequent negative viral serology and bacterial cultures. MRI showed bilateral temporal lobe hyperintensity and leptomeningeal enhancement concerning for meningoencephalitis. Patient was initially treated for presumed HSV encephalitis with acyclovir despite negative HSV PCR. Two weeks after presentation, CSF returned a positive NMDAR antibody, confirming anti-NMDAR encephalitis. Patient was started on high dose steroids and IVIG with improvement though he continued to experience episodes of agitation, psychosis, and catatonia, alternating with periods of lucidity. During episodes of agitation, he demonstrated Kluver-Bucy syndrome, as he would sporadically become hypersexual and impulsive. Due to continued agitation, he was started on rituximab with significant improvement, though far from his baseline. Patient was discharged to neurocognitive rehabilitation. Primary malignancy was never identified despite extensive radiologic and serologic workup.

**Discussion:** Anti-NMDAR encephalitis is part of an expanding group of autoimmune encephalitides that generally affect younger patients (median age of 21 years). Given association with paraneoplastic syndromes, this diagnosis should prompt workup for a primary malignancy. The majority of patients with underlying neoplasm are female with an ovarian teratoma. When present, tumor resection with immunotherapy leads to favorable outcomes. In males it is common that no tumor is discovered. First line treatment for those without identifiable tumors is immunotherapy with IVIG, glucocorticoids, or PLEX. For those failing first line treatment, therapy with rituximab or cyclophosphamide showed improved outcomes, as well as significant reduction in relapses. Despite severity of disease, patients often improve with supportive care support, immunotherapy and lengthy recovery with multidisciplinary care.

## PLATYPNEA-ORTHODEXIA SECONDARY TO PATENT FORAMEN OVALE: A RARE BUT DRAMATIC CAUSE OF RESPIRATORY FAILURE

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**Introduction:** The platypnea-orthodeoxia syndrome is a rare disorder characterized by both dyspnea (platypnea) and arterial desaturation (orthodeoxia) in the upright position with improvement in the supine position.

**Case:** This is a 73 year old female with a past medical history significant for sudden cardiac death status post single chamber ICD, mitral regurgitation, patent foramen ovale (PFO), and chronic lymphoid leukemia who was sent from the PCP office for evaluation of hypoxia and chest discomfort. She reported dyspnea when she is upright, improved when she lay flat. Initial evaluation showed oxygen saturation of 85% on room air, this did not improve with nasal cannula or non-rebreather mask necessitating admission to ICU on BiPAP. Physical examination was benign, EKG showed left atrial enlargement, CT angiogram ruled out pulmonary embolism. Cardiology was consulted, Transthoracic echocardiogram was remarkable only for a positive bubble study. Transesophageal echocardiogram demonstrated a PFO with large right-to-left shunt, aneurysmal interatrial septum and a prominent Eustachian valve directing blood towards the PFO. Right and left heart catheterization ruled out pulmonary hypertension and Eisenminger syndrome or other cardiac pathology. The patient underwent percutaneous PFO closure under guidance of fluoroscopy and intracardiac echocardiography via the right femoral vein using a 30mm Gore septal occluder. She had immediate and complete resolution of her symptoms, normal oxygen saturation on room air,

and was discharged home in two days.

**Discussion:** Platypnea-orthodeoxia is a rare manifestation of PFO, and closure of the atrial defect is curative. Few cases are reported in the literature but the severity of presentation is rarely as dramatic as this case.

## A TYPICAL PRESENTATION OF AN ATYPICAL PROBLEM

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**Introduction:** Cryptogenic organizing pneumonia is a rare and diffuse idiopathic organizing form of interstitial pneumonia. The disease is characterized by acute to subacute onset of vague systemic and pulmonary complaints often leading to a difficult clinical diagnosis of exclusion.

**Case Presentation:** We describe the case of a 77-year-old farmer with a 1-month history of a persistent minimally productive cough with associated fatigue, malaise, dyspnea, and orthopnea found to be hypoxic in clinic in the setting of previously failed macrolide and fluoroquinolone therapy. Admission review of chest x-rays indicated a progression from atypical infiltrates and pulmonary edema to bilateral peripheral infiltrates. A chest CT confirmed parenchymal reticulation, septal thickening, and ground glass opacities consistent with cryptogenic organizing pneumonia. Clinical diagnosis and treatment with high dose steroids lead to improved pulmonary function and exercise capacity.

**Discussion:** Cryptogenic organizing pneumonia is characterized by a variable clinical course and a time to treatment dependent disease severity. Timely diagnosis and early intervention is key to abating a destructive and potentially reversible disease process. The induction of alveolar injury, recruitment of fibroblasts, and excessive proliferation of granulation tissue leads to intraluminal plugs and polyps. The resulting airway consolidation injury often lead to the presenting clinical picture of a nonproductive cough, fevers, dyspnea, malaise, and weight loss. Further investigation frequently identifies peripheral pulmonary infiltrates on imaging as well as a reduction in oxygen saturation and functional capacity of the patient. Pulmonary function tests frequently indicate a reduction in diffusion capacity as well as a restrictive flow pattern. Treatment with glucocorticoids early in the disease course reverses identifiable pulmonary deficits in approximately 66% of patients. Overall prognosis remains positive with early recognition and treatment.

## 5-OXOPROLINE (PYROGLYTTAMIC ACID) ASSOCIATED INCREASED METABOLIC ANION GAP ACIDOSIS: ROLE OF ACETAMINOPHEN

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Acute acetaminophen hepatotoxicity is associated with anion gap metabolic acidosis secondary to lactic acidosis and renal failure. However, severe anion gap metabolic acidosis in patients consuming acetaminophen at therapeutic levels, secondary to 5-oxoprolinemia, is rare and seldom reported.

A 63-year-old severely malnourished female with history of osteoporosis, chronic pancreatitis, diabetes mellitus 2, anemia of chronic disease fibromyalgia and depression was brought in unresponsive by EMS. En route, the patient was intubated since she was found to have agonal breathing and unresponsiveness. She had elevated white blood cell count, acute kidney injury, elevated anion gap metabolic acidosis, and elevation in her beta-hydroxybutyrate of 4.04 initially consistent with diabetic ketoacidosis, as well as septic shock secondary to pneumonia diagnosed on a CT scan performed to rule out pulmonary embolism. She was started on Vancomycin and Levofloxacin for sepsis. Initial arterial blood gas analysis showed a pH of 6.81, PCO<sub>2</sub> of 27, bicarbonate of 3 with an anion gap of 23. Rapid blood screen was negative for alcohol. Salicylate levels were 2.5 (2.0-29.9) and acetaminophen levels were slightly elevated at 13 (0-10). Patient had been taking acetaminophen for chronic pain. Diabetic ketoacidosis treatment protocol was initiated with regular insulin drip and frequent monitoring of fluid status and serum electrolytes. Bicarbonate drip was given for metabolic acidosis. Despite above measures anion gap failed to correct although blood glucose levels returned to less than 200. N-acetylcysteine was started suspecting acetaminophen-related liver toxicity while awaiting urine 5-oxoproline levels. For renal failure and electrolyte disorders, continuous renal replacement therapy was initiated. She continued to stay unresponsive and neuron specific enolase was elevated at 34. Vasopressors (norepinephrine and vasopressin) were started for hypotension refractory to volume resuscitation. Urine 5-oxoproline came back elevated at 8800  $\mu\text{mol/mol}$  creatinine (reference range: <50).

In severely malnourished patients, chronic ingestion of acetaminophen can cause high anion gap metabolic acidosis secondary to elevated blood levels of 5-oxoproline.

## LOCALIZED OCULAR AMYLOIDOSIS: CASE SERIES

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**Introduction:** Immunoglobulin light chain amyloidosis (AL) is a clonal plasma cell neoplasm in which clonal immunoglobulin light chains, either  $\lambda$  or  $\kappa$ , misfold into amyloid and deposit in tissues. The pathogenesis of AL depends on the degree of systemic deposition of amyloid fibrils into vital organs. Localized ocular amyloidosis without systemic involvement is rare.

**Four Cases:** (A) 31 year-old female presented to eye clinic with left ptosis, watery eyes, a left inner eyelid lesion and a foreign body sensation (FBS) in her left eye. Physical exam was unremarkable except for left eye ptosis. Laboratory findings were significant for Congo red positive staining on left upper lid conjunctival excision with liquid chromatography-tandem mass spectrometry (LCTMS) positive for AL (lambda) type amyloid deposition. Workup for systemic amyloidosis was within normal limits. (B) 49 year-old female with migraine headaches presented to eye clinic with left-sided proptosis, left-sided FBS, limited eye movement and binocular horizontal diplopia. Past MRI showed enlargement of left extraocular muscles (EOM) thought to be secondary to orbital pseudotumor and treated with a course of prednisone that did not seem to help. Physical exam was unremarkable except for left eye proptosis, diplopia and enlarged EOM on left side. Laboratory findings were significant for anterior orbitotomy with medial rectus muscle biopsy and Congo red positive staining with LCTMS positive for AK (kappa) type amyloid deposition. Workup for systemic amyloidosis was within normal limits with the exception of mildly elevated  $\kappa$  levels in serum. (C) 69 year-old male with history of coronary artery disease, atrial fibrillation and stroke presented to his PCP with left ptosis for the past month with a change in vision. Physical exam was unremarkable except for left upper lid ptosis with significant visual field changes. Laboratory changes were significant for left orbicularis muscle and full-thickness wedge left upper lid excision with immunohistochemistry subtyping suggestive of AA, however weak staining for  $\lambda$  and  $\kappa$  was also present. Workup for systemic amyloidosis (no serum immunofixation or bone marrow biopsy) was within normal limits. (D) 66 year-old male with history of posterior vitreous detachment, cataract and dermatochalasis of bilateral eyelids presenting to eye clinic with new floaters and flashes in left eye with restricted right EOM. Physical exam was unremarkable. Imaging showed right inferior rectus mass and laboratory changes were significant for Congo red positive right inferior rectus biopsy with LCTMS positive for AL (lambda) type amyloid deposition. Workup for systemic amyloidosis was within normal limits.

**Discussion:** Patients with localized ocular amyloidosis do not appear to be at an increased risk of developing systemic involvement; watchful waiting is appropriate.

## SUCCESSFUL TREATMENT OF LUPUS MESENTERIC VASCULITIS WITH CYCLOPHOSPHAMIDE

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**Introduction:** Lupus mesenteric vasculitis (LMV) is one of the most serious GI complications in SLE. Steroids are considered as a first line therapy; in only few steroid unresponsive cases Cytoxan has been tried. We are presenting a case of LMV which carried therapeutic challenge with pulse dose steroid with ongoing risk for sepsis, but Cytoxan treatment provided symptom improvement.

**Case Presentation:** 39 YO male, PMH of APLS and SLE presented with acute onset abdominal pain, diarrhea. Had stable vital signs. On examination had diffuse abdominal tenderness, no rebound tenderness and normal BS. Normal CBC, CMP, Lipase, infectious, autoimmune, vasculitic workup. INR 4.5. CTA abdomen showed generalized small bowel edema with perienteric fluid around terminal ileum consistent with enteritis, No large vessel thrombosis. Gastroscopy shows severe scalloping and villous loss in duodenum, erythematous friable terminal ileal mucosa with ulcers/exudate and granular colonic mucosa. Histopathology shows mucosal capillary vasculitis, excessive plasma cells in lamina propria, consistent with autoimmune and/or small vessel vasculitis related enteritis. He was started on 1mg/kg/day Steroids. He developed worsening abdominal pain, fever and hypotension. No bowel perforation on repeat CT. Repeat Infectious work up was negative. After stabilization over a period of 72 hours, he received one dose of Cytoxan 800mg with concomitant steroids. His symptoms improved within few days and were discharged home with plan to continue Cytoxan therapy for 3-6 months and slowly wean steroids.

**Discussion:** Prevalence of LMV in SLE patients is 0.2-6.4%. LMV related ischemia carries high risk for infarction and mortality. There are no RTC or guidelines in literature regarding LMV treatment, nevertheless, based on high steroid responsiveness in retrospective studies, it has been considered first line treatment. Cytoxan have been tried in few rare steroid unresponsive situations. LMV carries high risk for gut bacterial translocation and sepsis and use of pulse dose steroids can be challenging, in that situation cytoxan use can help improve symptoms and mortality.

## HSV IN ECZEMA'S CLOTHING

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A 20-year-old female 3 months post-partum with a past medical history significant for asthma, allergies and atopic dermatitis presented to the ED with 2 weeks of fatigue, fever to 103 F, nausea and a progressively worsening rash involving the face, hands, legs and back. On presentation T 99.9 F, BP 82/40 mm Hg, SpO<sub>2</sub>: 92 % on room air, WBC 4.0, CRP 18.9 and CXR was clear. She was fluid resuscitated and started on ceftriaxone and vancomycin. She was transferred to another hospital 2 days later for continued hypotension increasing oxygen requirements and concern for sepsis secondary to skin and pulmonary source. CXR now revealed bilateral patchy infiltrates and she was given a single dose of IV acyclovir for concern of herpetic rash. She was transferred to UW the following day for persistent hypoxemia and specialty care. At our facility, the patient reported a similar rash all her life, usually limited to face and hands. The rash had worsened during pregnancy for which she received oral steroids within last 3 months but had progressed over preceding 2 weeks. Rash was tender without pruritis. She denied a history of HSV and had chicken pox as a child.

On exam, thick erythematous plaques with punched out bases and overlying yellow crusts were noted on her forehead, cheeks, nose and chin, and forearms. Her lower legs had many 2-3 mm monomorphic vesicles with eroded centers and an umbilicated appearance. Some vesicles coalesced into larger plaques with hemorrhagic crusts. HSV1 PCR of fresh lesions was positive and Tzanck was positive for multinucleated giant cells. She was diagnosed with eczema herpeticum and continued on IV acyclovir. Mupirocin 2% ointment was applied for secondary impetigenization and moisturization with white petrolatum was used. Oxygen requirements slowly improved over the course of her 7 day hospital stay, no pathogen was ever identified for her bibasilar pneumonia but she did receive IV zosyn for 5 days. The erosions and plaques eventually sloughed off revealing denuded skin and she was ultimately discharged on oral valacyclovir for 7 days.

This case illustrates eczema herpeticum as one of the dermatological emergencies and the importance of recognizing the characteristic presentation of fever and clusters of pustular vesicles and/or punched out erosions that most often occurs as a complication of atopic dermatitis. It also highlights the importance of prompt lab tests and treatment with acyclovir as this condition has high rates of morbidity and mortality.

## HICKAM'S DICTUM OR OCCAM'S RAZOR? USE PRN!

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Decision making in medicine relies a lot upon heuristics. We present a case in which Hickam's dictum was used initially to explain multiple issues the patient had. However, in the hindsight, after we had more information, it appeared that Occam's razor would have been more appropriate for explanation.

63 yo F with a history of alcoholic liver disease, presented to her PCP with concerns for acute mental status changes and inability to perform ADLs. She was referred to an ED where she was found to have severe hypercalcemia (corrected Ca : 14.98) and elevated ammonia. She was given IV fluids, calcitonin and zoledronic acid and transferred to this hospital. A thorough evaluation of hypercalcemia did not reveal anything specific. CBC showed pancytopenia and so did the peripheral blood smear. SPEP showed a polyclonal gammopathy with normal light chain ratio. Hematology was consulted due to high suspicion for an underlying bone marrow malignancy. Hematology advised that the polyclonal gammopathy was likely a consequence of liver disease and the pancytopenia was likely a consequence of splenomegaly. They further recommended to check RUQ US and screening for Liver malignancy due to history of liver cirrhosis. Both these tests returned negative. However, the next day, the shifts changed and a different hematologist saw the patient, and recommended doing a bone marrow biopsy to evaluate the cause of hypercalcemia and pancytopenia and rule out underlying bone marrow disorder or lymphoma. The final pathology report for bone marrow biopsy was read as non-caseating granulomas consistent with sarcoidosis. Further evaluation revealed that her sarcoidosis was only limited to her Bone marrow. She has been following Rheumatology and is being treated with steroids.

Medical decision making is a complex process and physicians certainly should be aware of the cognitive errors and biases in decision making. Although we may be able to explain clinical cases using either Occam's razor or Hickam's dictum, we should certainly be willing to reconsider our pre-assumptions and challenge ourselves till we find a satisfactory explanation and see actual clinical improvement in our patients.

## SUPRADIAPHRAGMATIC ECTOPIC HEPATIC TISSUE

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**Introduction:** Ectopic liver tissue is a rare developmental anomaly most commonly found incidentally during surgery in intra and retroperitoneal spaces. Only a few cases of supradiaphragmatic ectopias are reported in the literature. Detection of an abnormality by imaging before surgery or autopsy is also unusual. Most of the reported supradiaphragmatic cases are found in neonates causing respiratory distress or hydrothorax. Erroneous dorsal budding of hepatic tissue before closure of the pleuroperitoneal membranes may explain how ectopic liver develops in the thoracic cavity.

**Case:** A 38-year-old generally healthy female presented with a productive cough of 4 days duration with acute dyspnea. She had no chest or calf pain, fever or chills. Vital signs were stable but she demonstrated decreased breath sounds in the right lung field on exam. CXR and subsequent CT revealed a right loculated hydropneumothorax with a small fluid component and several non-parenchymal 1.2-1.4 cm lesions above the right hemidiaphragm. The patient denied any occupational exposures or travel. Pulmonology contemplated a parapneumonic fluid collection, hepatic deposits, or catamenial pneumothorax, however unlikely since she had menstruation 3 weeks prior. ID did not believe this to be infectious. She underwent video assisted thoracoscopy with decortication. Pathology was positive for benign liver tissue with hemosiderin. No malignancy or endometrial tissue was seen. AFP was normal.

**Discussion:** This is an extremely rare case of ectopic hepatic tissue. Not only are the patient's age and diaphragmatic ectopic location impressive, but the majority are found incidentally in asymptomatic adult patients; this ectopia caused a symptomatic hydrothorax making this even more unusual. Most importantly, ectopic deposits have a higher incidence of hepatocellular carcinoma, independent of disease or tumor in the regular liver. Small ectopic liver tissue is thought to have an incomplete functional architecture leading to longer exposure to carcinogenic factors. Awareness of this entity is important for prevention of future malignancies. This patient, therefore, will have close monitoring with consideration for future surgical resection.

## AN UNDERRECOGNIZED CAUSE OF ANION GAP METABOLIC ACIDOSIS

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**Introduction:** The causes of pure anion gap metabolic acidosis are taught early in medical education through the mnemonic MUDPILES. However, this mnemonic overlooks other causes of metabolic acidosis such as oxoprolinemia. Oxoprolinemia is directly linked to the use of acetaminophen, and its diagnosis is likely limited by awareness of clinicians and by availability of testing.

**Case:** A 72-year-old female with a history of CKD stage 3 and chronic back pain presented to the emergency department with a chief complaint of back pain after running out of hydrocodone/acetaminophen, acetaminophen/codeine, and acetaminophen. The patient's daughter reported that the patient was acting confused and somnolent. Review of medications revealed chronic intake of at least 4000 mg of acetaminophen from 3 different sources per day for the past year. A basic chemistry panel revealed a bicarbonate of 9 mmol/L with a serum creatinine of 1.63 mg/dL, a BUN of 38 mg/dL, and an anion gap of 31. A subsequent ABG revealed a pH of 7.25, CO<sub>2</sub> < 20 mmHg. Her workup was negative for lactic acid, volatile alcohols, or other ingestions; there was no evidence of DKA, and a drug screen was negative for salicylates. She was found to be appropriately compensating via Winter's Formula and her Delta-Delta indicated a pure metabolic acidosis. She was started on a bicarbonate infusion which corrected her bicarbonate but her anion gap remained elevated. A urine sample was sent for organic acid evaluation given her long term use of acetaminophen and found to be highly positive for 5-oxoproline. She was started on n-acetylcysteine and IV fluids, acetaminophen was held, and her anion gap slowly recovered over 10 days in the hospital, but did not fully normalize for another 7 days after discharge.

**Discussion:** 5-oxoproline is a byproduct of acetaminophen metabolism that builds up in the blood with continued acetaminophen use. Glutathione depletion and cysteine deficiency occur secondary to chronic use of acetaminophen. Alongside malnutrition, these factors result in the depletion of ATP stores, which leads to the inability to convert oxoproline to glutamic acid. Oxoprolinemia is most likely to be seen in elderly women with chronic kidney disease, and with the increasing prevalence of kidney disease due to diabetes and hypertension, and the ubiquity of acetaminophen-containing pain relievers used daily, it is likely that oxoprolinemia anion gap metabolic acidosis is under-diagnosed due to lack of awareness and access to testing. There is no defined treatment; per case reports n-acetylcysteine is often used given the physiology of glutathione depletion, as well as bicarbonate infusions, with no clear benefit of either. The only proven treatment is abstaining from acetaminophen. High clinical suspicion and awareness remains the key for diagnosis as confirmatory labs tests are not readily available and can take weeks for a final result.

# Research Based Vignettes

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## BLOOD-BASED GENOMIC TESTING FOR NEWLY DIAGNOSED LUNG CANCER PATIENTS TO FACILITATE RAPID TREATMENT DECISIONS

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**Background:** Despite advances in the treatment of lung cancer, it remains a challenging disease to manage. Several patients present at an advanced stage where systemic therapy and biomarker testing are required. From studies on non-small cell lung cancer (NSCLC), 21% of patients had biomarker results available at their initial oncology consultation which lead to shorter median time from consultation to treatment decision (0 vs. 22 days,  $p=0.0008$ ) and time to treatment start (16 vs. 29 days,  $p=0.004$ )<sup>1</sup>. Of those patients with positive EGFR or ALK results, 19% started chemotherapy before biomarker results were available. Our institution's multi-disciplinary team used blood-based genomic testing to expedite treatment decisions and facilitate more informed conversations with lung cancer patients. 1 Lim, C., et.al. 2015. Biomarker Testing and Time to Treatment Decision in Patients with Advanced Non-Small Cell Lung Cancer. *Ann Oncol* first published online April 28, 2015

**Methods:** Commercially-available, blood-based genomic testing was ordered for all clinical patients that includes genomic test, GeneStrat, included a targeted panel for EGFR sensitizing and resistance mutations, ALK fusions, KRAS and BRAF mutations.

**Results:** Of the patients ( $n=32$ ) submitted for genomic testing with results were available within 72 hours of blood draw. This is a cohort of patients that were diagnosed with adenocarcinoma between January to June of 2016. Amongst these patients, ~ 28% ( $n=9$ ) had a mutation identified by GeneStrat (KRAS G12D=4, EGFR T790M=4, EML4-ALK=1, EGFR L858R=1). One patient had dual EGFR L858R/EML4-ALK mutations identified by the test. There was one patient in whom the test was not able to identify G719A (exon 18) as the test looks for only exon 19,20 and 21. The sample size was limited for positive predictive value but the negative predictive value is 94%.

**Conclusions:** Blood-based genomic provides valuable treatment information regardless of disease stage. Early identification of the mutations will benefit the patient with early initiation of specific chemotherapy.

## BIAS IN THE EYES OF RESIDENT PHYSICIANS

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**Introduction:** The utilization of patient characteristics can allow health-care providers to arrive at diagnosis or decide on treatment options, however, the subjective nature of patient characterization can negatively affect patient care. A 2003 Institute of Medicine (IOM) report, *Unequal Treatment*, recognized that bias or stereotyping may affect provider-patient communication or the care offered. We investigated residents' recognition of bias.

**Method:** We indirectly assessed recognition of bias among resident physicians by asking their opinion in an anonymous manner about their fellow residents. We asked residents the following two step question; "Have you observed a colleague of yours SAY, PORTRAY, or ACT in a biased manner towards a patient while providing inpatient service?" If the answer was yes, we subsequently asked them to elaborate on the bias.

**Result:** The survey was sent to thirty nine postgraduate (PG) internal medicine residents, in their 1st to 3rd year of training. Half of the responders (20/39) were female. The response rate was 100%. Forty six percent (18/39) reported observing their colleague(s) being biased toward patients. Of those who reported bias, 77.8% (14/18), reported one or more examples about the content of the perceived bias. The largest category, 42.8% (9/21), was about bias towards patients with past or current "drug/substance abuse" or "narcotic seeking" behavior; 14.3% (3/21) involved patients with repeated admissions or so called "frequent fliers"; 9.5% (2/21) related to race/ethnicity; 14.3% (3/21) indicated providers not wanting to care for patients who were perceived to be "difficult". Interestingly, another 9.5% (2/21) reported witnessing preferential service for "affluent/VIP" patients. Other examples included bias against obese patients, female patients, and general stereotyping with no specifics given.

**Conclusion:** Given the evidence that implicit bias can be recognized and improved up on, this study reinforces the need for implicit bias training/discussion to be included in residency programs.

## REDUCING CLABSIs IN PEDIATRIC ONCOLOGY PATIENTS

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**Background:** Central line associated blood stream infections (CLABSIs) are preventable, hospital-acquired conditions that increase morbidity, mortality, length of stay, and health care costs. Implementation of central line insertion and maintenance bundles have reduced but not completely eliminated CLABSIs. Daily treatment with chlorhexidine gluconate (CHG) antiseptic has been shown to reduce CLABSIs in a variety of populations including adult, pediatric, and neonatal intensive care, burn, adult medical and surgical, and long term acute care units. We hypothesized that daily CHG treatments would reduce the incidence of CLABSIs in pediatric oncology and bone marrow transplant (BMT) patients.

**Methods:** All pediatric oncology and BMT patients received daily treatment with 2% CHG-impregnated cloths during the one-year intervention period unless contraindicated. All primary blood stream infections in patients with a central line during this period (wCHG) and the preceding twelve months (preCHG) were recorded as a CLABSI. CLABSI rate was calculated as events per 1000 central line days. Cultured pathogen, microbe sensitivity and CHG compliance were also collected. Patient characteristics were evaluated to determine relationship to mucosal barrier injury (MBI) per CDC criteria.

**Results:** Compliance with CHG treatment remained >90% over the wCHG period. The CLABSI rate did not improve with CHG use (2.90 preCHG v 3.39 wCHG). Most patients affected were undergoing treatment for hematologic malignancy (*hematologic* 11 preCHG v 15 wCHG, BMT 2 preCHG v 3 wCHG, *solid tumor* 3 preCHG v 2 wCHG). There was no alteration in the type of pathogens isolated (*Gram positive* 6 preCHG v 6 wCHG, *Gram negative* 7 preCHG v 13 wCHG, Fungus 3 preCHG vs 0 wCHG) or incidence of antibacterial-resistant infections (*Vancymycin resistant enterococcus* 1 preCHG v 0 wCHG). A large proportion of CLABSIs qualified as an MBI in both intervention periods (11/16 preCHG v 15/20 wCHG). The Non-MBI CLABSI rate still remained unchanged with CHG use (0.91 preCHG v 0.85 wCHG). However, 100% of Non-MBI CLABSIs in the wCHG period had mucositis or neutropenia and were not classified as an MBI based only on growth of non-intestinal organisms (*mucositis or neutropenia* 100% all MBIs, 40% preCHG NonMBI, 100% wCHG NonMBI).

**Discussion:** CHG did not appear to reduce CLABSIs based on strict infection-source definitions. However, all patients with CLABSIs in the wCHG period had severe neutropenia and/or mucositis. There were few CLABSIs in immunocompetent patients receiving CHG treatment, specifically, patients undergoing chemotherapy for a solid tumor. This suggests an endogenous source of bacteremia in severely immunocompromised patients.

## UTILITY OF A REMOTE IMAGE ACQUISITION AND FEEDBACK TOOL IN PROMOTING POINT-OF-CARE ULTRASOUND SKILLS AMONG CRITICAL CARE TRAINEES

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**Introduction:** Point-of-care ultrasound (POCUS) is increasingly incorporated into both clinical practice and training programs. Implementation of POCUS curriculums is challenged by a limited number of experts available to teach ultrasound skills and oversee interpretation at the bedside. Recent image database software products now allow faculty members to remotely supervise and teach learners by reviewing recorded, interpreted studies followed by provision of feedback on the POCUS skill components of image acquisition, image interpretation, and clinical application skills. We sought to assess the level of basic critical care echocardiography (BCCE) skills attained among critical care trainees after introduction of a remotely supervised POCUS curriculum.

**Methods:** A POCUS curriculum incorporating hands-on training was introduced in July of 2015. A structured BCCE exam with required image set was taught along with a process for using an ultrasound archiving and quality assurance software product to record, interpret, and submit performed exams. In September of 2015, trainees began wirelessly submitting independently performed exams for remote over-read by a faculty member who was an expert in critical care ultrasonography. After a 6 month period, an analysis of all submitted and over-read studies was performed with a primary focus on appropriateness of clinical application of ultrasound findings. Image quality and interpretation scores were also evaluated.

**Results:** 81 BCCE exams were performed and submitted for over-read. 72% of all submitted studies were graded as having good or excellent image quality. Overall diagnostic accuracy was 77%. Accurate interpretation of ultrasound exams was associated with appropriate clinical application in greater than 90% of cases. In cases of inaccurate interpretation of ultrasound images, 25% were associated with inappropriate clinical application. Incorrect clinical applications included inappropriate fluid management (60%) and use of inotropic agents (40%).

**Conclusion:** After introduction of a POCUS curriculum founded upon remote image over-reading with written electronic feedback, critical care trainees achieved high levels of image acquisition, interpretation accuracy and appropriateness of clinical application.

# Displayed Posters

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## 1) PULMONARY HEMORRHAGE LIKE NO OTHER: A RARE CASE OF PRIMARY PULMONARY ANGIOSARCOMA

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Angiosarcomas are malignant endothelial cell tumors of vascular or lymphatic origin, and they account for approximately 2% of all soft tissue sarcomas. An angiosarcoma found in the lung most likely represents metastasis from another primary site. In contrast, primary pulmonary angiosarcomas are extremely rare with only a few cases documented in the literature.

We describe a case of an 85-year-old female with no history of pulmonary pathology, who presented with worsening cough, hemoptysis and exertional dyspnea over the preceding 6 weeks. Her vital signs were within normal limits, other than mild hypoxia. Examination was unremarkable except for diffuse, coarse lung crackles bilaterally. Pertinent laboratory results revealed a hemoglobin of 7.8 g/dL; white blood cell count of 12.6 103/uL; International Normalized Ratio of 1.2; C-reactive protein of 8.0 mg/dL; negative anti-nuclear antibody panel and a weakly positive antineutrophil cytoplasmic antibody panel. Chest X-Ray demonstrated moderately-extensive areas of air-space opacities in the mid and upper lung zones with small bilateral pleural effusions, suggestive of organizing pneumonia or multifocal pulmonary hemorrhage. Chest computerized tomography revealed ground glass opacities and pulmonary nodules. Bronchoalveolar lavage was pursued, and revealed diffuse pulmonary hemorrhage. The patient continued to deteriorate and passed away on the 7th day of hospitalization. Autopsy revealed a primary pulmonary epithelioid angiosarcoma with metastatic spread to the left adrenal gland. The pulmonary origin of the tumor was confirmed by specialized immunohistochemistry staining.

Patients with primary pulmonary angiosarcoma can present with varying symptoms, and the work up often yield results that mimic other pathologies. Reaching the diagnosis is currently not possible without a tissue biopsy. Given the profound rarity of this malignancy and its late presentation, no proven treatment modality exists. Mortality is 100% within a few months of presentation.

## 2) PRIMARY HYPOTHYROIDISM PRESENTING WITH MYXEDEMATOUS ASCITES

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**Introduction:** Ascites as a presenting symptom of primary hypothyroidism is rare occurring in <1% of cases. An uncommon complication of hypothyroidism, myxedema ascites is often diagnosed after lengthy workups. Treatment with levothyroxine leads to complete resolution of true cases. This is the first documented presentation of myxedema ascites in a solid organ transplant recipient.

**Case:** A 61 year old male presented initially to primary care with complaints of generalized weakness, dyspnea, and weight gain over five months. The patient was referred to transplant nephrology given medical history of end stage renal disease secondary to type 1 diabetes mellitus status post kidney-pancreas transplant in 1991 complicated by post-transplant lymphoproliferative disease in remission. Initial findings were notable for anasarca, bulging flanks, abdominal fluid wave, pitting edema, and positive cardiac enzymes. He was admitted for acute coronary syndrome rule out. Further workup revealed albumin of 2.6 g/dL, TSH of 186 uIU/mL, and free T4 of 0.1 ng/dL. Chest x-ray demonstrated bibasilar pleural effusions. Serial electrocardiograms remarkable for sinus bradycardia with low voltage QRS. Transthoracic echocardiogram was normal and diagnostic paracentesis revealed fluid low in protein with a serum-ascites albumin gradient (SAAG) of 1.5g/dL. Aggressive diuresis with albumin and furosemide was begun and levothyroxine immediately initiated at half maintenance dose. Complete abdominal ultrasound revealed normal liver morphology/hemodynamics and a benign LLQ peri-pancreatic cyst. Complete hepatitis serology for HBV, HAV, and HCV were negative along with normal liver function tests, ceruloplasmin, alpha-1-antitrypsin, anti-nuclear/ mitochondrial/ smooth muscle antibodies, and serum protein/ immunoglobulins. Patient was discharged with outpatient follow up and documented symptom resolution at one months' time.

**Discussion:** Primary hypothyroidism rarely presents with ascites. This clinical finding in a patient with history suggestive of hypothyroidism should prompt thyroid testing. Diagnostic workup should include a paracentesis and determination of the SAAG. Review of the literature revealed sixty three well documented cases of myxedema ascites. A majority of these had SAAGs of >1.1g/dL and total proteins of >2.5 g/dL. Myxedema as a cause of ascites should be considered and ruled in by exclusion. Treatment with thyroid replacement remains an effective solution with an excellent prognosis.

### 3) BACILLUS CALMETTE-GEURIN (BCG) ASSOCIATED GIANT CORONARY ARTERY ANEURYSM

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**Introduction:** Documented cases of mycotic aneurysm (MA) secondary to bacillus Calmette-Guérin (BCG) immunotherapy in the adjunctive treatment of bladder cancer are exceedingly rare. Twenty five cases of MA after BCG instillation have been documented in the literature. This report describes the first occurrence of BCG associated mycotic coronary artery aneurysm (CAA).

**Case:** A 63 year old male with history of disseminated Mycobacterium bovis infection was transferred to medicine from the vascular surgery service for management of malnutrition. He was three years post transurethral resection of a T1 transitional cell carcinoma treated with adjunctive intravesicular BCG. Over the past four months, he developed recurrent bilateral lower extremity MAs secondary to disseminated Mycobacterium bovis. At transfer, patient was being treated with a three drug anti-tuberculin regimen and nutritional needs managed via N-G tube feeds and TPN. During hospitalization on medicine, a rapid response was called for hypotension and presyncopal symptoms. Examination revealed a new systolic murmur over the right costal border in a cachectic appearing male. Cardiology was consulted for elevated cardiac enzymes without EKG changes, thought to be demand ischemia due to hypotension and anemia. Due to concerns of endocarditis, the patient underwent transthoracic echocardiogram which was unrevealing. Eleven days later, the patient developed new popliteal pain and fever. CT angiogram (CTA) of the abdomen, pelvis and lower extremities demonstrated a popliteal hematoma and incidentally discovered a large coronary mass. CTA of the chest revealed a 6.1cm x 5.3cm CAA that was subsequently defined via angiography as a partially clotted aneurysm of the LAD artery. Review of prior imaging showed evidence of a smaller aneurysm on initial CTA chest. After discussions with infectious disease, surgery and cardiology, medical management was pursued with a six week course of five IV anti-tuberculin agents. Nine month course of oral agents continued with planned surgical re-evaluation after completion.

**Discussion:** To the best of our knowledge, this is the first documented case of BCG associated coronary artery aneurysm. CAAs are classified by etiology and size, with those larger than 5cm classified as giant. In the general population, the incidence of CAA is 1.4% - majority caused by atherosclerosis, inflammatory disease, or trauma. Mycotic CAAs account for < 3% of cases and are often associated with infective endocarditis. BCG immunotherapy carries a small risk of disseminated infection and an incredibly rare chance of MA. This is the first reported case of giant mycotic CAA due to BCG. Close collaboration with surgical and medical subspecialties is required to determine individualized management for the patient.

### 4) 6,025 MILES WEST OF THE NILE

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A 59-year-old male with a history of rheumatoid arthritis treated with immunosuppression was admitted to an outside hospital with 4 days of viral syndrome-like symptoms, dyspnea, and right arm weakness. He was diagnosed with pneumonia, stroke evaluation was negative and ceftriaxone was initiated. Over the next day, he rapidly developed global weakness. He was transferred to the MICU of a nearby hospital with flaccid paralysis in all extremities, altered mental status, and respiratory failure requiring endotracheal intubation. His presentation was attributed to Guillain-Barre syndrome (GBS). Infectious and paraneoplastic workup was unrevealing. Initial brain MRI was unremarkable. Repeat brain MRI three days later showed dural enhancement. Spine MRI revealed hyperenhancement of the anterior cervical cord. CSF revealed lymphocytic predominance, elevated protein, and low glucose. After empiric antibiotics, stress-dose steroids, and 5 days of plasmapheresis, his condition was unimproved. The patient was transferred to our institution for further evaluation. CSF was positive for West Nile Virus IgM. A diagnosis of West Nile virus encephalomyelitis was made, treatment with IV Immunoglobulin was begun, and supportive cares were continued. The patient had minimal improvement during his stay. At the time of discharge, he was non-verbal, opened his eyes spontaneously, localized gaze to voice, and had minimal movement of his tongue, mandible, and toes. He required nutrition via gastric tube and mechanical ventilation via tracheostomy.

This case illustrates the importance of suspicion for arbovirus infection when evaluating neurological processes in an immunosuppressed patient population. Moreover, it highlights the importance of distinguishing an auto-immune process like GBS from an infectious process like West Nile virus, as this distinction is paramount to appropriate, potentially disease-altering treatment.

## 5) A CASE OF CRITICAL ANEMIA WITH MULTIPLE SEVERE VITAMIN DEFICIENCIES

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**Introduction:** Anemia in the elderly is common with iron deficiency secondary to GI losses being one of the most frequent causes. Other causes, like vitamin deficiencies, are less common but must always be considered.

**Case Description:** An 81 year-old male with a history of alcohol abuse presents after falling at home; he was unable to get up due to generalized weakness. The patient reported drinking “a couple” beers on most days but heavier use was suspected. On physical exam, he was pale with conjunctival pallor, a positive conjunctival ring sign, a flow murmur, and a MOCA score of 17/30. Laboratory studies demonstrated pancytopenia with a WBC of 3.9, hemoglobin level of 3.4, platelet count of 60,000, and an MCV of 119. He was given 4 units of packed red blood cells. Additional studies found ferritin to be normal, B12 <60 (normal: 200-500 pg/mL), folate 4.2 (7.0-31.4 nmol/L), vitamin A 36 (38-98 mcg/dL), 25-hydroxy vitamin D 5.7 (30-100 ng/mL), INR of 1.5; homocysteine and methylmalonic acid were both elevated (51.3 [5.8-11.9  $\mu$ mol/L] and 19.4 [0-4.7  $\mu$ g/dL], respectively); albumin and prealbumin were both low (2.7 [3.5-5.5 g/dL] and 12.5 [20-40 mg/dL], respectively). Parietal cell and intrinsic factor blocking antibodies were negative as was TTG IgA with normal total IgA levels. Further workup of the etiology of this patient’s vitamin deficiencies is ongoing.

**Discussion:** We present a case of multiple severe vitamin deficiencies and protein calorie malnutrition. Such vitamin deficiencies are rare in the U.S. and, when found, should raise suspicion for a malabsorption syndrome such as celiac disease. In the absence of evidence for malabsorption, poor nutrition was considered most likely in this case coupled with alcoholism. In the U.S., alcoholism is one of the main risk factors associated with severe malnutrition and vitamin deficiencies.

## 6) ACQUIRED VON WILLEBRAND’S IN ESSENTIAL THROMBOCYTHEMIA

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**Introduction:** Acquired Von Willebrand’s Disease (aVWD) is a rare bleeding disorder often associated with myeloproliferative neoplasms (MPNs). Essential Thrombocythemia (ET) is a MPN that results in a persistent and isolated thrombocytosis<sup>1</sup>. Patients with ET can suffer a variety of complications, including both thrombosis and bleeding, the latter of which is likely secondary to aVWD as platelets bind the highest weight vW multimers.

**Case Study:** The patient was a 75 year old male who first presented with thrombocytosis in 2011. He first established with hematology in November of 2015: at that time, he was asymptomatic, but his platelet count was 786,000. Bone marrow biopsy and JAK-2 panel were consistent with ET.

Within a week of his biopsy, the patient awoke with severe disequilibrium, alert and oriented only to person. His wife promptly brought the patient to the ED, where workup was notable for a platelet count of 900,000 and a left intraventricular hemorrhage on CT Head. Ristocetic cofactor activity panel returned with activity at 48% (normal 60-130%). He was started on hydroxyurea 1500 mg to treat thrombocytosis secondary to ET.

His platelet count improved with treatment; repeat ristocetic cofactor assay revealed improvement in VWF activity to 89%. A few days later the patient suffered a massive pulmonary embolism which resulted in his death.

**Discussion:** This is a very classic presentation of aVWD. The patient was initially asymptomatic with no history suggesting coagulopathy until his platelet count skyrocketed<sup>3</sup>. His coagulopathy resolved with resolution of his thrombocytosis, as proved by his repeat ristocetin cofactor assay<sup>3</sup>. He also suffered both an intracranial hemorrhage and a pulmonary embolism. Patients with aVWD secondary to MPN are at higher risk of both thrombosis and bleeding<sup>4</sup> which presents a special dilemma for clinical management<sup>5</sup>. It also argues for the importance of trending platelet counts; when thrombocytosis resolves, so does the coagulopathy and anticoagulation should be reconsidered.

In conclusion, aVWD is a rare disorder most commonly seen in the elderly that can be caused by a variety of underlying diseases including ET. Most patients present with mucocutaneous bleeding but they can present with severe bleeds. It is important to trend platelet counts while in house as patients are at risk for both thrombosis and bleed. When the platelet count normalizes, these patients should not be considered coagulopathic. It would be prudent to consider resuming prophylactic anticoagulation at this point in time.

## 7) CEFEPIME-INDUCED ENCEPHALOPATHY IN A PATIENT WITH ESRD

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**Introduction:** Cefepime is an antibiotic used empirically for many severe infections. The drug is primarily excreted (~85%) by the kidneys. The incidence of Cefepime-induced encephalopathy is unknown as it is often under recognized or not reported. As the use of Cefepime is steadily increasing, it is important to recognize this clinical entity, especially in a patient with end stage renal disease (ESRD).

**Case:** A 63 year old male with a history of DM type 2, COPD, CVA, ESRD on hemodialysis (HD), presented with increased drainage from a plantar foot ulcer, accompanied by a temperature of 100.4 and subjective chills. He was admitted for suspected osteomyelitis that was confirmed by CT scan showing cortical destruction and sclerosis. Infectious disease was consulted and he was treated with empiric IV cefepime (2g) and vancomycin (1g). Three days later vascular surgery performed a toe amputation and first metatarsal resection and sent bone biopsy for culture. Cultures showed no growth but the decision was made to empirically treat for 6 weeks. On hospital day 8, the patient became confused during HD and had difficulty grasping objects with his right hand, CT scan of head was obtained that was negative for acute intracranial pathology. He had no further symptoms. Two days later, he again became confused and inattentive, with a failure to return to baseline. He was disoriented with intermittent expressive aphasia, delayed responses to commands, and agitation of variable severity. Neurological examination was limited, but no focal deficits were apparent on daily exam. He had no symptoms to suggest infection as the cause of acute mental status changes. His wound was healing well. Laboratory tests were unremarkable except for a high TSH and slightly low T3. Brain MRI and EEG did not reveal the cause of his encephalopathy. All sedating and psychotropic medications were held with minimal improvement. Dose of thyroxine was increased as well with no change in mentation. On careful review, the only other contributing factor was cefepime. Apparently, for two days following surgery the patient received it without being renally dosed. Cefepime was discontinued immediately (hospital day 12). The patient's mental status slowly improved and two days later the patient was back to his mental status baseline without any neurological sequelae. He was switched to ertapenem and vancomycin to complete the course. He was alert, awake and oriented x4 at the time of discharge.

**Discussion:** Cefepime is a parenteral fourth-generation cephalosporin used frequently for broad coverage of severe infections. The drug can lead to drug accumulation in patients with impaired renal function. Cefepime-induced neurotoxicity has been reported to cause encephalopathy, myoclonus, seizures, non-convulsive status epilepticus and mortality. Most affected patients have some degree of renal impairment or incorrect dosing, but cases have also been reported in patients with normal renal function. Symptoms usually present between 1 and 10 days, and resolve within 2 to 7 days after discontinuing the antibiotic. We report a case of cefepime-induced encephalopathy in a patient with ESRD in whom the symptoms resolved almost exactly two days after cessation of the antibiotic and clearance with HD. Cefepime toxicity should be suspected if a patient experiences a change in mental status or myoclonus, after exclusion of other possible causes. If not recognized, cefepime induced-encephalopathy can have severe, sometimes fatal outcomes.

## 8) A PERICARDIAL EFFUSION WITH A CLASSIC TRIAD

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**Introduction:** Adult onset Still's disease (AOSD) is a rare systemic inflammatory disorder of unknown etiology. The estimated prevalence of AOSD is 1/100,000 population with an equal distribution between the sexes. Diagnosing AOSD is challenging due to the presence of several nonspecific signs and symptoms and absence of specific serological biomarkers. In a patient with a pericardial effusion and systemic symptoms, AOSD should be considered in a differential diagnosis.

**Case:** We discuss a 46 year old female who presented to the emergency department with chest pain, shortness of breath, and cough in the setting of a six month history of fevers, night sweats, 40 pound weight loss, polyarthritis, rash, and benign lymphadenopathy s/p two previous unremarkable lymph node biopsies. Prior workup demonstrated mild elevation of ANA and RNP antibodies; however, evaluation of anti-smith, dsDNA, chromatin, SSA/SSB, Jo-1, C3/C4, and ANCA was negative. Previous infectious workup was unremarkable. Initial evaluation at our institution revealed persistent of elevated inflammatory markers, including leukocytosis to 14.3 (>80% segs), and bilateral knee effusions. CXR showed an enlarged cardiac silhouette. Transthoracic echocardiogram demonstrated a moderate pericardial effusion with no evidence of tamponade. The patient underwent a left knee arthrocentesis that demonstrated a non-infectious, inflammatory process. Given the aforementioned findings, the diagnosis of AOSD was made and oral prednisone therapy was started. Four weeks after discharge, the patient had symptomatic improvement; however, daily fevers persisted thus she was started on Anakinara (IL-1 inhibitor) and meloxicam with resolution of fevers.

**Discussion:** AOSD is an inflammatory disorder characterized by a triad of symptoms including high fevers, arthritis, and an evanescent rash. AOSD is a diagnosis of exclusion. Yamaguchi criteria can aid in diagnosis and has 96% sensitivity and 92% specificity. Serosal involvement occurs in 25% to 60% of AOSD cases. Cardiac involvement is significant with pericarditis occurring in 10% to 40% of cases and is complicated, in approximately 20% of patients, by pericardial effusion or cardiac tamponade. Current therapies for AOSD include NSAIDs, corticosteroids, and DMARDs. The prognosis is generally favorable, but relapse rate is high. Prompt consideration of AOSD in work-up of a pericardial effusion can avoid unnecessary procedures and start early treatment to avoid potential complications such as cardiac tamponade.

## 9) PJP PNEUMONIA IN A NON-HIV HOST

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**Case:** A 62 year old man with a living unrelated renal transplant in 2008 for polycystic kidney disease and cryptococcal meningitis in 2012 presented with hypoxic respiratory failure. He complained of fevers, fatigue and progressive dyspnea for the past several weeks. Relevant medications included azathioprine 75mg, prednisone 10mg and valgancyclovir 450mg daily, and tacrolimus 0.5mg BID. On physical examination, he had diffuse rales and was alert and oriented x3. He underwent a bronchoscopy with BAL at an outside hospital prior to transfer and PJP PCR was positive. CXR and CT chest showed diffuse patchy parenchymal infiltrates.

**Hospital Course:** He initially received primaquine and clindamycin due to risk for hyperkalemia with Bactrim along with high dose IV steroids for 4 days prior to transfer. Upon transfer, his oxygen requirements acutely increased from 3L to 15L. He was switched to IV Bactrim and IV steroids were continued. He continued to require high-flow nasal cannula and intermittent BPAP. He desaturated with any movement. Despite 10 days of IV Bactrim, his respiratory status worsened. Per infectious disease recommendations, IV Micafungin was added. He was intubated due to respiratory fatigue. The following day, he became persistently hypotensive and tachycardic despite 3 pressors and became non-responsive. Comfort care was pursued and he unfortunately passed away.

### Discussion:

-Patients with hematopoietic cell or solid organ transplant, cancer, connective tissue disease, those receiving steroids, chemotherapy or other immunosuppressants have a substantial risk of PJP pneumonia.

-The incidence of non-HIV PJP pneumonia is increasing.

-Non-HIV patients with PJP pneumonia do worse than HIV patients as PJP pneumonia rapidly progresses in these patients compared to HIV patients.

-In a retrospective study of 72 patients, non-invasive ventilation (NIMV) failed in 71% of HIV-negative patients vs 12% of HIV patients. Mortality was higher in non-HIV patients (47% vs 17%, OR 3.73, CI 1.1-12.60). 80% of HIV-negative patients with NIMV failure died.

-Adding echinocandins may worsen outcome.

-PJP prophylaxis in immunosuppressed or transplant patients may be important. In a meta-analysis, incidence of PJP pneumonia was reduced by 91% (NNT of 15) in this patient cohort.

## 10) GRANULOCYTIC SARCOMA CAUSING SEVERE INTRACEREBRAL BLEED IN A CML PATIENT

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**Introduction:** Granulocytic sarcomas are rare, destructive, extramedullary tumors that consist of immature granulocytic cells. They present more commonly in association with AML compared to CML. These tumors can manifest prior to, during, or upon relapse of the underlying myeloid disorders. The location of the tumor varies extensively and can cause complications secondary to severe bleeding and/or mass effect.

**Case Presentation:** 59 yo white male with no relevant past medical history, was referred to heme clinic due to abnormal blood counts at an annual exam including a wbc of 20,000, hgb of 7.6 and plts of 78,000. His wbc further increased to over 400,000 on follow up labs. There were obvious concerns for an evolving hematological malignancy and he was admitted to the hospital to initiate management. Patient was presumed to have promyelocytic leukemia and was started on trans-retinoic acid (ATRA), hydroxyurea and high dose steroids, followed by bone marrow biopsy. Patient was asymptomatic initially and responded partially to these treatments. Peripheral blood RT-PCR and bone marrow FISH showed a positive BCR-ABL translocation that confirmed CML following which ATRA was discontinued. On day 4 of hospitalization, he was symptomatic with severe headache/bradycardia. CT head revealed multiple high density masses in the brain. These findings were most consistent with several granulocytic sarcomas with superimposed hemorrhage. Unfortunately, the patient developed cerebral herniation and deteriorated and life support was later withdrawn per family's wishes.

**Discussion:** Myeloid sarcoma is a rare disease that can present as an isolated extramedullary leukemic tumor, concurrently with or at relapse of AML, CML or myelodysplastic disorder. This is reported in 2-8% of patients with AML and has an extremely rare association with CML. Pathological reason is thought to be an abnormal homing signal for the leukemic blasts that prevents BM localization. Most common signs and symptoms associated with these tumors include localized mass effects like severe pain and spontaneous bleeding. Diagnosis involves peripheral and bone marrow chromosomal studies, cytochemical analysis and radiological studies. Early initiation of chemotherapy is the most successful approach with results comparable to chemotherapy treatment in AML patients. Other approaches with local therapies, stem cell transplant and targeted approach are still under consideration.

## 11) BLOOD-BASED PROTEOMIC TESTING FOR NEWLY DIAGNOSED LUNG CANCER PATIENTS TO FACILITATE PROGNOSTIC CONVERSATIONS

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**Background:** Despite advances in the treatment of lung cancer, it remains a challenging disease to manage. Several patients present at an advanced stage where systemic therapy and/or newer tyrosine kinase inhibitors are needed. In the early 2000s, Paul Bunn and David Carbone lead a team to develop VeriStrat, because they wanted a test that would predict patient response to the new EGFR TKI therapies. They had patients who had long-term stable response to the EGFR TKI or good response, and they had patients who appeared not to have any response to the TKI or poor response. The poor-responding patients progressed rapidly through the EGFR TKI therapy. To test these patients, they put the patient's serum samples through a mass spectrometer. Based on the number of peaks on spectrometer they are classified into patient who respond well (VeriStrat – Good) and poorly (VeriStrat – Poor). The host response to the tumor, measured by a VeriStrat Poor signature, is a reaction to a new growth pathway that has been invoked by the tumor. As we will show with data, the additional growth pathway connotes an aggressive tumor that responds poorly to most therapies in NSCLC. We also believe the mechanism is the same for different tumor types and extended testing to different stages.

**Methods:** Commercially-available, blood-based proteomic testing was ordered for all NSCLC patients from January to June 2016. The proteomic test, VeriStrat, provided prognostic information for outcomes and predictive information for Chemotherapy/TKI therapy benefit using a classification of Good or Poor. Testing results were used to make treatment decisions and to aid in prognostic conversations with patients.

**Results:** Of the patients (n=36) submitted for genomic and proteomic testing, with results available within 72 hours of blood draw. This is a cohort of patients that were diagnosed with NSCLC between January to June of 2016. Amongst these patients, ~ 80.5% (n=29) had VeriStrat good diagnosis and ~ 19.5% (n=7) had VeriStrat poor diagnosis. Out of the total VeriStrat good patients 90% (predictive value) of the patients did well on conventional / TKI chemotherapy. 71.4%(predictive value) did poorly who had the VeriStrat poor test.

**Conclusions:** VeriStrat test provides prognostic information for newly diagnosed lung cancer patients. This can facilitate a more informed conversation with patients.

## 12) UNUSUAL CASE OF TICKBORNE ILLNESS: A CASE OF ANAPLASMOSIS

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**Introduction:** Anaplasmosis is a tickborne disease caused by the obligate intracellular bacterium *Anaplasma phagocytophilum*. Cases are typically seen in the upper Midwest and Northeastern parts of the United States. It presents as a febrile illness with symptoms including cough, diarrhea, vomiting, malaise, and arthralgia. However, non-specific clinical presentations of this illness pose a challenge for physicians to diagnose.

**Case:** A 52 year old male with no significant past medical history presented to his primary physician (PCP) with 3 days of fever, cough with yellow sputum, nausea, vomiting, and watery diarrhea. Of note, he had a tick bite exposure 2 weeks prior to this. His PCP suspected viral gastroenteritis and prescribed ondansetron. He presented again to urgent care 3 days later with persistent symptoms. Vital signs were significant for BP 99/66 mmHg, HR 94, and SpO<sub>2</sub> of 94% on room air. On exam, he appeared distressed and diaphoretic with the remainder of exam normal, including no rash. Significant labs revealed leukopenia, thrombocytopenia, hyponatremia, mild transaminitis, and hematuria. Chest x-ray suggested possible pneumonia. He was started on therapy for pneumonia without improvement. On further testing, the patient had an elevated LDH, anemia, low reticulocyte count, and elevated haptoglobin. Given high clinical suspicion, he was started empirically on doxycycline for potential zoonotic illnesses. Hematology was consulted, and blood smear analysis revealed neutrophil intracellular bodies. Human granulocytic ehrlichiosis IgM resulted in high titer. The diagnosis of anaplasmosis was made, and the patient clinically improved in regards to pancytopenia, transaminitis, and renal function. He was then discharged with oral doxycycline for a total of 10 days of treatment. Therapy was extended to 21 days when Lyme IgM/IgG was equivocal but Western blot was positive.

**Discussion:** Anaplasmosis has been increasing in incidence in the last decade, but fortunately fatality remains in less than 1% of cases. Incidence begins in spring and peaks during the summer months. This patient had an early presentation as he contracted the disease in April. Typically males older than 40 with an increased time of exposure to tick habitats are at risk for infection. Patients are diagnosed by visualization of morulae in neutrophils on blood smear or buffy coat, PCR, or serology. Serology is recommended for use in retrospective diagnosis of previously infected patients, but acute serology tends to have low specificity. Treatment involves IV or oral doxycycline 100mg twice daily for 10 days. Providers should be aware that anaplasmosis shares the same tick (*Ixodes scapularis*) as babesiosis and Lyme disease. Coinfection, as seen in this patient, has been documented and should be assessed for as treatment duration varies for each.

### 13) A PERNICIOUS ERROR: MISDIAGNOSING B12 DEFICIENCY ANEMIA

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**Introduction:** Anemia is a common problem in hospitalized patients (prevalence being 24-50%). Correlating the patient history and exam to the complete blood count can be invaluable in reaching the correct etiology. Here, we discuss a case of pernicious anemia presenting as pancytopenia which was misdiagnosed as iron deficiency anemia.

**Case Description:** A 53 year old white woman presented to our hospital on 5/2/16 with a two week history of worsening fatigue, dyspnea on exertion, and paresthesias. She also noted a 25 lb unintentional weight loss over the past three months. Past medical history was significant for longstanding iron deficiency anemia attributed to menorrhagia. She had received parenteral iron infusions for the past few years. She had a family history of anemia, a history of alcohol use, and reported regular meat and vegetable consumption. Laboratory data revealed a recent onset of pancytopenia (see table). Given her profound chronic macrocytosis, further investigations were pursued, showing a low serum B12 level of 90 ng/L, high methylmalonic level of 1727 nmol/L, and a positive intrinsic factor blocking antibody assay. Given symptomatic anemia, she was transfused packed red blood cells and given parenteral vitamin B12 supplementation with an improvement of blood counts. A retrospective review of records revealed persistent anemia with initial microcytosis with a gradually increasing mean corpuscular volume.

Date	Hgb (g/dL)	MCV (fL)	WBC (K/uL)	PLT (K/uL)
4/20/2012	8.6	70	6.1	296
9/17/2014	13.4	96.7	6.7	222
2/10/2016	11.7	119.3	7.0	254
5/02/2016	6.5	108.7	3.0	41
5/10/2016	9.1	105.3	4.4	103

**Discussion:** Though the overall incidence is only 0.1%, pernicious anemia accounts for 20-50% of B12 deficiency anemia. In general, B12 deficiency anemia should be considered a “not-to-miss” diagnosis: it is easily tested for and treated, but if missed, can lead to serious consequences like pancytopenia and neurological sequelae. Reexamining the MCV in treatment-refractory anemia can be an invaluable, cost-effective test which can help inform a focused work up.

### 14) PANIC: PROFOUND QT PROLONGATION AND HYPOPHOSPHATEMIA

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**Case Description:** A 28 year old female with a history of anxiety presented to the emergency department at our institution after she developed acute onset lightheadedness, dizziness, chest pressure, and tachypnea. In the emergency department she reported paresthesias, bilateral leg tremor, and persistent chest pressure. Her exam was remarkable for an anxious-appearing female who was tachypneic and tachycardic. Lungs were clear to auscultation bilaterally and her heart was regular without murmur. The remainder of her exam was normal. On laboratory evaluation, she was found to have a significant hypophosphatemia to 0.7 mg/dL, hypokalemia to 2.7 mmol/L, an elevated lactate of 5.9 mmol/L, and calcium of 9.5 mg/dL. An electrocardiogram was obtained and was remarkable for a QTc of 695 ms. She received a dose of Lorazepam, 1 gm calcium gluconate, 15 mmol of phosphate, and 30 mEq of potassium replacement. After resolution of her symptoms and incomplete supplementation of potassium and phosphorus, her repeat lab testing demonstrated potassium of 4.4 mmol/L, phosphate of 4.8 mg/dL, and lactate of 1.1 mmol/L. Repeat ECG demonstrated a QTc of 473 ms. She was admitted for observation overnight. After normalization of labs and symptomatic improvement, she was discharged home.

**Case Discussion:** Panic attacks lead to acute respiratory alkalosis and can cause profound ECG and laboratory findings. While in this case we do not know the degree of respiratory alkalosis due to the lack of a blood gas, we can assume that it was significant given her laboratory abnormalities. Perhaps the most worrisome finding in this case is the profound QT interval prolongation. QT prolongation in acute respiratory alkalosis is due in part to increased calcium binding to albumin leading to decreased ionized calcium, and intracellular shifts leading to hypokalemia. QT prolongation to this degree places the patient at risk for torsade des pointes, thus prompt treatment is warranted. In this case, calcium gluconate helped replete her hypocalcemia and potassium supplementation helped her hypokalemia. However, the most important factor was treatment of her panic attack, which allowed her respiratory alkalosis to resolve. While not contributing to QT prolongation, phosphate can be profoundly low with acute respiratory alkalosis. Lactate elevation also resolves with correction of respiratory alkalosis. Long-term treatment focuses on psychotherapy.

## 15) BLACK ESOPHAGUS IN ACUTE UPPER GASTROINTESTINAL BLEEDING

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Acute esophageal necrosis (AEN), otherwise known as “black esophagus”, is a rarely reported event, with less than 90 reported cases in totum. It usually presents as an acute upper GI bleed, is often complicated by esophageal perforation or hemorrhagic shock, and has mortality upwards of 35%.

A 59 year old male presented with acute alcohol intoxication, substernal chest and epigastric abdominal pain, and mental status changes. A CT abdomen and pelvis for aortic dissection revealed circumferential thickening of the distal esophagus with surrounding soft tissue stranding. Shortly thereafter the patient developed hypotension, tachycardia, profuse hematemesis and melanic diarrhea, associated with a drop in his hemoglobin from 15 to 10 mg/dL. Emergent esophagogastroduodenoscopy revealed white exudates and black eschars lining the entire esophagus to the GE junction, with an adherent clot in the distal esophagus. These findings are consistent with acute esophageal necrosis. The patient was hemodynamically supported with multiple blood transfusions. He was started on high dose PPI and Carafate, as well as broad spectrum antibiotics and antifungals for protection against infectious peritonitis in the event of esophageal perforation. Over the ensuing days of his hospitalization he had no further hemodynamic instability or evidence of active bleeding or esophageal perforation. He was discharged home in stable condition on hospital day seven.

One proposed hypothesis for the etiology of AEN involves a two hit mechanism. In brief, ischemic conditions are created in the esophagus, which is preceded by gastric acid reflux causing a burn injury to the vulnerable ischemic esophageal mucosa. In this patient we posit that chronic alcohol abuse and gastroesophageal reflux led to erosive esophagitis. He then developed a GI bleed from an esophageal artery while acutely intoxicated, which created conditions for esophageal ischemia. Subsequent reflux of gastric acid, then led to esophageal necrosis.

## 16) PERSISTENT NAUSEA WITH WEIGHT LOSS: A RARE CASE OF BIRT-HOGG-DUBÉ SYNDROME

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**Introduction:** Birt-Hogg-Dubé (BHD) syndrome is a rare autosomal dominant disorder characterized by fibrofolliculomas of the skin, renal tumors and multiple lung cysts with or without spontaneous pneumothorax. In this case a thorough physical exam, characteristic lung cysts and family history were critical in the diagnosis of a rare disorder that was overlooked leading to a diagnosis later in life.

**Case:** A 76 year-old male presented with persistent nausea, loss of appetite and 20 lb weight loss of 4 months duration. Past medical history included COPD, remote pneumothorax, Barrett’s esophagus, Parkinson’s disease and liver cysts. Due to persistent symptoms the patient was admitted to the hospital. On physical examination, flesh colored skin papules were noted over the face.

CT scan of the abdomen revealed multiple liver and lung cysts. The largest liver cyst (17 cm) was compressing the stomach causing his symptoms. Drainage of the liver cyst revealed 1900 mL of brownish fluid improving the patient’s appetite and nausea. On the basis of skin lesions and pulmonary cysts as well as strong family history of spontaneous pneumothorax, a diagnosis of BHD syndrome was suspected. Skin biopsy was performed resulting in findings consistent with a fibrofolliculoma, a characteristic skin lesion of BHD syndrome.

**Discussion:** The proposed diagnostic criteria for BHD syndrome require the presence of one major or two minor criteria. Major criteria include skin lesions (fibrofolliculoma) and a *Folliculin* gene mutation. Minor criteria include multiple lung cysts, early onset renal cancer, and a first-degree relative with BHD syndrome.

Screening chest radiography and ultrasonography or MRI for renal cancer for the patient and family members is advised. This patient met criteria by having characteristic skin lesions and bibasilar lung cysts. The patient’s brother was recently diagnosed with BHD syndrome. Evidence suggests that BHD syndrome exhibits diverse clinical heterogeneity and is not always associated with the three characteristic phenotypes.

## 17) LYME CARDITIS: A REVERSIBLE CASE OF HEART BLOCK

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**Introduction:** Lyme carditis should be suspected in any patient with new onset cardiac manifestations; especially those from endemic areas.

**Case:** A 67 year-old male with a past medical history of diabetes mellitus and hyperlipidemia presented with new onset exertional dyspnea and chest pressure. Two weeks prior to admission he noticed a skin lesion on the left shoulder described as a round shape and red rash that developed soon after he returned from clearing heavy vegetation in the rural town of Wautoma, WI. His PMD prescribed topical steroids, Lyme serology was sent, and he was prescribed doxycycline 4 days prior to admission after Lyme serology came back positive. At that time he developed exertional dyspnea and chest pressure and presented to our Emergency Room for evaluation.

On exam the patient was bradycardic with 1st degree heart block. Subsequent EKG revealed 2nd degree Mobitz Type II with 2:1 block and a PR interval of 424 ms. The patient was admitted to the CICU for close observation. Infectious disease was consulted and the patient was started on IV ceftriaxone.

Structural and ischemic heart disease was ruled out. A TTE revealed a normal ejection fraction, and a stress test was negative for reversible ischemia. The patient's symptoms improved dramatically with a 7-day course of IV antibiotics. The PR interval shortened to 278 and he was able to be discharged home on oral antibiotics.

**Discussion:** This case represents an uncommon manifestation of Lyme disease, Lyme carditis. Lyme carditis is seen in 4-10% of patients diagnosed with Lyme disease, and it is a potentially reversible cause of heart block. It also represents the importance carrying a high index of suspicion to diagnose Lyme carditis, and the importance of obtaining a thorough history.

## 18) THE WAY TO A MAN'S HEART—AND NERVOUS SYSTEM—IS THROUGH HIS STOMACH: A CASE OF MALNUTRITION MIMICKING NEUROLOGICAL DISORDER

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**Introduction:** The diagnosis of adult undernutrition is made if it fulfills two of the following criteria: insufficient energy intake, weight loss, loss of muscle mass, loss of subcutaneous fat, fluid accumulation, and diminished hand-grip strength. This case demonstrates that malnutrition can have accompanying symptoms such as numbness, tingling and weakness of the extremities and clinicians should keep malnutrition in mind during workup of neurological symptoms.

**Case:** A 62 Y male with recent bilateral DVT presented with numbness and weakness of the lower extremities for 2 months. He could not stand for long enough to prepare meals due to weakness. He denied fever and lost 10lbs in the last month. Physical exam showed a thin male with BMI 17, 4/5 strength and 2+ edema in bilateral lower extremities, and weak grip strength. Sensation to light touch was intact over bilateral upper and lower extremities, and reflexes were normal. CBC showed normocytic anemia with H/H 13.3/36 and BMP showed low electrolytes. He also had decreased creatinine, prealbumin, vitamin B6, and folate. Further workup included normal ESR and CRP and infectious, autoimmune, and metabolic labs. Imaging with MR of C and T spine was unrevealing. An EMG demonstrated a chronic polyradiculoneuropathy which was concerning for an inflammatory process. A nerve/muscle biopsy showed perivascular inflammation, active axonal degradation, and other changes that indicated probable vasculitis. During this workup, nutritional resuscitation was initiated with high-protein, high-calorie supplements. His weakness improved and he was discharged in stable condition to subacute rehab.

**Discussion:** Malnutrition presents primarily as weight loss, but patients can have neurological symptoms. In this case a full workup was still warranted, as it could have elucidated infection, autoimmunity, metabolic abnormality, or malignancy. This patient's largely unrevealing workup culminated in a biopsy that showed probable but not definitive vasculitis. This coupled with the fact that his weakness improved with nutrition indicates that malnutrition should remain at the top of the differential. Another possibility is malignancy, although imaging was negative. An outpatient bone marrow biopsy was also considered, but the patient was lost to follow up.

## 19) UNUSUAL PRESENTATION OF PRIMARY HYPOTHYROIDISM

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**Introduction:** Primary hypothyroidism is a relatively common metabolic disorder. The underlying pathophysiology is due to mitochondrial impairment and diminished catabolism causing a build up of mucopolysaccharides and hyaluronic acid, creating an osmotic gradient. This leads to a manifestation of signs and symptoms such as bradycardia, constipation, weight gain, hyperlipidemia and pericardial effusion.

**Case:** A 41-year-old male with a history of hyperlipidemia, asthma, anemia, and a recent history of viral pneumonia presented with a two-week history of chest pain. Patient was hypotensive and bradycardic on exam. On presentation laboratory findings were significant for a creatinine elevation of 1.61 (baseline of 1.24), an AST elevation of 54, and a creatine kinase (CK) elevation of 3445.12-lead ECG showed sinus bradycardia at 47 BPM and low voltage QRS complexes. LFTs were within normal limits. The patient was taking atorvastatin and was stopped due to significant rhabdomyolysis. Patient was started on IV fluids. Acute kidney injury (AKI) evaluation was initiated. Urine was negative for blood, protein and casts. Renal ultrasound was unremarkable and pharmacological stress echo testing did not show cardiac abnormalities. TSH ordered as part of workup for sinus bradycardia was found to be elevated at 63.74 with low free T3 and T4. Endocrinology was consulted for further recommendations. Thyroid ultrasound showed bilateral atrophy. Myeloperoxidase antibodies, thyroid peroxidase antibodies, and sedimentation rate were all negative, ruling out Hashimoto's thyroiditis. Levothyroxine was initiated after ruling out adrenal insufficiency and patient improved. Patient was diagnosed with rhabdomyolysis and AKI related to primary hypothyroidism. The patient was lost to follow up with endocrinology.

**Discussion:** Renal failure, due to rhabdomyolysis, is a rare complication of hypothyroidism. Here we report a patient whose rhabdomyolysis with AKI was due to underlying primary hypothyroidism. Impaired thyroid function causes a decrease in mitochondrial oxidative capacity leading to a decrease in muscle's ability to contract appropriately, including cardiac muscle. This leads to hypothyroid myopathy, rhabdomyolysis, and bradycardia. Poor renal perfusion and elevated CK severely affects renal function, but improves with hydration and correction of the underlying cause. It is important to suspect an underlying metabolic disorder such as hypothyroidism in the setting of elevated CK levels and AKI.

## 20) POEMS SYNDROME--A MEDICAL MYSTERY

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**Introduction:** POEMS syndrome is rare and usually a difficult diagnosis to make. The syndrome is defined by polyneuropathy, organomegaly, endocrinopathy, monoclonal plasma proliferation, and skin changes. The chronic progressive nature and similarities to other disorders make this syndrome challenging to recognize leading to potential misdiagnosis and delay of appropriate treatment.

**Case:** A 46 year-old-male presented with early symptoms of extremity paresthesia and intractable headaches. He soon developed two acute thrombotic strokes seen on brain imaging. The likely contributing factor was the discovery of the patient's significant polycythemia for which he received anticoagulation, hydroxyurea and therapeutic phlebotomies. A polycythemia vera and hypercoagulability work up were negative. Over the following month, the patient's extremity paresthesia worsened and he began to demonstrate significant LE weakness. Neurology was consulted and recommended an EMG which revealed severe peripheral neuropathy. There was high suspicion for CIDP vs POEMS. The patient was started on treatment for CIDP with IVIG and prednisone, however, his weakness continued to worsen resulting in immobility requiring hospitalization. Further testing revealed elevated VEGF. After thorough review of the patient's presentation, labs, and multidisciplinary discussions between hematology-oncology, neurology and internal medicine, the patient had met diagnostic criteria for POEMS syndrome. His case was submitted to Mayo clinic and the diagnosis was confirmed. The patient is now on appropriate treatment. Response to treatment has not yet been assessed.

**Discussion:** POEMS syndrome is difficult to diagnose because many presenting symptoms are shared with other diagnoses. Untreated, this syndrome can be life-threatening. Increasing awareness and encouraging multidisciplinary discussions can help decrease the time of symptom onset to that of a definitive diagnosis

## 21) OCP USE IN A PATIENT STATUS POST CHOLECYSTECTOMY

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Oral contraceptive pills have been shown to increase the risk of gallstone formation and clinical gallstone disease. Data on oral contraceptive pill (OCP) use pertaining to gallstone pathology post cholecystectomy is limited. No established guidelines or prospective studies on contraceptive use after cholecystectomy and the incidence of related gallstone pathology are described in the literature. Here, we describe a 28 year-old female status post cholecystectomy for gallstone-related cholecystitis who presented 3 months postoperatively with epigastric abdominal pain, elevated LFTs, and hyperbilirubinemia. The patient's history was significant for continued OCP (levonorgestrel/ ethinyl estradiol 0.15-0.03) use post cholecystectomy up until presentation. Of note, no intraoperative cholangiogram was performed and her 3-month postoperative course was uncomplicated. She described 5 days of intermittent, sharp, epigastric and RUQ pain that radiated to the back, with associated nausea and vomiting. Pain was similar in nature to that experienced before her gallbladder removal. LFTs were noted to be 10 times the upper limit of normal with peak total bilirubin of 4.3. RUQ ultrasound was significant for intrahepatic ductal dilation and common bile duct measuring 6 mm with no identifiable stone. Abdominal CT was significant for extrahepatic ductal dilation to 5 mm, again, without apparent stone. She was treated with empiric piperacillin/tazobactam for cholangitic coverage (despite being afebrile) and was admitted for further work-up. An ERCP was performed on day two; and results showed no filling defects on retrograde contrast instillation. However, patient's bilirubin down trended prior to procedure and it was strongly suspected by both the Medical and Advanced Endoscopy teams that a stone had likely passed prior to intervention. In this patient without significant past medical history, OCP use was the only identifiable risk factor for stone formation. Risks, benefits, and alternatives of OCP use were discussed with patient prior to discharge with plans for ongoing evaluation with primary care physician on safety of continuance. This case illustrates the importance of mitigating risk factors for stone formation in post cholecystectomy patients. In women of child-bearing age, a post-operative contraceptive plan should be discussed. Here we attempt to review literature and synthesize available information on this topic. While gallstone-related cholecystectomy is not a contraindication for OCP use at this time, further investigation with prospective and/or retrospective studies may be of benefit in the ongoing treatment of these patients.

## 22) A CURIOUS CASE OF CANCER

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**Introduction:** Management of a patient with coexisting conditions and an unclear clinical course proves a difficult task; a task that we often fail to achieve. Diseases don't read text books, interventions are not without adverse effects, and medicine is too complicated for a single specialty.

**Case Presentation:** We present the case of a 69-year-old smoker with a 2-week history of progressive shortness of breath, lethargy, and confusion after a ground level fall. She was previously seen 3 times in outpatient clinic being diagnosed and treated for pneumonia after a chest x-ray showed minimal right lower lobe infiltrate and an elevated right hemidiaphragm. On admission the patient was unwilling to lie flat, had been sleeping in a recliner for weeks, and had become progressively confused for weeks. She was afebrile, hemodynamically stable on room air, had hemoglobin of 10.1 (chronic), a WBC of 23.9, and sodium of 114. Standard of care for these ailments only exacerbated her already frail condition. In a matter of hours she became progressively weak, confused, and clinically worse. Re-examination of chest x-ray showed continued elevated right hemidiaphragm and the patient continued to insist on sleeping upright. Worsening confusion and clinical decompensation necessitated intubation and further investigation. A CT chest was performed showing a large mass originating from the anterior chest wall which was displacing the mediastinum, heart, and liver to the left. The patient was taken to the operating room where a de-roofing procedure was performed and 5 liters of blood tinged fluid were removed from the encompassing sac. The patient's hemoglobin dropped 5 units postoperatively and she was transfused. The mass was identified as adenocarcinoma. The patient awoke from the procedure, adjusted her code status to palliative, and passed less than 3 hours later.

**Discussion:** A multidisciplinary approach to medicine is crucial to the success of patient care; each day we attempt to alleviate pain and lessen disease burden by the identification and treatment of disorders. Lifesaving, life prolonging, and pain relieving interventions takes a multidisciplinary team. We as physicians need to remain the spearhead of this effort.

## 23) POTT'S PUFFY TUMOR, A RARE COMPLICATION OF SINUSITIS

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**Introduction:** Sinusitis is a common disorder that rarely results in serious complications including meningitis, epidural abscess and cerebral abscess. Pott's puffy tumor is defined by subperiosteal abscess associated with osteomyelitis of the frontal bone. It is a rare condition, mostly seen in adolescents and is related to sinusitis or facial trauma. Complications of Pott's puffy tumor include extension of infection into the orbits and CNS. Treatment includes surgical drainage and broad-spectrum antibiotics.

**Case:** A 62 year-old male with history of chronic back pain presented to the emergency room with 6 months of worsening facial pain in a frontal distribution. A CT without contrast was performed showing a 2.8 x 0.9 cm fluid collection in the frontal sinus with subperiosteal abscess and osteomyelitis of the frontal bone. Findings were consistent with a diagnosis of Pott's puffy tumor. The patient was admitted and began IV ceftriaxone and vancomycin, which were subsequently changed to ertapenem and vancomycin. The patient was taken to the operating room for abscess drainage by endoscopic sinus surgery where copious pus was found throughout the right maxillary and right frontal sinuses. Surgical cultures grew *Prevotella* species, *Dialister* species and *Fusobacterium naviforme*. These results were consistent with oral flora and it was thought that poor dentition was the source of his sinusitis and Pott's puffy tumor. Throughout his course, the patient remained afebrile without leukocytosis and there were no radiological or physical exam findings of CNS involvement. Because of his poor dentition and source of infection, the patient had multiple tooth extractions prior to discharge. He then completed a 6 week course of ertapenem and daptomycin.

**Discussion:** Sinusitis is a common condition with rare and serious complications if untreated. One of these complications includes Pott's puffy tumor, which can extend into the CNS and manifest as epidural, subdural or brain abscess as well as venous thrombosis. In one review, 59% of cases resulted in CNS extension. Fortunately, our patient did not suffer from this complication. Mainstays of treatment include surgical drainage and broad spectrum antibiotics for 6-8 weeks. Early diagnosis and treatment are key to prevention of CNS complications of sinusitis.

## 24) SPHENOID SINUS NON-HODGKIN LYMPHOMA PRESENTING AS 6TH CRANIAL NERVE PALSY

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**Background:** Primary non-Hodgkin lymphoma (NHL) of sphenoid sinus is extremely rare. Initial symptoms of this tumor may mimic nasal obstruction and sinusitis. Here we report a case of B-cell NHL in a 74 year old female.

**Case:** A 74 year old female with no past oncologic history presents to the hospital with 5-month history of headaches and recent-onset diplopia. Computed tomography scan and magnetic resonance imaging showed an abnormal lesion involving the clivus and posterior aspect of the left sphenoid sinus. Sphenoid sinus biopsy obtained by endoscopic left sphenoid sinusotomy demonstrated CD20 positive and CD3 negative diffuse large B-cell lymphoma. Upon referral to oncology R-CHOP chemotherapy regimen and intrathecal Methotrexate were initiated after 18F-FDG PET/CT findings showed no evidence of metastasis. After completion of R-CHOP and intrathecal Methotrexate, regional radiation therapy was given at a total dose of 4800 cGy in 24 fractions. Subsequent MRIs showed significant decrease in soft tissue thickening related to lymphomatous involvement of the posterior sphenoid sinus and superior clivus. Patient's diplopia and headaches resolved completely. During her latest follow-up she was reportedly doing exceedingly well.

**Conclusion:** Sphenoid sinus NHL may have to be considered among the differentials in patients presenting with symptoms of persistent headaches with new-onset cranial nerve dysfunction (II, III, IV, V, and VI).

## 25) A DIAGNOSTIC CHALLENGE: SARCOIDOSIS AS THE CAUSE OF HEPATIC GRANULOMAS

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**Introduction:** Hepatic granulomas are present in a wide array of conditions and have been reported in 2–15% of liver biopsies. While the lesions themselves are unlikely to be of significant consequence, their associated systemic disease may have implications on treatment and prognosis. The most common causes of hepatic granulomas in the United States include: tuberculosis, sarcoidosis, primary biliary cirrhosis and drug reactions.

**Case Report:** A 65 year-old former prison guard with history of hypertension, type II DM and ulcerative proctitis on sulfasalazine presented with fatigue, unintentional weight loss, hypercalcemia and transaminitis. Laboratory evaluation revealed low parathyroid hormone (PTH), low parathyroid hormone-related protein (PTHrp), and elevated 1,25-dihydroxyvitamin D. CT imaging revealed hepatomegaly with diffuse heterogeneity, splenomegaly with innumerable splenic lesions, mesenteric lymphadenopathy and multiple small (<3mm) apical pulmonary nodules. There was no evidence of malignancy or mediastinal/hilar lymphadenopathy. Liver biopsy revealed non-necrotizing granulomatous inflammation without evidence of primary biliary cirrhosis. Diagnostic testing including AFB smears, quantiferon gold, galactomannan, blastomyces antigen, histoplasma antigen, fungal cultures, CMV, EBV, immunofixation, Q fever Ab, Brucella, HIV and AMA among other testing returned negative. Sulfasalazine was held given a well-documented association with granulomatous hepatitis. Bronchoscopy with blind transbronchial biopsy was performed which revealed granulomas consistent with sarcoidosis. Given the absence of an infection, the patient's presentation was felt to be consistent with sarcoidosis with involvement of the lungs, liver, spleen.

**Discussion:** Sarcoidosis is a common cause of hepatic granulomas. The extent to which hepatic granulomas cause clinical consequences is variable. Majority of patients have elevated transaminases but are otherwise asymptomatic. However, cases have been reported of patient's developing cirrhosis, portal hypertension, and hepatic vein thrombosis. While histopathology and localization of the lesions can be helpful in determining the etiology of the granulomas, they are insufficient to make the diagnosis of hepatic sarcoidosis. A diagnosis of hepatic sarcoidosis requires identification of extrahepatic manifestations and ruling out alternative etiologies.

## 26) HEROIN-INDUCED DIFFUSE ALVEOLAR HEMORRHAGE

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**Introduction:** Drug-induced diffuse alveolar hemorrhage (DAH) is a common finding in patients with cocaine abuse, but is rare in those using heroin. Common pulmonary complications of heroin use include foreign body aspiration, aspiration pneumonia and naloxone acute withdrawal (massive sympathetic response). Seen more infrequently is heroin-induced non-cardiogenic pulmonary edema (NCPE), the mechanism hypothesized to be due to either ischemic injury, direct toxicity or hypersensitivity reaction. NCPE patients may have some small alveolar hemorrhages, but a patient with DAH causing hemoptysis significant enough for multiple blood transfusions has yet to be seen in the literature.

**Case:** A 43 year-old African American female with past medical history of anxiety, obesity and substance abuse presented to the Emergency Department with shortness of breath and hemoptysis. Upon presentation, patient was in respiratory distress, tachycardic and hypoxic requiring oxygen NC. On physical exam patient was anxious, tachypneic using accessory muscles, tachycardic with HR 120 and with diffuse crackles in lungs. Laboratory findings were significant for positive urine methadone and opiates (no cocaine, THC, or amphetamines), normal renal function and Hb 6.1 g/dL. CXR and CT chest showed diffuse ground-glass opacities more pronounced in the bilateral upper lobes with relative sparing of the lung bases consistent with DAH, heroin-induced lung injury, acute interstitial pneumonia, or eosinophilic pneumonia. Patient was intubated for acute respiratory failure and admitted to the ICU. She received Ativan for heroin withdrawal and was transfused 2 units PRBCs. Patient was eventually extubated, still requiring oxygen. Bronchioalveolar lavage showed a significant amount of RBCs and hemosiderin-laden macrophages suggestive of DAH. Bronchoscopy report showed right upper lobe edema and minimal left upper lobe dark blood with no active bleed. Biopsy showed negative cytology/culture with 90% macrophages, 5% neutrophils and 5% lymphocytes. Upon further questioning, patient admitted to snorting heroin two to three times per day for the past 14 years and had been hospitalized four times at multiple institutions in last 6 months for shortness of breath and hemoptysis; these episodes were precipitated by heroin use. Further workup was negative for blood, respiratory and urine cultures, fungal/atypical infections, ECG changes, ANCA, HIV NAAT, ANA, RF, anti-GBM, hepatitis C Ab, lupus anti-coagulant, anti-phospholipid Ab, complement changes, and elevated CRP/ESR. Based upon the history of heroin use, clinical presentation and workup the diagnosis of heroin induced DAH was made. Patient improved over 48hrs.

**Discussion:** Here we report a case of acute DAH with hemoptysis in a patient with significant heroin use. NCPE would be the best-fit diagnosis with hemosiderin-laden macrophages, but the severity of blood loss makes DAH more likely.

## 27) GASTROINTESTINAL AMYLOIDOSIS WITH ACCESSORY ORGAN INVOLVEMENT

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**Introduction:** Amyloidosis is a multisystem disease that typically presents with cardiac, musculoskeletal, renal, gastrointestinal and dermatologic findings. Symptoms arise from the deposition of plasma proteins, called “amyloid protein,” in the extracellular tissue. Only 3.2% of patients with amyloidosis have biopsy-proven gastrointestinal (GI) involvement, and of those, 21% lack evidence of systemic disease. Given the nonspecific symptoms at presentation, physicians often face a diagnostic challenge.

**Case:** A 62 year old female without significant past medical history presented with a 7-month history of weight loss, weakness, and early satiety. Her symptoms began soon after her husband died. Initially, she attributed her symptoms to fatigue and bereavement but when her symptoms persisted she sought medical attention. Her physical examination was unremarkable except for bilateral leg edema and laboratory studies showed an elevated alkaline phosphatase, and proteinuria. An abdominal MRI revealed a hypoechoic abnormality in the pancreatic head measuring 3.0 x1.6cm. She underwent an endoscopic ultrasound that grossly revealed a nodular stomach mucosa with friability and evidence of an infiltrative process. Biopsies from the pancreatic body, gastric mucosa, and the duodenal mucosa showed nodular homogenous eosinophilic deposits consistent with amyloid. Congo red stain on the biopsies showed an apple green birefringence on polarized light consistent with amyloidosis. Urine protein electrophoresis and immunofixation showed IgA kappa and free kappa light chains. The patient was started on loop diuretics to relieve her leg swelling, and is likely to begin steroids for supportive management.

**Discussion:** Localized GI amyloidosis is unusual and the involvement of accessory organs (such as the pancreas) to the GI tract is rare. A clinical suspicion for GI amyloidosis should be paid to patients presenting with chronic GI bleeding, unexplained malabsorption, weight loss, constipation, vomiting, malaise, weakness, and early satiety in the presence of an elevated alkaline phosphatase and proteinuria. Tissue biopsy with positive Congo red stain and amyloid proteins on electron microscopy remain definitive diagnostic tools. Current therapy is directed at symptomatic relief with diuretics, antiemetics, antidiarrheals, and corticosteroids.

## 28) MAY-THURNER SYNDROME-AN UNDERDIAGNOSED CONDITION

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**Introduction:** May-Thurner syndrome (MTS) is a condition of venous flow obstruction from extrinsic compression due to intrinsic anatomy, most commonly compression of the left common iliac vein by overlying right common iliac artery. This leads to complete or partial obstruction of flow with the possibility of developing deep vein thrombosis (DVT). The prevalence of MTS is estimated at 2-5% of patients with a lower extremity venous disorder. Risk factors of MTS include multiple pregnancies, postpartum period, oral contraceptive pills, immobilization, and dehydration. MTS is also thought to be an underdiagnosed condition.

**Case:** A 54-year-old female with past medical history of migraines and lumbar stenosis presented with a five-day history of left leg pain and swelling. She recently returned from a 7-hour car drive, had a 17.5 pack-year smoking history, and was on conjugated estrogens for hot flashes. Vitals were significant for tachycardia and dyspnea. Physical exam demonstrated a tender, edematous, cool, dry left lower extremity with purplish discoloration and delayed capillary refill, consistent with phlegmasia cerulea dolens. Labs were significant for leukocytosis with neutrophilia, low bicarbonate, and high anion gap. Ultrasound (U/S) showed an acute occlusive thrombus in the left common femoral vein with extension into the upper/deep femoral, and great saphenous veins. Pulses were palpable. CT abdomen/pelvis showed obstruction of the left common iliac vein at the inferior vena caval confluence suggestive of MTS. The patient was started on Lovenox. Thrombolysis with TPA and heparin was performed to prevent post-thrombotic syndrome. A repeat venogram showed minimal improvement so venous angiojet was used to clear the clot. Stenosis of the left common iliac vein was angioplastied and stented. The patient experienced significant improvement following the procedures and long-term anticoagulation with Coumadin was scheduled.

**Discussion:** Here we present MTS in a patient at risk for DVT. It is crucial to accurately diagnose MTS as standard anticoagulation therapy used for patients with DVT is not sufficient to prevent recurrence of MTS. U/S is the initial and most reliable test for clinical suspicion of MTS. CT with venous phase and MRI venography may also be used as a diagnostic test due to the low sensitivity of U/S above the inguinal plane. MRA is the gold standard since CT may have limited resolution in pelvis due to bony artifacts. Treatment for MTS is dependent on the presence of DVT. If no DVT is present, endovascular management consists of stenting the lesion with a self expanding stent followed by balloon dilation. If a DVT is present, treatment includes catheter-directed thrombolysis followed by stent placement.

## 29) EPIDURAL ABSCESS CAUSED BY MRSA: A CHALLENGING CASE

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**Introduction:** Thoracic epidural abscess (TEA) is a rare infections defined by the accumulation of purulent material in the space between the dura matter and the osseo-ligamentous structure making up the vertebral canal. TEA is caused by direct inoculation, hematogenous spread, from remote infection or iatrogenic source. The common risks include spinal surgery, immune suppression, and intravenous drug abuse. The most common organism is *Staphylococcus aureus* (63%). Important factors essential to the management of TEA include early recognition and aggressive treatment with IV antibiotics or surgical debridement.

**Case:** This patient is a 71 year old male with past medical history of rheumatoid arthritis treated with cimzia+methotrexate and history of laminectomies who presented to the hospital with severe bilateral flank pain, fever and lower extremity weakness associated with pain. Previously back pain and fever with positive UA was treated with course of Bactrim without resolution of back pain. Review of symptoms was negative. Patient was afebrile on admission with vitals significant for a BP of 148/72 and new onset atrial fibrillation (AFib) with RVR. Physical exam was normal except for back pain with movement. Lab values were significant for white blood count of 11.9. Blood cultures were drawn and CT spine showed soft tissue thickening w/ erosive endplate changes. MRI spine revealed small enhancing collection within the dorsal spinal canal concerning for abscess. Patient was started on IV vancomycin and neurosurgery did not deem patient eligible for surgical candidate due to risks. Blood cultures confirmed methicillin resistant *staphylococcus aureus* positive bacteremia. Patient was discharged after 11 days in hospital to sub-acute rehab on 6 week course of IV vancomycin. Patient got readmitted for 19 days with worsening back pain, fever and persistent MRSA bacteremia and was IV daptomycin and linezolid because of elevated creatinine due to vancomycin. Patient was readmitted one week after completing course of antibiotics with continued severe back pain, fever, bacteremia, supratherapeutic INR and worsening afib with RVR. Repeat MRI spine indicated progression of infection and subsequent aspiration revealed MRSA-positive purulent drainage. Patient's symptoms and function improved with continued administration of antibiotics and at time of discharge was able to ambulate well and on day 11 was discharged to home care.

**Discussion:** Here we present a challenging case of MRSA epidural abscess in an immune compromised patient. The patient lacked direct inoculation and initially presented with confounding UT in the setting of flank pain and fever. It is crucial to recognize and treat TEA early to prevent morbidity and mortality. The important take home point of this case is keep a high level of suspicion of TEA in the setting of the immune compromised patient with fever and severe back pain.

## 30) FIBROSING MEDIASTITIS AND PANHYPOPITUITARISM IN AN ATYPICAL PRESENTATION OF DISSEMINATED HISTOPLASMOSIS

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**Introduction:** *Histoplasma capsulatum* is an endemic fungus that most commonly causes pulmonary disease. However, it may present as disseminated infection with atypical presentations, making diagnosis difficult. Fibrosing mediastinitis and panhypopituitarism are rare initial presentations of Histoplasmosis.

**Case:** A 39 year old Hispanic woman with no known past medical history presented with 2 months nausea, vomiting, and 12-lb weight loss. Social history is significant for emigration from Mexico 22 years ago. She has a pet dog and parrot. Initial vitals were T 97.2F, P 54, BP 85/56, O2 97% on room air. Exam was overall unremarkable including normal neurological exam and visual fields. Lab work revealed sodium 118 mmol/L, glucose 60 mg/dL, serum osmolality 259 mOsm/kg. In the first 24 hours, the patient's glucose remained <60 despite 3 vials of D50. Blood pressure remained low despite fluid resuscitation and she was transferred to the ICU. Follow-up labs showed morning cortisol 1.0 ug/dL, undetectable ACTH, low free T3 and T4, and normal prolactin. MRI brain showed a heterogeneously enhancing sellar lesion with calcifications. CT chest significant for extensive soft tissue within the mediastinum and large areas of calcification consistent with fibrosing mediastinitis. Serum fungal antibody and urine fungal antigen tests returned negative. Final fungal cultures were negative as well. Mediastinal lymph node biopsy showed focally necrotizing granulomatous inflammation concerning for Histoplasmosis. Biopsy stains were negative for malignancy and fungal elements. She started Levothyroxine and Hydrocortisone for panhypopituitarism, and empiric Itraconazole for Histoplasmosis with plan for close follow-up.

**Discussion:** The case demonstrates how a ubiquitous fungal organism, *Histoplasma*, can present in atypical ways regardless of immune status. It also presents how concrete evidence of Histoplasmosis can be difficult to obtain; therefore, suspicion must remain high. Her diagnosis of Histoplasmosis was a culmination of characteristic findings on imaging, pathology, and history. While her mediastinal findings were characteristic of previous *Histoplasma* infection, her pituitary findings could represent active or inactive infection. Patients with *Histoplasmosis* and fibrosing mediastinitis also run the risk of complications such as superior vena cava syndrome that are the result of excess host responses leading to worsening fibrosis. She is on a modest dose of Hydrocortisone for secondary adrenal insufficiency to avoid reactivation of the *Histoplasma*. On follow-up visit, she has had resolution of symptoms, stable lab work, and decreased size of pituitary lesion.

### 31) RECURRENT DVT IN A PATIENT WITH ANTITHROMBIN III DEFICIENCY AND MAY-THURNER SYNDROME: A CASE REPORT

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**Introduction:** Inherited Antithrombin III (ATIII) deficiency is a rare Autosomal dominant disorder manifesting as lack of thrombin and Factor X inactivation, with clinical implication of increased thromboembolism (VTE) risk. Clinical management consists of unfractionated heparin (UFH) and low molecular weight heparin (LMWH) while transitioning to Warfarin, with described theoretical risk of heparin resistance.

**Case:** 21 year old female with history of ATIII deficiency and recurrent VTE presenting with left lower extremity swelling and pain. Most recent VTE was two weeks prior to admission, secondary to poor medication compliance. On re-presentation, INR was 3.9 with superior propagation of thrombus noted via ultrasound. CT venogram demonstrated compression of left common iliac vein (CIV) by right common iliac artery (CIA) concerning for May Thurner's syndrome. Patient admitted on UFH drip. ATIII activity levels were 49%; monitored UFH levels remained <0.10. Given burden of thrombus, dose of recombinant ATIII was administered, after which activity levels increased to 102%. UFH levels improved to 0.22. Given the severity of thrombus, patient underwent catheter-directed thrombolysis, with left CIA stent placement. She was discharged on enoxaparin titrated to LMWH level of 0.88, with bridging to Warfarin.

**Discussion:** This patient presents at a young age with recurrent DVT, with two separate risk factors for hypercoagulability. May Thurner syndrome is a known anatomic variant with increased risk of treatment-resistant left iliac vein thrombosis. Treatment requires anticoagulation, thrombolysis and/or venous stent placement. Administration of ATIII for surgical/obstetric prophylaxis has been studied, with less research regarding heparin-refractory thrombosis. Given this patient's underlying hypercoagulability and heparin resistance, aggressive treatment with anatomical stenting and ATIII replacement was required to achieve therapeutic anticoagulation.

### 32) 54-YEAR OLD WOMAN WITH PCR NEGATIVE CLOSTRIDIUM DIFFICILE INFECTION (CDI)

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**Introduction:** We use this case to explore the validity of PCR testing for CDI, and how negative PCR tests be interpreted.

**Case:** The patient presented to emergency department with a complaint of abdominal pain, watery, non-bloody diarrhea associated with nausea and vomiting. Relevant labs included Cr of 1.7 mg/dL, WBC of 25.9 k/mcl; Albumin of 2.6 g/dL. Stool PRC test using BD MAX Cdiff assay (BD Diagnostics) for clostridium defficile was positive. She was treated with Vancomycine 250 mg per os (p.o) every six hours and discharged home stable, but returned to ER shortly after completing full course of oral Vancomycine complaining of diarrhea of up to 12 times. Given her known CDI history, she was empirically started on Vancomycine 500 mg p.o every six hours. Stool PRC test using BD MAX C.diff assay (BD Diagnostics) for clostridium defficile was negative. While CDI remained a strong possibility, in the light of negative test, post C.diff irritable bowel syndrome was considered. Vancomycine was discontinued and decision was made to do colonoscopy, which showed extensive pseudomembranous colitis. There are at least 3 commercially available real-time PCR assays that detect c.diff toxin (tcdB). A study by Dalpke et al puts the sensitivity and specificity of BD Max cdiff assay at 90.5%, 97.9% compared to culture as gold standard.

**Discussion:** Reviewing the literature we learned two scenarios under which PCR testing might be negative. First scenario relates to a case of atypical toxin production as evidenced by Eckert et al in their 2015 paper. Second scenario relates to issues with PCR result interpretation. According to Leis JA et al report, negative PCR might just be technical related to amplification curve developed to interpret the PCR result. This, according to them, has nothing to do with technique-related, equipment-related, or CDI strain.

### 33) A CASE OF PURPURA FULMINANS IN A BACKGROUND OF SEPTIC SHOCK CAUSED BY STREPTOCOCCUS PNEUMONIAE

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**Introduction:** Purpura fulminans (PF) is a life threatening and rare complication caused by microvascular occlusion and hemorrhagic infarction of the dermis resulting in large purpura and limb ischemia. It is associated with very high mortality rates ranging from 33 to 90% in reported literature.

**Case:** A 60-year-old female with a history of COPD presented with progressive shortness of breath and cough for 24-48 hours. In the ED, the patient was intubated and was started on empiric broad-spectrum IV antibiotics and fluids. She was admitted to the medical ICU for septic shock with multi-organ failure. Labs on admission later confirmed *Streptococcus pneumoniae* pneumonia and bacteremia, which were successfully treated empirically.

On day 5, the patient's extremities were noted to be cool, and well-demarcated mottling purpura quickly developed in all 4 distal limbs. Differential diagnoses including disseminated intravascular coagulopathy (DIC), thrombocytopenic thrombotic purpura (TTP), heparin-induced thrombocytopenia (HIT) were considered but were subsequently ruled out with normal fibrinogen level, mildly diminished ADAMTS13 levels, and negative PF4 respectively. Common rheumatologic causes were ruled out via negative autoimmune markers. A punch biopsy was obtained and subsequently ruled out Stevens-Johnson syndrome/toxic epidermal necrolysis and angioinvasive organisms. Clinical correlation with the punch biopsy led to a diagnosis of PF.

Meanwhile, the patient's limbs were monitored daily by the vascular and plastics surgery teams, and the lesions were protected via loose gauze, lamb's wool boots, and petroleum jelly.

The patient was hospitalized for 1.5 months and suffered many complications including respiratory failure, renal failure, cardiomyopathy, ischemic hepatitis, and significant hematomas, along the course before being discharged to a long-term acute care facility. She was readmitted 6 weeks later and received elective wound debridement and bilateral above knee amputations and right hand amputation.

**Discussion:** It is believed that bacterial endotoxin-induced endothelial damage and disturbance in pro- versus anticoagulation factors are key elements leading to acute sepsis-related PF. Perfusion is often compromised as a result of thromboembolic lesions, and the rates of amputation have been reported ranging from 90 to 100%. In addition to prompt implementation of antibiotics and aggressive supportive therapy, close monitoring of the limbs is crucial. The management of limb ischemia differs from case to case, ranging from emergent radical debridement to more conservative measures depending on stability of the patient and presence of wet gangrene.

### 34) DAPTOMYCIN INDUCED EOSINOPHILIC PNEUMONIA

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**Introduction:** Eosinophilic pneumonia is a rare lung pathology associated with a broad differential. Here we present a case of eosinophilic pneumonia induced by daptomycin administration in the setting of diastolic heart failure exacerbation.

**Case:** A 60 year-old woman presented with 2 days of severe shortness of breath. She had bilateral crackles and significant bilateral lower extremity edema. Laboratory results were remarkable for elevated creatinine kinase, leukocytosis and abnormal creatinine. Chest X-ray revealed new bilateral patchy opacities and bilateral pleural effusions. Two weeks prior to admission, she had been started on a 6 week course of I.V ertopenem and daptomycin for cervical discitis.

A CT chest revealed multifocal consolidations with surrounding ground glass opacities and adenopathy. Further labs revealed abnormal peripheral eosinophilia and elevated serum IgE. A bronchial-alveolar lavage revealed marked eosinophilia.

A diagnosis of daptomycin induced eosinophilic pneumonia was made once other causes were ruled out. Fortunately, daptomycin had already been switched to linezolid on admission. A chest x-ray was repeated 1 week after admission and showed complete clearing of the bilateral interstitial thickening.

**Discussion:** We found 8 cases of definite daptomycin induced eosinophilic pneumonia. Diagnostic criteria included were 1) bilateral pulmonary infiltrates, 2) hypoxia 3) bronchial-alveolar lavage with 25% eosinophils, 4) exposure to drug or toxin 5) no other cause of pulmonary eosinophilia, 6) clinical improvement after cessation of drug and finally, 7) recurrence of symptoms after re-introduction of toxin or drug. While bronchial-alveolar lavage revealed only 19% eosinophils, our case still met 5 of 7 requirements for diagnosis.

Daptomycin causes injury by forming a complex with pulmonary surfactant, activating alveolar macrophages and leading to IL-5 release by Th-2 cells, leading to eosinophilic release and inflammation. In our patient, this damage led to a CHF exacerbation. Once the offending agent was removed and fluid taken off, our patient rapidly improved.

Daptomycin is an increasingly common second line antibiotic prescribed due to the emerging incidence of multi-drug resistant organisms. It is important for hospitalists to recognize the clinical symptoms of possible drug induced lung injury so as to discontinue the medication promptly.

### 35) A RARE CAUSE OF FULMINANT LIVER FAILURE

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**Introduction:** Primary Hepatic Angiosarcoma (HAS) is a rare cause of hepatic malignancy with an annual incidence of 200 cases worldwide. HAS is a high grade vascular neoplasm and is often caused by exposure to toxins such as vinyl chloride, anabolic steroids, arsenic, and radiation. Most patients are men over the age of 60 who generally present with abdominal pain, ascites, and hepatomegaly. It is extremely rare for patients with HAS to present with fulminant hepatic failure. We found only 5 such cases documented in the literature.

**Case:** An 81 year old male presented to an outside hospital with one week of confusion and slurred speech. Initial evaluation included a CT of the head that was unremarkable, a complete blood count notable for leukocytosis and urinalysis concerning for infection. He was treated for sepsis secondary to a urinary tract infection but continued to worsen and was transferred to our tertiary care facility. Upon transfer, laboratory analysis showed a white blood cell count of 49,800 / uL with neutrophil predominance, platelet count of 82000/ uL , international normalized ratio of 17, aspartate aminotransferase of 204 unit/L, alanine aminotransferase of 91 unit/L, alkaline phosphatase of 321 unit/L, total bilirubin of 2.4mg/dL, and creatinine of 2.21mg/dL. Peripheral smear was negative for schistocytes and blast cells. Haptoglobin and fibrinogen levels were within normal limits. Infectious work up, including blood and urine cultures and viral serologies, was negative throughout his stay. His lactic acid was elevated on transfer at 7.2mmol/L and continued to rise daily. A PET scan revealed diffuse liver uptake concerning for non-specific hepatitis, although all viral and autoimmune panels were negative. The patient's mental status deteriorated and he developed circulatory shock with oliguric renal failure requiring transfer to the ICU for intubation, vasopressor support, and continuous veno-venous hemofiltration. He had multiple GI bleeds during this time requiring transfusion of blood products. A liver biopsy was performed that showed invasive angiosarcoma. At this point, given the poor prognosis of multiple organ failure combined with a treatment resistant malignancy, the patient's family elected to transition to a comfort plan of care. The patient expired within hours.

**Discussion:** While HAS is the most common sarcoma arising in the liver, it is an uncommon primary hepatic malignancy and rarely presents with fulminant hepatic failure. It often involves both lobes of the liver, and is diagnosed by biopsy. The tumor is very aggressive, and the majority of patients die within 6 months of diagnosis, regardless of treatment. There are no established treatment regimens: the tumors are radioresistant, and chemotherapy is only palliative. Liver resection is the most successful treatment modality, however recurrence is high and a minority of patients live long enough for a chance at resection. Median survival is 5 months.

### 36) CAPNOCYTOPHAGA CANIMORSUS, AN UNCOMMON, FREQUENTLY LETHAL ZONOTIC INFECTION RECOGNIZED BY CLINICAL FEATURES AND CONTEXT IN RAPIDLY PROGRESSIVE SEPTICEMIA WITH DIC AND MULTI-ORGAN SYSTEMS FAILURE

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**Introduction:** *Capnocytophaga canimorsus* is a fastidious, gram-negative rod, commonly found in oral flora of dogs, that infrequently causes rapidly progressive sepsis with high mortality in humans. Infection presents as high grade bacteremia with frequently associated symptoms of shock, DIC, multi-organ dysfunction, meningitis, purpura or petechiae, and necrosis of extremities in asplenic or alcoholic patients with dog exposure.

**Case Report:** Previously well 66-year-old female status post splenectomy at age 32 for Hodgkin's lymphoma presented to the ED with fever, altered mental status, and pain at the site of a dog bite sustained two days prior. She was febrile and tachycardic with slowed mentation and localized inflammatory response at the DIP of the fourth digit, but otherwise stable. Within hours she had worsening lactic acidosis refractory to broad spectrum antibiotics and fluid resuscitation requiring multiple vasopressors. Within 24 hours, she had hypoxic respiratory failure, shock liver, and acute renal failure. She developed localized purpura and demonstrated early gangrenous change of the entire hand. Platelet count dropped precipitously. Gram stain demonstrated PMNs with intracellular gram-negative rods and blood smear revealed schistocytes with Howell-Jolly Bodies consistent with diagnosis of DIC from *Capnocytophaga canimorsus* before MALDI-TOF confirmed the diagnosis. The patient made a full recovery with source control of her septic DIP, and supportive care. Three months after admission, she will require amputation of the distal ring finger, but avoided loss of life or entire limb due to early initiation of appropriate antibiotics and source control based on high clinical suspicion.

**Discussion:** *Capnocytophaga canimorsus* is an uncommon fastidious zoonotic pathogen capable of causing devastating infection in humans. Rapid progression and high mortality necessitates high suspicion for the organism and start of appropriate antibiotics based on clinical context well before culture data is available and even before advanced diagnostics can yield definitive diagnosis to minimize likelihood of mortality and severe morbidity.

### 37) BILIARY OBSTRUCTION MASQUERADING AS ACUTE HEPATITIS

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**Introduction:** Grossly elevated liver enzymes are known to occur in hepatocellular injury, including autoimmune, viral, and medication induced, but they can also be induced by biliary obstruction alone.

**Case:** A 44 year-old female with a history of cholecystectomy presented complaining of epigastric burning pain with radiation to the left side of the abdomen. Her symptoms followed ingestion of food that was diagnosed one day prior as gastritis. Evidently, she reported the pain was exactly like the pain she experienced from cholelithiasis before undergoing cholecystectomy. Along with worsening epigastric pain, the patient's ALT and AST increased from 298 and 301 to 1606 and 1192, respectively. Total bilirubin went from 1.2 to 3.7, and alkaline phosphatase increased from 168 to 256 while lipase remained normal. She denied recent travel, sick contacts, herbal medicine, excessive Tylenol intake and had one new sexual partner who was asymptomatic. She was up to date regarding her vaccinations.

Subsequent work-up included an abdominal CT, an ultrasound with Doppler of the liver, and MRCP without acute findings of hepatitis, bile duct dilatation or evidence of Budd- Chiari Syndrome. Her pain persisted but her LFTs continued to improve. Biliary obstruction was considered in light of other negative studies. Anti-Smooth muscle antibody came back positive, however, which prompted liver biopsy to rule out autoimmune hepatitis. With a negative liver biopsy, an ERCP was performed with initial difficulty cannulating the bile duct that eventually led to the diagnosis of choledocholithiasis. The patient's symptoms improved after biliary stent placement.

**Discussion:** This case had laboratory values suggestive of an acute hepatitis rather than biliary obstruction. While considering different causes of acute LFT elevations, this case demonstrates the importance of the patient's actual symptomatology and how it related to a past experience of cholelithiasis rather than a red herring of a positive anti-smooth muscle antibody.

### 38) ANTICOAGULATION IN A PATIENT WITH ATRIAL FIBRILLATION AND LEFT ATRIAL APPENDAGE EXCISION

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Anticoagulation in patients with atrial fibrillation and high stroke risks remains the preferred method for decreasing incidence of embolic events. It is usually well tolerated but in patients who have high risk of falling and history of gastrointestinal (GI) bleed, the risks may outweigh the benefits, especially in patients who have had their left atrial appendage excised.

68 year-old male who presented with increasing shortness of breath and bilateral lower extremity edema likely secondary to an exacerbation of his heart failure. He has ischemic cardiomyopathy with ejection fraction of 25-30%, atrial fibrillation with CHA2DS2-Vasc = 5 on warfarin status post maze surgery and excision of left atrial appendage in 2012, distant history of upper GI bleed, diabetes mellitus type 2, and hypertension. He has had multiple admissions for diuresis due to non-compliance, so when he presented, it was assumed that he just needed aggressive intravenous diuresis. Physical examination revealed signs of left sided heart failure, including elevated jugular venous pressures, bilateral crackles, and lower extremity edema. His cardiac exam was significant for a S3 gallop and an irregularly irregular heartbeat, with normal rates. Laboratory values were significant for an elevated BNP, but also a hemoglobin level that had been down trending for the last 2 years to 7.8 g/dl.

Esophagogastroduodenoscopy (EGD) revealed a small gastric ulcer that was not actively bleeding but likely contributed to his anemia, especially given his history of upper GI bleeds. Even though his stroke risk was high, warfarin was stopped as he has had excision of his left atrial appendage, where the majority of thrombi are formed.

Warfarin should have been stopped in this patient shortly after the closure of his left atrial appendage due to his history of GI bleed. He had down trending hemoglobin for the last 2 years, and with careful chart review, warfarin could have been stopped earlier before his hemoglobin dropped to a level that required transfusion.

### 39) INFECTIVE ENDOCARDITIS IN A PATIENT WITH A BIVENTRICULAR IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR

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Infective endocarditis (IE) in patients with artificial cardiac devices or valves is problematic as they act as a nidus for bacteria, creating vegetations that can eventually embolize and become detrimental to the patient.

63 year-old male who presented with hypotension and fever. He has ischemic cardiomyopathy with an implantable cardioverter-defibrillator (ICD) placed in 1996 that was upgraded to cardiac resynchronization therapy (CRT) ICD in 2014 with improvement of ejection fraction from 10-15% to 20-25%. Physical examination revealed a blood pressure of 70/49, fever of 101F, and a 2/6 holosystolic murmur at the left sternal border. Blood cultures grew *Staphylococcus epidermidis*. He was started on broad-spectrum antibiotics and pressor support.

Transesophageal echocardiogram (TEE) showed large vegetations on the tricuspid valve and all of his leads. How should leads be extracted, given one was placed in 1996 and likely fragile and fibrotic? Should they be extracted percutaneously and risk showering of septic emboli that can induce a cytokine storm causing cardiovascular collapse while he loses CRT? Or should leads be extracted via open surgical approach and risk the complications of cardiothoracic surgery? These were our clinical questions that we asked ourselves.

It is important to quickly identify IE on physical exam, especially in patients with artificial cardiac devices, as their mortality can increase dramatically if detected late.

### 40) SEVERE THIGH PAIN AS AN INITIAL PRESENTATION OF SYSTEMIC LUPUS ERYTHEMATOSIS

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**Introduction:** Systemic Lupus Erythematosus can present with vague complaints across multiple organ systems. In this setting, a broad differential diagnosis should be encouraged. SLE prognosis is variable among patients. Treatment consists of steroids and immune modulators based on severity of disease.

**Case:** A 34-year-old African American female with no significant past medical history presented with two weeks of severe bilateral thigh pain and two days of atypical chest pain with shortness of breath. She also reported fevers, chills, and nausea. On admission, she was tachycardic to the 140s with a respiratory rate in the 30s. Chest wall and thighs were severely tender to palpation. Initial laboratory evaluation was significant for myoglobinuria, CK 9276, CK-MB 183, and Troponin T 0.837. C3, C4, and TSH were normal. CT chest with contrast showed mild reactive lymphadenopathy. Transthoracic echocardiogram was significant for a trace pericardial effusion. On ECG there was low voltage with 1 mm elevations in V1 and V2. The patient was started on colchicine and aspirin for treatment of pericarditis as well as IV fluids for rhabdomyolysis. Further labs resulted and were positive for SS-A, SS-B, Anti-Smith antibody, Anti-RNP antibody, Antichromatin antibody, and ANA (1:1280); dsDNA was negative. With a diagnosis of SLE, she was started on IV methylprednisolone and showed clinical improvement. Cardiac MRI was normal. MRI of both thighs showed diffuse myositis and subcutaneous edema bilaterally. The patient was discharged on prednisone, mycophenolate mofetil, aspirin, and colchicine.

**Discussion:** The variability in presentation of new onset SLE has been well established. Pericarditis is representative of a classic presentation, but concurrent rhabdomyolysis in our patient was an unexpected finding. There are only a handful of case reports in which rhabdomyolysis is associated with SLE presentation, with most finding an inciting factor such as drug therapy or viral infection. Mainstays of treatment for rhabdomyolysis continue to be generous IV fluids, correction of electrolyte abnormalities, and sodium bicarbonate in some instances. Treatment of SLE focuses on decreasing disease burden and increasing life expectancy with steroids and immune modulators.

## 41) A NOVEL CASE OF IDIOPATHIC PANCREATITIS AND ATYPICAL HEMOLYTIC UREMICSYNDROME

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**Introduction:** Atypical hemolytic uremic syndrome (aHUS) is a complement mediated disease characterized by microangiopathic hemolytic anemia, thrombocytopenia and renal impairment. It is a rare condition and requires a high index of suspicion for definitive diagnosis. Treatment requires plasma infusions, apheresis and or immune modulating therapies.

**Case:** A 39 year-old female with history of recurrent thrombotic events on chronic enoxaparin was admitted with severe abdominal pain and elevated lipase. CT scan was consistent acute pancreatitis. Hematology was consulted on hospital day 2 for thrombocytopenia with platelet count decreasing from 243 to 67. HIT was excluded with a low pretest probability (4T score =2) and negative PF4-heparin antibody ELISA. Over the next 24 hours, the patient had a 4 gram drop in hemoglobin with a 13mg/dL associated rise in total bilirubin and LDH of 2402. Haptoglobin was <10, and direct antiglobulin test was negative. Creatinine continued to rise despite aggressive fluid resuscitation for pancreatitis. Peripheral smear at the time showed < 1 schistocyte per hpf. Over the next day renal function worsened from creatinine of 1.6mg/dl to 2.8mg/dl with persistent hemolysis and dropping platelet to nadir of 18. ADAMTS13 activity at this time was normal. Peripheral smear identified increase schistocytes to 3-5 per hpf. Due to worsening clinical picture, she was started on plasmapheresis for presumed TTP. aHUS diagnosis was considered as the patient had microangiopathic hemolytic anemia, thrombocytopenia, renal impairment with no history of diarrhea and normal ADAMTS13 activity. She received a total of 6 days of plasmapheresis with normalization of platelet count and hemolysis labs as well as improving renal function. aHUS panel was remarkable for deficiency of MCP (Membrane-cofactor protein, or CD46), consistent with heterozygous mutation.

**Discussion:** Atypical HUS is a rare condition with patients predisposed to developing thrombotic microangiopathy due to mutations in one or more complement factors. aHUS can follow a relapsing remitting course with high morbidity and mortality in the acute phase. Mutations in the complement system lead to dysregulation and activation of complement; however it is unclear at this time the exact mechanism that lead to pancreatitis induced aHUS. Our patient has a unique genetic variant of membrane-cofactor protein. Appropriate treatment of aHUS requires interdisciplinary team collaboration and timely diagnosis for plasma exchange and apheresis as well as discussion regarding ongoing immune modulating therapy to prevent reoccurrences.

## 42) A CURIOUS CASE OF CLONUS

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**Introduction:** Serotonin syndrome is a disease spectrum characterized by increased central nervous system serotonergic activity. It represents a rare and life threatening disorder that should be considered in the setting of autonomic instability with use of selective serotonin reuptake inhibitors (SSRIs) or other serotonin medications.

**Case:** 62 year old male with a history of restless leg syndrome and depression who presents from home with sweating and agitation. Over the last 5 years, he reports intermittent one day episodes of diffuse diaphoresis and uncontrollable body movements. Three days prior to presentation, patient had been “thrashing about on the floor” with poor oral intake. Medication review was significant for ropinirole, and venlafaxine. On exam, he was tachycardic and tachypneic with nystagmus, diffuse muscle rigidity, akathisia, hyperreflexia, clonus, and multiple ecchymosis on extremities. Labs demonstrated elevated creatinine kinase to 10,150 and lactic acidosis to 4.3 mmol/L. Infectious workup was negative. Clinical picture was thought to be most consistent with serotonin syndrome, given medication history of ropinirole and venlafaxine. The offending meds were held and he was treated with aggressive fluid hydration for rhabdomyolysis and lorazepam for symptoms. His symptoms resolved within 48 hours. At 1 week follow up, he reported no subsequent episodes.

**Discussion:** Serotonin syndrome can be a dangerous complication of pharmacologic treatment of mental health disorders. With increased utilization of serotonergic medications, clinicians should strongly consider this diagnosis in the setting of focal neurological findings and altered mentation. A distinguishing feature of serotonin syndrome is the presence of neuromuscular finding, which is not typical of other causes of agitated delirium. Diagnosis is based on Hunter Toxicity Criteria which states they must have taken a serotonergic agent and have one of the following: spontaneous clonus, inducible clonus with diaphoresis/agitation, ocular clonus plus diaphoresis/agitation, hyperreflexia or hypertonia with temperature > 38 and inducible clonus. Treatment involves supportive cares, removal of offending agent, sedation with benzodiazepines and consideration of serotonin antagonist including cyproheptadine.

### 43) HYPERCALCEMIA IN A PATIENT WITH SARCOIDOSIS

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**Introduction:** Sarcoidosis is a granulomatous disease of unknown etiology most commonly affecting the lungs, skin, and lymph nodes. Although patients are often asymptomatic, they may present with complaints specific to any organ system affected. Studies have shown high relative incidence and greater likelihood of chronicity with fatal outcome in African Americans. Hypercalcemia, caused by macrophage conversion of 25-hydroxyvitamin D to 1,25 dihydroxyvitamin D, amongst other potential mechanisms, is a rare occurrence in sarcoidosis, reported in just 11% of patients.

**Case:** We describe the case of a 48-year-old African American female with history of sarcoidosis, type II diabetes mellitus, and stage III chronic kidney disease who presented with a 4-week history of polyuria and polydipsia. She subsequently developed 4 days of nausea, vomiting, dizziness, and malaise. Ultimately, outpatient lab workup revealed serum calcium of 12.9 mg/dL with evidence of pre-renal azotemia. Fluid replacement was initiated and diuretics were held. Initial studies showed normal phosphorus with an appropriately suppressed intact PTH. Workup for thyroid disease and multiple myeloma was unremarkable. Since the patient had a documented history of sarcoid-induced hypercalcemia 3 years prior, vitamin D studies were obtained to evaluate for possible exacerbation of granulomatous disease. Notably, 25-hydroxyvitamin D was decreased while 1,25-hydroxyvitamin D was elevated, which is consistent with a sarcoid-mediated hypercalcemia. Although there was initial improvement in calcium with rehydration, calcitonin, and home azathioprine, she was readmitted shortly thereafter for refractory hypercalcemia. Prednisone was initiated with a goal of weaning after 3 months of therapy. Calcium levels have been within baseline since that time.

**Discussion:** Documented as a rare manifestation of disease in sarcoidosis, hypercalcemia is suggested to precipitate from autonomous activity of macrophage enzyme 1- $\alpha$  hydroxylase, which converts 25-hydroxyvitamin D to the active form of vitamin D. This has been suggested to occur in response to sarcoid-mediated inflammation in a similar manner to the patient we have presented here. Although most patients with sarcoidosis respond conservatively, glucocorticoid therapy and/or additional immunosuppressive regimen is required for a subset of those affected. Treatment response may require monitoring for 1-3 months as short courses are not likely to achieve benefit.

### 44) SEVERE HYPERTRIGLYCERIDEMIA-INDUCED ACUTE PANCREATITIS TREATED WITH PLASMA EXCHANGE

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**Introduction:** The most common etiologies for acute pancreatitis are gallstone disease and alcohol use, although hypertriglyceridemia-induced acute pancreatitis (HTG-AP) has become more prevalent and presents with a more severe disease course. HTG-AP has been associated with hypothyroidism, increased estrogen states, and uncontrolled diabetes mellitus. The triglyceride level often measures greater than 1000 mg/dL. We present a case of a young individual with metabolic risk factors, new-onset diabetes, and HTG-AP.

**Case Description:** The patient is a 24 year old man with schizophrenia on risperidone who presented from prison with nausea, vomiting, and abdominal discomfort for one day with a concerning rash over the last 1.5 months. Physical exam was remarkable for tachycardia, lipemia retinalis on funduscopy, and small erythematous white papules on his elbows, knees, and back. Admission labs were notable for HgbA1c of 14.6%, glucose of 375 mg/dL, triglycerides greater than 5680 mg/dL, and lipase of 2458 U/L. He had ketonuria on urinalysis, although without an elevated serum anion gap. He was initially treated with oral restriction, IV fluids, fenofibrate, and pravastatin. As vital signs worsened, insulin drip was started without improvement in triglyceride level. With further progression, the patient was transferred to the ICU with initiation of plasmapheresis for two sessions with goal triglyceride levels less than 500 mg/dL. Additional complications during his hospitalization included marked saponification leading to hypocalcemia, significant third-spacing manifesting as mild abdominal compartment syndrome with pressures up to 30 mmHg without surgical intervention, and focal ileus. He was discharged a diabetic diet and new-start insulin.

**Discussion:** Patients with HTG-AP often have more severe complications than other etiologies of pancreatitis. Initial management remains restriction of oral intake, intravenous fluids, and adequate pain control. Insulin drip lowers the triglyceride concentration and prevents/treats ketoacidosis, although may be employed in nondiabetics. Indications for starting plasmapheresis are unclear, but can rapidly lower triglyceride concentrations. Long-term management for lipid-lowering therapies is appropriate, often with fibrates, omega-3 fatty acids, and the inclusion of statins.

## 45) ANTI-PHOSPHOLIPID SYNDROME PRESENTING WITH NECROTIC ESOPHAGITIS

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**Introduction:** Necrotizing esophagitis is a rare clinical diagnosis most commonly associated with vascular and/or luminal obstruction. In patients with arterial thrombus, thrombophilia should be considered in the differential diagnosis. In this case of necrotizing esophagitis we highlight the underlying thrombophilia of anti-phospholipid syndrome (APS) including both clinical diagnosis and management.

**Case Presentation:** A 53 year old woman with prior history of immune thrombocytopenia (ITP), recent mitral valve replacement, and chronic left atrial thrombus on warfarin presented with four day history of progressive nausea, vomiting, dysphagia and abdominal pain. She was diagnosed with ITP many years prior to presentation following initial evaluation including normal bone marrow biopsy. She had poor therapeutic response to corticosteroids, IVIg, and rituximab with current platelet count maintained on chronic romiplostim therapy. She presented with stable hemodynamics with 3/6 holosystolic and diastolic decrescendo murmur at LLSB, diffuse abdominal tenderness and petechial rash on the forearms. She was anticoagulated with enoxaparin although developed an acute episode of melena early on in her clinical course. Given persistent dysphagia and melena she underwent EGD with extensive esophageal necrosis. Evaluation included CT angiogram showing similar 3cm filling defect in the L atrium unchanged from that visualized at the time of mitral valve replacement. Due to the presence of multiple sites of presumed thrombus, she underwent secondary work-up notable for marked elevations in anti-cardiolipin IgM and anti-beta2-glycoprotein IgM with confirmatory testing for lupus anticoagulant. She was treated with a course of IV corticosteroids and a continuous heparin infusion with serial improvement in follow-up EGD that correlated with her overall clinical course.

**Discussion:** The traditional understanding of thrombocytopenia includes a propensity towards increased bleeding risk. Included in the differential of thrombocytopenia is the diagnosis of anti-phospholipid syndrome, provided an appropriate clinical context. Using an evolving clinical history APS was diagnosed overcoming an underlying the anchoring bias of ITP. In this case, the diagnosis played a significant role in altering clinical management with initiation of corticosteroid therapy and systemic anticoagulation as well as discontinuation of romiplostim. This case highlights a unique hypercoagulable state with thrombocytopenia and builds upon prior reports of APS diagnosed over the course of thrombopoietin agonist therapy.

## 46) ANCA VASCULITIS AND BREAST CANCER: CONNECTION OR COINCIDENCE

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A 49 year old women with a history of hypertension, type 2 diabetes mellitus and infiltrating ductal carcinoma of the breast treated with lumpectomy, chemotherapy and radiation presented to an urgent care facility with three weeks of progressive malaise and nausea and several days of fever, chills and anorexia. Initial assessment revealed acute renal failure with a creatinine of 18.8 and a BUN of 144 and she was admitted to the hospital. A urinalysis revealed nephrotic range proteinuria and red cell casts. She was immediately started on high dose steroids and hemodialysis. By hospital day 2 a renal biopsy was obtained which showed cresenteric glomerulonephritis and she underwent a second round of dialysis along with plasmapheresis. Though not her chief complaint, on admission the patient mentioned a firm lump in her right breast that had been present for one month. Exam revealed a 4cm firm, non-tender, non-mobile lesion with overlying bruising. Given these concerning findings a biopsy was obtained. In the interim, treatment with cyclophosphamide was begun for her pauci-immune glomerulonephritis after discussion with oncology, given concern for active malignancy. The patient continued on dialysis for several more days. An autoimmune work-up revealed a 1:160 homogenous speckled ANA and positive perinuclear ANCA with elevated MPO. The patient's breast biopsy showed grade 3 invasive ductal carcinoma. She was discharged from the hospital and began outpatient neoadjuvant chemotherapy. She tolerated only two cycles and went on to have partial mastectomy and axillary dissection. Six month after discharge she remains dialysis-dependent.

Were these two disease process related? Was the vasculitis a paraneoplastic phenomenon? Most would suggest not, however there have been some case reports to support this idea. One study at a single center identified 15 patients with both a diagnosis of vacuities and a solid tumor within 12 months of each other over a 15-year time period<sup>1</sup>. Interestingly they found that in several cases the vasculitis was more responsive to treatment of the malignancy than standard glucocorticoid therapy. This case adds to the small number of case reports of concurrent vasculitis and malignancy.

1. Solans-Lasque R et al. Paraneoplastic vasculitis in patients with solid tumors: Report of 15 cases. *J Rheumatol* 2008;35:294-304

## 47) FUSOBACTERIUM NECROPHORUM IN AN OTHERWISE HEALTHY 19-YEAR-OLD MALE

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**Introduction:** *Fusobacterium necrophorum* is a commensal anaerobic oral and gut bacterium associated with Lemierre syndrome, a syndrome characterized by internal jugular vein thrombophlebitis and septic emboli. We aim to inform practitioners of the complicated picture of Lemierre syndrome in a patient who presents with sore throat.

**Case:** A 19 yo male presented with pharyngitis and *F. necrophorum* infection with bilateral pleural effusions. He was seen 2 weeks ago for sore throat and fevers. Strep culture was negative and patient was diagnosed with viral pharyngitis. After 1 of worsening symptoms, patient presented to an ED with fever (Tmax 102), SOB, chest pain, sore throat, chills, and headaches. He had a WBC of 4000 with 19% bands. EKG showed sinus tachy and CT showed pleural nodules and splenomegaly with no neck abscess. Abdominal US showed thickened gallbladder wall. Blood cultures grew *F. necrophorum*. Patient was admitted with bilateral exudative pleural effusions that were incompletely drained secondary to septations. Patient then transferred to our hospital seven days later. Upon transfer admission, patient was hemodynamically appropriate with diminished bibasilar breath sounds. He was started on IV clindamycin. ID expressed concern for Lemierre syndrome after reviewing a chest CT revealing multiple septic pulmonary emboli. Patient was switched to IV metronidazole and ceftriaxone. Neck CT came back negative for IJV thrombosis. TTE ruled out cardiac source of septic emboli. Repeat CT showed no splenic abscesses. IR placed bilateral pigtail catheters and CT surgery was consulted for decortication with tPA. Chest tubes were removed within a week. Patient's dyspnea improved and he was discharged on day 10 with PICC for ertapenem. On follow-up, patient's respiratory status was back to baseline.

**Discussion:** Here a healthy 19 yo male was erroneously diagnosed with viral pharyngitis and later found to have sepsis with *F. necrophorum* and septic pulmonary emboli. Lemierre syndrome is rare and predominantly affects young adults. Its innocuous presentation can mislead the physician leading to delayed treatment. *F. necrophorum* has been found in up to 27% of cases of tonsillitis. It is thus imperative that the physician be aware of the possibility of Lemierre syndrome particularly in patients presenting with sore throat. Incomplete Lemierre syndrome has been described as a fusobacterium infection with septic emboli, but without IJV thrombosis, as seen in our case. Its severity ranges from uncomplicated pharyngitis to subdural empyema and death, with a 5% mortality rate. Treatment includes 3-6 weeks of IV antibiotics, supportive care, anticoagulation, abscess drainage, and/or surgical debridement. Episodes of streptococcus negative tonsillitis may need to be cultured for *F. necrophorum*.

## 48) SPHENOID SINUS SCEDOSPORIOSIS PRESENTING WITH ACUTE BILATERAL BLINDNESS

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**Introduction:** Opportunistic fungal pathogens infect the severely ill and immunocompromised. Although *Aspergillus* is the most common pathogen, others such as *Scedosporium* are becoming more prevalent. Clinical manifestations of such infections are dependent on localization and include respiratory distress, keratitis, CNS symptoms, skin lesions, and very rarely, changes in visual acuity, which must be recognized and treated expediently to avoid permanent vision loss.

**Case:** Patient is a 78 yo M with PMH of DMII, SIADH, and HTN presenting with acute-onset bilateral vision loss and presumed sinusitis. Patient had been vacationing in Mexico when he developed headaches and intermittent jaw pain. A physician saw and discharged him with no definitive diagnosis. About 10 days prior to admission, the patient experienced bilateral acute vision loss, after which the patient returned to the US. His initial evaluation including a head CT, carotid US, and brain MRI were found to be unremarkable. MRI of orbits showed abnormal enhancement within the anterior cranial fossa and bilateral orbital apices concerning for pachymeningitis. Empiric ceftriaxone and amphotericin B was begun. He was also hyponatremic (121) and hyperglycemic (365). Extensive ophthalmological exam was unremarkable. Patient was then transferred to our tertiary care center with a BP of 186/80, Na of 119, and WBC of 10.5 with 86% segs. A reread of initial imaging revealed a left sphenoid sinus mucocele, which ENT proceeded to drain in the OR. LP findings suggested *Aspergillus* infection. However, despite amphotericin B, he continued to deteriorate with respiratory distress and worsening headaches without return of his vision. Tissue and CSF cultures then identified aseptate hyphae, specifically *Scedosporium*. Based on these results, he was switched from Amphotericin to voriconazole, micafungin, and terbinafine to cover for full spectrum of *Scedosporium*. This patient remains hospitalized on broad fungal medication coverage and will need an outpatient ophthalmology appointment for fluorescein angiogram. Patient's prognosis regarding vision remains unclear.

**Discussion:** *Scedosporium* is a growing cause of invasive infections, mainly in immunocompromised patients. It is rare compared to *Aspergillus*. It also needs both histopathology to prove tissue invasion and a culture positive for mold. Due to its dangerous sequelae, a high index of suspicion in patients who present with suspected fungal infections is required for early diagnosis and treatment with proper antifungals, as *Scedosporium* is resistant to common antifungals.

## 49) HASHIMOTO'S ENCEPHALOPATHY PRESENTING WITH RECEPTIVE APHASIA

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**Introduction:** Hashimoto's encephalopathy or Cortico-Responsive Encephalopathy associated with Autoimmune Thyroiditis (SREAT) is a relatively rare condition characterized by neuropsychiatric symptoms.

**Case report:** We present a rare case of Hashimoto's encephalopathy in a 66 year old female who was hospitalized for receptive aphasia and altered mental status. She was known to have chronic lymphocytic thyroiditis and on levothyroxine for 4 years. Her symptoms evolved over a course of few hours and were evaluated in emergency department for possible stroke. Physical exam revealed hemodynamically stable woman who was alert, with receptive aphasia. Positive for right homonymous hemianopsia, otherwise normal neurological findings. Other system exam was within normal limits. CT and MRI head ruled out stroke. Initial laboratory work up was within normal limits including TSH except elevated ESR of 28. She had continuous EEG to rule out seizures which was negative. Further work up such as paraneoplastic panel, thyroglobulin antibody and anti TPO antibodies were requested. Anti TPO antibodies were significantly elevated > 1300. Lumbar puncture showed high levels of TPO antibodies and negative for infectious etiology. She was placed on high dose of IV methylprednisolone for which she showed remarkable response. Speech and occupational therapy were involved. At her 4 week follow up with neurology and Endocrinology, she was almost back to her baseline and currently on tapering dose of steroids.

**Discussion:** Hashimoto's encephalopathy can be easily misdiagnosed in clinical practice. Especially in the elderly this can mimic an acute neurological event, dementia, psychosis, Creutzfeldt-Jacob disease and so on. Above patient had normal TSH, T3 and T4. This could be due to her being on levothyroxine. However having positive TPO in serum as well in the CSF makes her condition diagnostic. Prompt response to steroids is classical and hence the name Steroid Responsive encephalopathy.

## 50) UNCONVENTIONAL THERAPY IN THE ERA OF TARGETED AGENTS: ROLE OF CURCUMIN IN TREATMENT OF PANCREATIC CANCER

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**Introduction:** Pancreatic cancer is a notoriously aggressive malignancy with a poor prognosis and limited treatment options. We present a case of a patient with advanced pancreatic cancer who has been maintained on oral curcumin for three years after traditional chemotherapy without disease progression.

**Case:** A 67 old male presented to the emergency department with three weeks history of anorexia, nausea and worsening epigastric pain. Abdominal magnetic resonance imaging (MRI) showed a large (4.5 x 7.4 cm), bulky non-enhancing mass in the pancreatic body and tail. The patient underwent endoscopic ultrasound with fine-needle aspiration of the pancreatic mass that was consistent with adenocarcinoma. He was staged as T3N1M0 (stage II B) and categorized as borderline resectable. Accordingly, neoadjuvant chemotherapy with FOLFIRINOX (oxaliplatin, leucovorin, irinotecan, and fluorouracil) was initiated. Restaging scan after four doses showed no significant decrease in the size of the mass. He was then started on weekly gemcitabine with concurrent radiation therapy. MRI of the abdomen after 6 doses again showed disease progression. At this point he was felt to be unresectable and surgery was not pursued. Thus, he was started on FOLFIRI (irinotecan, leucovorin, and fluorouracil). Repeat abdominal imaging after five cycles showed stable disease. The patient declined further chemotherapy. He was initiated on curcumin therapy, and has remained clinically stable for over three years without any evidence of disease progression on surveillance imaging. His CA19-9 trended down and has remained within normal limits during this time period.

**Discussion:** Pancreatic cancer is an aggressive malignancy, with an estimated 48,960 new cases and 40,560 deaths per year. The majority of the patients present in an advanced stage and treatment options are limited. Monotherapy with gemcitabine long remains the standard of care for advanced pancreatic cancer. One potential agent currently being investigated is curcumin, a compound derived from turmeric. Early phase studies of the safety and efficacy of curcumin in pancreatic cancer have been conducted. Several studies have found oral curcumin to be safe and tolerable in human subjects. The most common adverse effects include cytopenias, abdominal pain, nausea, and vomiting. The patient we present here has remained on oral curcumin therapy after receiving traditional chemotherapy for over three years with stable disease on serial imaging. Such a remarkable response is unusual but has been seen in other patients in early studies of curcumin use in pancreatic cancer. This supports the promising potential of curcumin as an antineoplastic agent.

## 51) AN UNUSUAL CAUSE OF RECURRENT SEPSIS: COLONIC PERFORATION FROM AN IUD

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**Introduction:** Fistulous tracts are well-documented phenomena in chronic inflammatory states or as post-surgical complications. Some tracts are readily apparent, but some fistulae may be very difficult to detect and result in substantial morbidity.

**Case Report:** A 57-year-old woman with COPD and chronic anemia was transferred for further evaluation of recurrent sepsis. She was hospitalized 8/5; blood cultures grew *E. coli* and *Bacteroides*. Further evaluation revealed cirrhosis. ERCP was without biliary obstruction, and paracentesis did not support peritonitis. She remained septic; repeat blood cultures grew *C. glabrata*. HIDA scan suggested a choleenteric fistula. CT enterography showed an IUD which had migrated out of the uterus; it was removed on 8/21. Barium enema showed a confined perforation tract. The patient was discharged on 8/24, but presented 11/23 with sepsis and GI bleeding. Extensive testing did not identify sources of bleeding or infection. After transfer, multiple attempts at colonoscopy failed due to recurrent hypoxia, tachycardia, and leukocytosis. Flexible sigmoidoscopy did not reveal pathology. Blood cultures were persistently positive for VRE despite multiple antibiotics. On 12/25 her cirrhosis decompensated and she became encephalopathic. A transjugular liver biopsy suggested acute cholangitis. Neither MRCP nor ERCP showed obstruction or choleenteric fistula. On 1/14 she developed septic shock, and was transferred to the ICU. Lung imaging demonstrated infection. She was subsequently resuscitated through three asystolic arrests until changed to comfort cares, and subsequently died on 1/15/16. Autopsy showed a colovenous fistula in the sigmoid colon.

**Discussion:** Despite high clinical suspicion for colonic source and numerous tests and interventions, the diagnosis of colovenous fistula was not identified until autopsy. An atypical fistulous tract should be kept in mind when more common causes of sepsis have been ruled out.

## 52) STEROIDS AND RITONAVIR: A CASE OF DRUG-INDUCED CUSHING'S SYNDROME

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**Introduction:** Managing co-morbidities in HIV infected patients can be complicated given the numerous drug interactions with antiretroviral (ART) medications. Ritonavir (RTV) is a protease inhibitor (PI) that is a potent inhibitor of cytochrome P450 CYP3A4, CYP3A5 and CYP2D6. Glucocorticoid medications are metabolized by the CYP3A4 enzyme system. Taken concurrently, RTV can increase the area under the concentration versus time curve (AUC) and half-life of glucocorticoid medications resulting in iatrogenic Cushing's syndrome.

**Case:** A 54 year-old male with 23 year history of HIV, calcium pyrophosphate disease (CPPD) and COPD presented with an 11 lb weight gain, facial swelling, new onset dyspnea with increased abdominal girth over the past 3 weeks. His HIV was well controlled on darunavir, efavirenz, raltegravir and RTV with a CD4 count of 448 and undetectable viral load. Prior to these symptoms, he received 120mg intra-articular (IA) triamcinolone (TMC) injections in his knees over two months relieving his CPPD. Additionally, he was taking beclomethasone nasal spray and formoterol/mometasone. His exam showed facial plethora, moon facies, hoarseness, lateral eyebrow loss and mild bilateral hand tremor. He had no abdominal striae, an enlarged pannus with internal umbilicus without lower extremity edema. A cortisol level was < 0.8 for 4 months. Over the next five months his symptoms resolved and his cortisol remained low at 1.2 with a relatively low ACTH level of 9 with post-cosyntropin cortisol of 6.5. He was placed on anakinra and colchicine for his CPPD, his beclomethasone nasal spray was discontinued and formoterol/mometasone inhaler dose was lowered to reduce his glucocorticoid exposure.

**Discussion:** This patient presented with iatrogenic Cushing's Syndrome secondary to the interaction between RTV and TMC injections for his CPPD. The TMC injections required no entry in the EMR prior to use as this was readily available in the clinic, thus evading the drug interaction warning. There are case reports describing similar instances in HIV patients taking glucocorticoids; however, different steroids vary in their metabolism making some safer than others. HIV patients are susceptible to osteoporosis, infection and DM highlighting the importance of this interaction. This case illustrates the importance of monitoring drug interactions in HIV patients on ART and speaks to the systems issue of having the ability to dispense medications and bypass the EMR drug interaction warnings.

## 53) SOCIAL FACTORS AND THE RISKS OF READMISSION FOR COPD EXACERBATION

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**Introduction:** Hospital readmissions add over \$41 billion to healthcare costs annually and worsen quality of life for patients. Patients of low socioeconomic status have 21% higher readmission rate for chronic obstructive pulmonary disease (COPD) exacerbation. Single marital status is also associated with readmission (odds ratio 4.18). This case illustrates a patient who was admitted for a COPD exacerbation and the social circumstances that led to readmission.

**Case:** 59 year old male with COPD presented with acute worsened shortness of breath and increased sputum. He was diagnosed and hospitalized for COPD 6 years ago and was discharged with 2L/min home oxygen, which he stopped using a year ago due to financial constraints. He was single, unemployed, and had no family or reliable social support. He was a former smoker, but reported drinking up to 10 beers every other day. He presented with SpO<sub>2</sub> of 79%, and was started on frequent ipratropium/albuterol nebulizer, prednisone burst, azithromycin, and supplemental oxygen, and was admitted for COPD exacerbation. After 3 days he was feeling “better than baseline”. He was set up with home oxygen and discharged with prescriptions to complete his steroid and azithromycin course. The patient did not fill his medications and presented with repeat exacerbation 2 days later. He did not understand the consequence of medication noncompliance, and was hesitant to inconvenience his roommate, whom he relied on for transportation, to take him to his pharmacy. He required emergent intubation for the first time, as well as IV methylprednisone, ipratropium/albuterol, and azithromycin. He was extubated the next day and he was ready for discharge after 2 days. He was reeducated on the importance of medication compliance and his prescriptions were dispensed by hospital pharmacy and delivered to his room prior to discharge. He has not had another readmission in a 60 day follow-up.

**Discussion:** Readmissions after COPD exacerbation are not uncommon and, on average, more costly than the original stay (\$11,100 vs. \$7,100). Lack of social support and low income are associated with increased readmission rate. Although patients may receive optimal treatment in hospital, efforts to ensure they have adequate education and resources for home care are essential. Measures such as having pharmacy provide education about medications and ensuring that patients receive medications before discharge may help those who are at higher risk of readmission.

## 54) NECK WEAKNESS IN AN ELDERLY PATIENT

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**Introduction:** Myasthenia gravis (MG) is an autoimmune disorder resulting in weakness, typically involving the ocular, bulbar, limb, and respiratory muscles. There is a bimodal distribution to age of onset with an early peak in second and third decade and a late peak in sixth to eighth decade. Symptoms are often transient early in the disease course and become more persistent as the disease progresses. Due to conditions that may mimic specific symptoms of myasthenia, a broad differential diagnosis should be formulated, and the diagnosis should be confirmed with immunologic and electro-physiologic testing.

**Case:** We discuss an 83-year-old female with history of HTN, DVT/PE, and COPD who presented with a 3-week history of difficulty holding her head up while walking. During the time her neck weakness was being evaluated in the outpatient setting, she presented to the ED with 2 days of sore throat and associated difficulty swallowing. Her systolic BP at the ED was >200, thought to be related to inability to swallow her medications. Physical exam showed weakened gag reflex, mild lower facial droop, and mild right sided ptosis. CXR was read as equivocal for pneumonia and empiric antibiotics were started. CT head and neck was negative for abscess or mass. Speech therapy reported severe oropharyngeal delay and poor bedside swallow. She was started on methylprednisolone for a possible Bell’s palsy and neurology was consulted. On day 4 of hospitalization, she was found to be obtunded and hypotensive. She subsequently developed an episode of ventricular tachycardia, and an ABG showed a pH of 7.14. She was intubated and transferred to the MICU due to acute hypercapnic respiratory failure. MRI brain showed left thalamic chronic microhemorrhage and old infarct, but no bulbar lesion or tumor. At this point, her bulbar weakness was most concerning for MG. EMG showed postsynaptic neuromuscular junctional disorder consistent with MG, and an antibody panel was positive for acetylcholine receptor blocking, binding, and modulating antibodies. CT chest was negative for thymoma. She was treated with plasmapheresis, physostigmine, and prednisone with improvement and was eventually able to be extubated.

**Discussion:** MG is caused by autoantibodies directed at the acetylcholine receptor in the postsynaptic membrane of the neuromuscular junction. A high index of suspicion should be maintained in elderly men and women who present with unusual symptoms such as isolated neck weakness. Myasthenic crisis may be precipitated by a variety of factors, and caution should be exercised in administration of steroids while myasthenia remains in the differential diagnosis. While high-dose glucocorticoids are used in treatment, the onset of benefit generally begins weeks after treatment, and there may be a transient worsening of symptoms after administration when the patient does not receive concurrent rapid immunotherapy treatment with plasmapheresis or IVIG.

## 55) LARGE PERICARDIAL AND PLEURAL EFFUSION DUE TO SEVERE PULMONARY HYPERTENSION AS A RESULT OF LIMITED SYSTEMIC SCLEROSIS

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**Introduction:** Systemic sclerosis (Ssc) is a rare connective tissue disease defined by thickened, sclerotic skin in addition to extracutaneous organ involvement. Here we present a case of limited Ssc which presented as a large pericardial and pleural effusion secondary to severe pulmonary artery hypertension (PAH).

**Case Description:** A healthy 66 year old male with history of GERD, three years of Raynaud phenomenon, and hypertension presented with 2-3 months of progressive shortness of breath and lower extremity edema. On examination, heart rate was 110 beats per minute and blood pressure was 100/65 mm Hg. He had an elevated JVP at 12cm, 1+ edema to mid shin, and decreased breath sounds on the right. A chest x-ray showed a large pleural effusion on the right. An echocardiogram showed a large pericardial effusion and PAH, later confirmed on right heart catheterization with PA pressure of 90/34. Pleural fluid was found to be transudative. On subsequent closer examination, he was noted to have scattered telangiectasias on his face as well as thickened, hardened skin of his digits, several with nail fold capillaries. His antineutrophil antibody (ANA), anticentromere antibody (ACA), and antitopoisomerase (anti-Scl70) testing was normal as was ESR and CRP.

**Discussion:** Despite the negative ANA, ACA and anti-Scl 70, our patient's history of Raynaud phenomenon and GERD in combination with his findings of sclerosis of skin distal to the wrist, telangiectasias, and severe pulmonary hypertension was sufficient to make the diagnosis of limited Ssc. Interestingly, while ANA is typically highly sensitive for limited systemic sclerosis (about 90%), ACA is positive in only about 50% of cases and anti-Scl70 in only 5-10%. Ten to fifteen percent of cases of limited Ssc involve PAH, typically late onset. When severe, PAH may lead to pleural effusions and, more rarely, large pericardial effusions. The mechanism for both effusions is likely related to right heart failure, as evidenced by the transudative nature of the pleural fluid in this case. Our patient is currently receiving continuous epoprostenol infusions for his severe PAH.

**Conclusions:** Severe PAH and right heart failure is a rare but ominous cause of large pericardial and pleural effusions. Careful clinical (re)examination in unexplained cases of severe PAH can help provide clues to establish the underlying etiology, in this case limited systemic sclerosis.

## 56) THE SODIUM SEESAW

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**Introduction:** Diabetes Insipidus (DI) is a rare condition involving defective renal water conservation that leads to a relative free water deficit within the body. Clinically characterized by thirst and copious water intake, and polyuria, it has many etiologies. It can be neurologic with decreased production of ADH, known as central DI, or renal, with unresponsiveness to ADH known as nephrogenic DI. Our case illustrates an unusual presentation of partial central DI secondary to a pituitary gland lesion.

**Case:** Our patient is a 49 y/o woman with history of systemic lupus erythematosus, lupus nephritis, and rheumatoid arthritis who presented with dizziness and hypernatremia. She reported poor oral intake and did not demonstrate polyuria, so initially she was felt to have a free water deficit and was started on IV fluids with dextrose 5% to replete this deficit. Her sodium downtrended appropriately, but after the fluids were stopped her urine output would increase considerably and her sodium would rebound to the 150s. Urine labs showed an inappropriately low urine osmolality and thus diabetes insipidus was suspected. Endocrinology was consulted to evaluate for DI, and a free water restriction test showed only a slight uptrend in urine osmolality with a considerable increase in serum osmolality. Administration of ddAVP then increased urine osmolality to 325, meeting criteria for partial central DI. This was followed by endocrine testing and a sellar MRI which demonstrated a 1.8cm pituitary lesion, explaining the etiology of our patient's presentation.

**Discussion:** Here we report a case of partial central DI resulting from a pituitary gland lesion. Initially it was hypothesized that the patient was dehydrated, given reported dizziness prior to admission, poor oral intake, and her lack of polyuria on presentation. However, she was euvolemic on exam and her serum sodium would only transiently respond to free water administration. Recent historical elements such as a lupus flare and recent cessation of hydrochlorothiazide further complicated the differential. However, studious workup of her hypernatremia with water restriction and administration of ddAVP eventually led to the true diagnosis of partial central diabetes insipidus, as judged by a >10% increase in urine osmolality with ADH administration and the discovery of a pituitary gland lesion. Our case illustrates the need to be constantly vigilant for data that does not fit the presumed clinical picture and to adjust the differential diagnosis accordingly.

## 57) OVERWHELMING SEPSIS AND WATERHOUSE-FRIEDERCHSEN SYNDROME CAUSED BY *CAPNOCYTOPHAGA CANIMORSUS* IN AN IMMUNOCOMPETENT HOST

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**Introduction:** *Capnocytophaga canimorsus*, found in cat and dog normal oral flora, is emerging as an identified cause of critical bacterial infection in both immunocompromised and immunocompetent hosts. Rare case reports have recognized that this bacterium can lead to overwhelming sepsis with multiorgan failure and the fatal Waterhouse-Friderichsen syndrome.

**Case Report:** A previously healthy 60 year-old male presented as a transfer to the ICU from a community hospital with septic shock and multi-organ failure. He had been in his normal state of health until approximately 24 hours prior to admission when he developed fevers/chills and generalized malaise. He then had rapid onset of overwhelming illness beginning with purpura fulminans and shortness of breath. Shortly after presentation to the outside hospital, cultures were obtained, he was aggressively fluid resuscitated and broad spectrum antibiotics were initiated. After transfer, he declined rapidly despite increasingly broad antimicrobial coverage, ultimately requiring intubation for hypoxic respiratory failure, dialysis, vasopressor support and stress-dose steroids. One of two blood cultures at the outside hospital grew pleomorphic gram negative rods, but these were unspiciated until post-mortem when *Capnocytophaga canimorsus* was identified. Despite aggressive care, he continued to deteriorate and his family elected to transition to comfort care measures. On autopsy, he was noted to have diffuse evidence of DIC with purpura fulminans, necrosis within the spleen, and microangiography in bilateral kidneys. He also had near total hemorrhagic and necrotic destruction of the adrenal glands, suggesting that acute adrenal insufficiency was most likely the cause of his rapid clinical deterioration and death. This is consistent with Waterhouse-Friderichsen syndrome or bilateral adrenal hemorrhagic infarction.

**Discussion:** Waterhouse-Friderichsen syndrome has been classically associated with sepsis secondary to *Neisseria meningitidis* and *Pseudomonas aeruginosa*, but it has more recently been identified in cases of *Capnocytophaga canimorsus*. This case serves as a reminder that even in immunocompetent hosts, severe rare infections can occur with fatal manifestations despite early intervention and management.

## 58) MULTISYSTEM FAILURE SECONDARY TO BABESIOSIS SEPSIS – ALL THAT CAN HAPPEN WITH BABESIOSIS

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**Introduction:** Babesiosis is an emerging parasitic tick-borne disease. In the United States, 95% of the cases were reported by 7 states: Connecticut, Massachusetts, Minnesota, New Jersey, New York, Rhode Island, and Wisconsin. *Babesia microti* is the most common pathogen in the US, with the primary vector for the parasite being deer tick, *Ixodes scapularis*.

**Case:** We report a case of 74 year old male with asplenia, who was admitted to the hospital with fever, weakness, rapidly worsening anemia, thrombocytopenia and renal function. He was tested positive for Babesiosis and was started on clindamycin and quinine initially. But his condition progressed towards multi organ dysfunction secondary to very high parasitemia ( $\approx 20\%$ ) and it necessitated addition of atovaqone and azithromycin to the antibiotic regimen along with erythrocyte exchange transfusions to clear the high parasite load, continuous renal replacement therapy for declining renal function and ventilator support for respiratory failure. He needed a 4 week hospital stay and remained positive for *Babesia* Nucleic Acid Amplification testing for 14 weeks.

**Discussion:** Severity of Babesiosis infection can range from asymptomatic to fatal and, though most infections are self-limited or cured with a short course of antibiotics, some patients have prolonged illness despite antimicrobial therapy, requiring prolonged hospitalization stays. Various host risk factors like immunocompromised state, pregnancy, chronic steroid use, asplenia etc. along with the level of parasitemia will determine the outcome of the patient infected with *Babesia*. The patients with high parasite load and other risk factors might need more than conventional antibiotic therapy, clindamycin & quinine.

**Conclusion:** In cases such as ours with risk factors as well as high parasite load, it is worthwhile considering the broad antibiotics, exchange transfusions and involving multidisciplinary teams earlier in the course of infection, to reduce complications and length of hospital stay.

## 59) RARE AND RARER: MORAXELLA CATARRHALIS BACTEREMIA WITH MEDIASTITIS IN A PATIENT WITH LOEYS-DIETZ SYNDROME.

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**Introduction:** Moraxella catarrhalis (MC) is a rare cause of bacteremia. While few cases of MC bacteremia have been reported in the literature, abscess or mediastinal involvement has never been described. Loeys-Dietz Syndrome (LDS) is a rare, genetic, collagen-vascular disease associated with multiple vascular aberrancies that was first described in 2005.

**Case:** A 56 year-old female with LDS, status post ascending and descending aortic graft placements for aortic dissection, was admitted with septic shock. Blood cultures were positive for MC. She was treated with a 4-week course of IV antibiotics. Within 24-hours of antibiotic completion, blood cultures again grew MC, and a mediastinal abscess was discovered adjacent to the ascending aortic graft. After discussions with the patient, multidisciplinary medical and surgical teams, an extended six-week course of high-dose ceftriaxone therapy was chosen over surgical intervention. The patient was subsequently transitioned to a life-long oral regimen of levofloxacin and rifampin for continued suppression.

**Discussion:** Endovascular devices carry a high infection risk, even from organisms of low pathogenicity. Management of MC bacteremia associated with mediastinal abscess has not yet been reported. Although antibiotic therapy without surgical intervention will not result in infection eradication, our approach using an increased dose and prolonged course of ceftriaxone followed by an oral suppressive regimen of levofloxacin and rifampin was a conservative choice made with patient involvement. Even after 24 months of suppressive antibiotic therapy, the patient continues to do well and has not required recurrent hospitalization.

**Conclusion:** MC is a pathogen of increasing importance requiring consideration in the differential of causative agents of prosthetic graft infections and mediastinal abscesses. In cases such as ours where surgical intervention is not pursued, a high dose and prolonged course of antibiotics followed by a long-term suppressive oral regimen was used successfully to limit further infectious complications.

## 60) COBALT SPURS HARD METAL LUNG DISEASE

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**Introduction:** Interstitial lung disease (ILD) is characterized by diffuse inflammation and destruction of the parenchyma of the lung. Giant cell interstitial pneumonia (GIP) is an uncommon variant of idiopathic ILD and is now used interchangeably to describe hard metal lung diseases. This rare occupation-related ILD is acquired by exposure to tungsten carbide cobalt, a metal used in diamond polishing industries.

**Case:** Patient is a 57-year-old gentleman with a 23-year history of giant cell interstitial lung disease. In 1993, at the time of initial presentation, patient presented to his PCP with increased shortness of breath and weight loss. Following initial work-up, patient had a transbronchial biopsy, which showed giant cell interstitial lung disease with characteristic lymphocytic infiltrates and giant cells in the alveoli. The shortness of breath remained relatively stable until 2007, when patient suffered a spontaneous pneumothorax and respiratory function subsequently decompensated. In 2010, patient was referred to Pulmonary Clinic for a formal evaluation for lung transplantation. Physical exam revealed a cachectic gentleman with visibly increased respiratory effort. Lung exam was notable for inspiratory and expiratory crackles at the bilateral bases. Arterial blood gas showed pH of 7.40, pO<sub>2</sub> of 64, pCO<sub>2</sub> of 47, and bicarbonate of 28.1. Pulmonary function tests were significant for an FVC of 58% predicted, an FEV<sub>1</sub> 70% predicted and a DLCO of 29% predicted. Chest CT was notable for fibrotic changes, nodules and ground-glass opacities. Social history later revealed that patient had used a diamond saw to create woodworking tools, an exposure associated with cobalt from tungsten carbide drills. In 2010, patient declined further consultation for lung transplant. Since then, he has maintained submaximal respiratory control at home using ipratropium, formoterol, and colistimethate. He has been hospitalized three times in the past year for respiratory failure secondary to pseudomonas pneumonia.

**Discussion:** Interstitial lung disease occurs when the lung is repetitively exposed to an internal or external injury. While many cases of ILD are idiopathic, there is a rare subset, such as GIP, which are known to be caused by cobalt exposure. Early identification is imperative to improve patient management and prognosis. This case is an important reminder to explore all occupational and environmental exposures, no matter how rare, in patients presenting with diffuse lung disease.

## 61) LYME CARDITIS BURIED BENEATH ST SEGMENT ELEVATIONS

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### **Objectives:**

1. Recognize the various presentations of Lyme carditis
2. Understand the importance of history-taking and physical exam when developing differential diagnosis for acute myocardial infarction in young patients

**Introduction:** Lyme disease is caused by the spirochete *Borrelia burgdorferi* and is carried to human hosts by infected ticks. There are nearly 30,000 cases of Lyme disease reported to the CDC each year, with 3-4% of those cases reporting Lyme carditis. The most common manifestation of Lyme carditis is partial heart block following bacterial-induced inflammation of the conducting nodes.

**Case:** A 45-year-old gentleman presented to the hospital with intense chest pressure and tightness. Lab studies were remarkable for elevated troponins and liver enzymes. EKG demonstrated normal sinus rhythm with mild ST elevations in the inferior and high lateral leads and Q waves in III and aVF. Following EKG, he was taken for left heart catheterization. The procedure was unremarkable. Transthoracic echocardiogram revealed normal ejection fraction and no wall motion abnormalities or effusion.

Three weeks prior to hospital presentation, patient had gone hunting near Madison. One week prior to admission, he noticed an erythematous lesion on his right shoulder. It had progressed from a small bug bite to a larger, targetoid bull-eyes rash. Because of his constellation of history, arthralgias and carditis, he was started on ceftriaxone to treat probable Lyme Disease. He made rapid improvement upon starting antibiotics with relief of cardiac and arthritic symptoms. Prior to discharge, he was switched to oral therapy as recommended by Infectious Disease. Lyme titers and Western Blot analysis ultimately returned as positive.

**Discussion:** This case illustrates the importance of thorough history taking and extensive physical examination when assessing a case of possible acute myocardial infarction. Lyme carditis is an uncommon cause of a comparatively common hospital presentation of acute myocardial infarction. Because Lyme carditis is reversible, recognition of this syndrome in young patients, whether in the form of AV block, myocarditis, or acute myocardial ischemia is critical to the initiation of appropriate antibiotics in order to prevent permanent heart block, or even death.

## 62) LOVE IT OR LIST IT: THE HIDDEN DANGER OF HOME RENOVATION

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**Introduction:** Prevalence of lead poisoning in the adult population in the United States has declined due to strict regulatory reforms in the work environment. However exposures from renovating older homes, use of unregulated dietary supplements and some industrial exposures continue to occur. The clinical manifestations of lead poisoning can often masquerade as other diagnoses and can result in significant morbidity and mortality.

**Case Description:** A 50 year old woman presented to the clinic with unexplained weight loss, decreased appetite, insomnia and poor concentration. She was dealing with several stressors such as unemployment and family issues at the time. While this could have explained some of her symptoms, she proudly showed pictures of how she was keeping herself occupied by renovating her older house built in the 1910s for the last four months, scraping paint by hand on a daily basis. She used a mask infrequently, used dust cloths to catch scraped paint and shook them off for re-use. Her exam was unremarkable and lab tests were ordered. Lead levels were elevated at 47  $\mu\text{g}/\text{dl}$ ! Patient was asked to stop working on her house and lead abatement measures were instituted and levels monitored. When lead levels did not decrease significantly on recheck, chelation therapy was initiated, leading to a sharp decline in lead levels to 15.8  $\mu\text{g}/\text{dl}$  within two weeks. Her symptoms began to slowly improve and she chose to discontinue chelation, however lead levels fluctuated over the next several months rising above the nadir at times. This was attributed to lead leeching from the bones into the bloodstream even after cessation of exposure, before finally decaying to 5.9  $\mu\text{g}/\text{dl}$  over 2.4 years.

**Discussion:** This case illustrates how a thorough history is critical to making a diagnosis of lead poisoning whose clinical manifestations can mimic several other diagnoses. This case is consistent with newer research which demonstrates that detrimental clinical manifestations from lead exposure can occur at lower levels than previously thought and recognition of lead poisoning has important therapeutic implications.

### 63) VITAMIN DEFICIENCY PRECIPITATES DIFFUSE THROMBOEMBOLI

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Thrombophilic disorders have a multitude of etiologic risk factors. Atypical presentations necessitate the investigation of unique and uncommon sources.

A 31 year old man was scheduled to see his primary care physician due to 2 weeks of progressive dyspnea. The morning of the appointment the patient experienced sudden and painful swelling of left upper extremity. The chest x-ray findings in clinic were suspicious for PE and the patient was referred to the UW ED. Physical exam in the ED revealed tachycardia, coarse breath sounds, painful inspiration and positional changes and swollen right lower and left upper extremities. ED work up was significant for CT angiogram of the chest which showed bilateral pulmonary emboli as well as suspicion for embolic involvement of the left subclavian and left brachiocephalic veins. Transthoracic echocardiogram revealed dilation of the right ventricle and thrombus-in-transit with partial occlusion of the left innominate vein. Peripheral doppler of the right lower extremity showed acute DVT in the right mid to distal femoral vein, popliteal vein and posterior tibial vein. Doppler of the left upper extremity showed occlusive DVT in the left proximal cephalic and subclavian veins and non-occlusive thrombus in the left axillary vein. Treatment was initiated with IV heparin and over the subsequent few days swelling and pain demonstrated subjective and mild objective improvement. During the initial work up, blood tests revealed a macrocytic anemia with a low reticulocyte count. Iron and vitamin studies showed undetectable levels of B12 and folate. Hematology was consulted, confirming the greatly elevated homocysteine levels were sufficient to predispose this patient to this level of thrombophilia. The patient was supplemented with IV B12 and folate; which then began to improve the reticulocyte count and anemia. He was later consented for oral anticoagulation in preparation for discharge on hospital day 5.

This case is a good demonstration of an atypical presentation of thrombophilia in a healthy young patient by a lack of the common predisposing risk factors such as recent surgical history, trauma, recent hospitalization, immobility, previous history of thromboembolism. Although genetic tests were not conducted, it is possible that there may have been coexistent genetic predisposition to thrombophilia in conjunction with the hyperhomocysteinemia that precipitated this diffuse thromboembolic disease.

### 64) ANOTHER REASON FOR AN ABNORMAL TROPONIN RESULT – HETEROPHILE ANTIBODIES

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**Introduction:** Cardiac troponin is the preferred biomarker for diagnosis of myocardial infarction (MI) because of its sensitivity and high specificity for myocardial injury. In common day-to-day use current assays are so accurate that we rarely entertain the possibility that an elevated troponin level might be a lab error.

**Case Presentation:** A 47 year old woman with a history of anxiety and ADHD presented with subjective palpitations and reported a pulse of 200 bpm measured by her smart phone. Medicines included methylphenidate and she reported liberal caffeine intake. She denied chest pain or shortness of breath. Aside from sinus tachycardia the initial electrocardiogram (ECG) was normal. Troponin was markedly elevated at 12.5 ng/ml (normal < 0.03 ng/ml). Her subsequent evaluation included a coronary angiogram, 3 echocardiograms, 6 ECGs, a cardiac MRI, CT pulmonary angiography and implantation of a loop recorder. She received empiric therapy with Verapamil, sublingual nitroglycerine, and aspirin 81 mg. All imaging studies and ECGs were normal. She had no further symptoms but troponin levels remained markedly elevated.

Of note, troponin results 1 year earlier with a different manufacturer's analyzer were too low to measure. A blood sample that was reported as having a very high troponin at UW Hospital was reanalyzed at the Middleton VA Hospital. The troponin was too low to measure on a different manufacturer's analyzer at the other hospital. When the same sample was treated with polyethylene glycol to remove heterophile antibodies, the analyzer at the first facility could no longer detect troponin.

**Discussion:** Elevated troponin levels are almost invariably due to myocardial injury. In this instance we were late to consider the possibility of a false positive lab result, in part because of the routinely excellent clinical performance of current troponin assays. Heterophile antibody interference in troponin testing and other immunoassays are uncommon but well described and should be considered when any immunoassay yields results that are discordant with the clinical circumstance.

## 65) PERSPECTIVES ON CONSCIENTIOUS OBJECTION IN FAMILY MEDICINE RESIDENCY PROGRAMS

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Conscientious objection is the refusal to participate in activities one finds morally objectionable. This study examines its application in practice from the perspective of family medicine residents and program directors.

Participants were randomly selected and questionnaires were mailed; results were analyzed using descriptive frequency statistics. A total of 205 resident physicians (12.5% response rate) and 22 program directors (24.4%) participated. Twenty-one (82%) programs reported that issues of conscientious objection occurred at least biannually. One hundred and forty (68%) residents reported objection to at least one of the listed procedures and 98 (48%) anticipated objection. Only 35 (37%) residents disclosed their concern and 71 (75%) indicated barriers to doing so. Seventy-one residents (72%) had developed a plan to facilitate care in the event of conscientious objection. Of program directors, two (10%) had a specific conscientious objection policy for residents, and eight (38%) had a 'general approach.'

Most residents reported a moral objection to at least one procedure and we recommend further exploration of the trends surrounding conscientious objection. This study's low response rate is likely related to the mailing of surveys, and moving forward, an electronic version of the survey will be used to assess current attitudes about this important issue.

## 66) MALARIA PARASITEMIA AS A CAUSE OF MUSCULOSKELETAL PAIN AND INTERMITTENT DIARRHEA

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**Introduction:** Malaria is a serious and sometimes fatal disease caused by parasites. Although this is a common and fatal disease in other parts of the world, only 1,500 cases are reported in the USA each year.

**Case:** An 84 year-old African female with a past medical history of severe lumbar spinal stenosis and recent travel to Kenya presented to the ED with a chief complaint of lower extremity pain and intermittent diarrhea of 1 month duration. There were no reports of fever, vomiting, or abdominal pain. Initial exam revealed left leg (LLE) weakness and absent distal pulses. Initial labs were notable for mild leukocytosis, and a stool sample was negative for parasites or enteric pathogens. No diarrhea was observed in house. The patient was diagnosed with severe LLE peripheral vascular disease (PVD), and underwent peripheral angiography and stent insertion to the L superficial femoral artery (SFA). She was discharged home with improvement in her symptoms.

13 days later the patient was re-admitted for worsening LLE pain, diarrhea, altered mental status, and fever of 102.2 F. Worsening anemia required a blood transfusion, and CT angiogram revealed a patent L SFA stent. Given recent travel to Africa a blood smear was done and revealed parasitemia consistent with malaria, *P. falciparum* type. Given a parasitemia load of 12%, the patient underwent exchange transfusion reducing the load to 2%. Confusion persisted, the patient was diagnosed with cerebral malaria, and was transferred to the ICU to initiate treatment with IV quinidine. She was later transitioned to oral Artusunate. The patient's mental status improved and she was discharged home on Malarone.

**Discussion:** This case illustrates that symptoms of malaria are generally non-specific and may be misdiagnosed by clinicians. Recognition of malaria is critical to institution of appropriate therapy and prevention of morbidity and mortality.

## 67) SYSTEMIC SEPTIC EMBOLIZATION IN TRICUSPID VALVE ENDOCARDITIS WITH PATENT FORAMEN OVALE

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**Introduction:** Infective endocarditis is a life-threatening condition with significant morbidity and mortality. Classically, IE presents with a persistent fungemia or bacteremia and acute valvular involvement with immunological, vascular, and peripheral embolic phenomena. However, this presentation occurs in a minority of patients, especially those with right-sided valvular disease more often associated with injection drug use. Early diagnosis and prompt initiation of antimicrobial therapy is essential.

**Case:** 52 year old woman with history of injection drug use who presented to an Emergency Department with neck pain. Blood cultures drawn on presentation grew methicillin sensitive *Staphylococcus aureus*, and subsequent transthoracic echocardiogram revealed a 26mmx12mm highly mobile vegetation on her tricuspid valve as well as evidence of an intracardiac shunt, consistent with a patent foramen ovale. Her neck pain on presentation was secondary to cervical epidural abscess; she additionally had evidence of septic pulmonary emboli on chest imaging. She had sustained bacteremia despite 4 days of antibiotic therapy; thereafter, cultures remained negative. She was transferred for tertiary care on hospital day 11, at which time her antibiotic coverage was narrowed to nafcillin. Despite negative blood cultures, she had a persistent leukocytosis and developed a large number of non-tender erythematous macules on her hands and feet consistent with Janeway lesions, representing systemic septic emboli. Her hospital course was complicated by acute hepatitis A on presentation, development of acute renal failure secondary to acute interstitial nephritis secondary to nafcillin versus emboli requiring hemodialysis, GI bleed secondary to ischemic duodenitis, culture-negative spontaneous bacterial peritonitis and small bowel obstruction. These complications were medically managed, and she was discharged on hospital day 43 to complete antibiotic therapy with cefazolin.

**Discussion:** The incidence of infectious endocarditis has been stable, though increasingly attributable to *Staphylococcus aureus*, especially in injection drug users. While right-sided endocarditis typically lacks features of classic endocarditis such as peripheral emboli and immunologic vascular involvement, patients with intracardiac shunts can experience paradoxical systemic septic embolization, including to the extremities and internal organs.

## 68) DASATINIB TOXICITY: A RARE CAUSE OF REVERSIBLE PULMONARY HYPERTENSION AND FLUID OVERLOAD

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**Introduction:** The mainstay of therapy for Chronic Myelogenous Leukemia (CML) patients is tyrosine kinase inhibitor therapy. Initial studies had been conducted on imatinib; eventually, other chemotherapeutic agents like dasatinib and bosetinib were available for use.

**Case:** A 76 year-old Caucasian female with a past medical history of CML in remission with dasatinib therapy presented with fluid overload. The patient was diagnosed with CML 5 years prior to presentation and had been in remission on dasatinib ever since. The patient also had a past medical history of CKD stage 3, HTN, and mild pulmonary HTN which was diagnosed prior to starting chemotherapy. On presentation, the patient described acute worsening of shortness of breath and greater than 15 pound weight gain over one month prior to admission. She was evaluated by the cardiology service, found to be fluid overloaded, and was started on diuretic therapy. Hematology service evaluated the patient and recommended holding dasatinib. Due to her underlying pulmonary hypertension which was found to be worse on a repeat echocardiogram, she was started on both IV and inhaled epoprostenol. The patient's hospital course was complicated by oliguric kidney failure, hypotension requiring vasopressors, and severe hyponatremia. She was started on CVVH but continued to decline. Ultimately, the family decided to pursue comfort care measures and the patient expired peacefully at the bedside.

**Discussion:** This case illustrates the potential side effects of dasatinib therapy including fluid overload and pulmonary hypertension. In patients with a history of pulmonary hypertension, it may be prudent to begin diuretic therapy concurrently or dose-adjust chemotherapy regimens to prevent hospitalizations, morbidity, and possibly mortality.

## 69) HERPES SIMPLEX 2 (HSV-2) MENINGITIS IN AN IMMUNOCOMPETENT PATIENT

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**Introduction:** Meningitis is an inflammation of the meninges that presents with headache, neck stiffness, and photophobia. It can be subdivided into two categories: bacterial and aseptic. Etiologies of aseptic meningitis include viruses, medications, and systemic illnesses. Early recognition of etiology is essential since treatment, severity, and prognosis differ. Herpes simplex virus 2 (HSV-2) is a common cause of aseptic meningitis resulting in severe morbidity and mortality in immunocompromised patients. Here we present a case of an immunocompetent adult with HSV-2 aseptic meningitis.

**Case:** A 31-year-old female with PMH of migraine headaches and genital herpes (last outbreak 2014) presented to the ED with a 24-hour history of throbbing headache, neck stiffness, photophobia, and nausea. On exam she was febrile to 101.5F and tachycardia. Laboratory data showed WBC count 6300/cm<sup>3</sup> and normal electrolytes. Due to concern for meningitis, she underwent a lumbar puncture and was started on empiric ceftriaxone, vancomycin, and acyclovir. CSF studies revealed an aseptic pattern: elevated WBC count (744/uL) with lymphocyte predominance (96%), mildly elevated protein (82 mg/dL), normal glucose level (53 mg/dL), and negative gram stain. Her fever resolved on hospital day 1 but she continued to have meningismus. Infectious disease (ID) was consulted. CSF viral studies were positive for HSV-2 on hospital day 2 and antibiotics were discontinued. Intravenous acyclovir was continued through hospital day 3 when she was discharged with oral valacyclovir for 14 days.

**Discussion:** Meningitis is an inflammation of the meninges and is broadly categorized as either bacterial or aseptic. HSV-2 is the second most common cause of aseptic meningitis and is seen in 8-25% of people with known HSV-2. Approximately 85% of patients with primary HSV-2 meningitis have genital lesions within the prior 7 days to presentation. Here we present a case of a patient who had not had an outbreak of genital herpes in nearly 2 years. CSF in patients with HSV-2 meningitis shows elevated leukocytes (lymphocyte predominance), normal glucose level, and elevated opening pressure. Despite HSV-2 being a fairly common cause of aseptic meningitis, clear guidelines for treatment do not exist and suppressive therapy with valacyclovir has not been shown to prevent recurrent HSV-2 meningitis. Aseptic meningitis due to HSV-2 should be considered as a cause of headache in immunocompetent patients with history of genital herpes especially in the context of neck stiffness, photophobia, headache, and fever.

## 70) CERVICAL ESOPHAGEAL FISTULALIZATION AS A COMPLICATION OF ANTERIOR CERVICAL DISCECTOMY AND FUSION

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A 63-year-old female with past medical history of degenerative joint disease, status post cervical spine fusion for persistent pain with redo procedure complicated by esophageal perforation and leak, retropharyngeal abscess, left vocal cord paralysis, cervical osteomyelitis and resultant stricture requiring intermittent dilations presented to gastroenterology lab for esophagogastroduodenoscopy (EGD) revision. EGD 1-month prior showed a large ulcerated lesion below the upper esophageal sphincter (UES). During this scoping, an esophageal fistula was found just below the UES connecting with a cervical plate. With this, the patient was admitted to the medicine service. Upon admission, the patient had a chief complaint of neck pain, odynophagia, nausea, and emesis. Initial examination was unremarkable. Due to dysphagia and worsening esophageal ulceration, the patient was made NPO and started on nasogastric tube (NGT) feedings. CT surgery, neurosurgery, and otolaryngology (ENT) were consulted. CT surgery ordered CT with contrast of the chest and neck and NGT removal due to her stable condition. Patient tolerated a soft diet and imaging showed air within the esophagus directly contacting the anterior cervical plate in the C5-C6 region. CT surgery and neurosurgery decided to treat patient conservatively with no surgical intervention. Patient was discharged with plan to follow-up with neurosurgery and CT surgery in 2-3 weeks. At follow-up, patient was tolerating soft foods without dysphagia and it was discussed with the patient that surgery would be fraught with complications and could worsen her condition. Patient saw ENT who stated she would need a regional pedicled soft tissue flap with gastric pull-up versus free tissue transfer. At best she would continue on a soft diet, but substantial improvement was unlikely.

Complications of anterior cervical plate and screw fixation include screw backout or breakage, and plate fracture or migrations. Neurological problems include dural tears, and spinal cord or nerve injuries. Those related to neck surgery may occur including dysphagia, hematoma, and esophageal perforations. Most esophageal injuries are iatrogenic. Diagnosing perforations is important due to possible infection. Signs include pain after adequate treatment with redness, swelling, tenderness, and crepitus of the neck. Also, air in the cervical fascial spaces or mediastinum indicates injury. Diagnosis is made via esophogram, esophagography with CT, and esophagoscopy. Extra esophageal air on CT is the most definitive finding for perforation. Treatment can be conservative with extraoral feeding via NGT or gastrostomy in minor cases. Severe cases require primary closure of the perforation with or without muscle flaps, surgical drainage, esophageal rest and nutritional support, and removal of hardware if necessary.

## 71) NASOPHARYNGEAL NEUROENDOCRINE CARCINOMA: A CASE REPORT

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Neuroendocrine carcinoma (NEC) of the sinonasal tract and nasopharynx remains rare. Common symptoms and signs include neck masses, nasal obstruction with bleeding, and occasional aural changes. The World Health Organization (WHO) classification of tumors of the head and neck identifies three categories of primary malignant neoplasms in the nasopharynx. While NEC exist within head and neck, it is not currently included in WHO classification, with the GI tract and lungs being more common sites of origin. Nasopharyngeal NEC are more infrequent and can mimic lymphomas, neuroblastomas, or mucosal melanomas. We herein describe a case of metastatic high grade combined small cell nasopharyngeal NEC.

A 31 year old gentleman initially presented for evaluation of 1 month of progressively worsening right sided neck swelling, sinusitis, nausea and retro-orbital tension type headache. Past medical history included obstructive sleep apnea (OSA), prior traumatic brain injury (TBI) with subsequent amblyopia and seizure disorder. He had additional difficulty swallowing, limited to liquids. He had associated weight loss of 5lbs and voice change but denied night sweats. Examination revealed asymmetric, soft tissue swelling of the right neck. No otalgia or otorrhea present. Tympanic membranes were clear and mobile. Nasal examination revealed moist mucosa, occluded right nasal vault and significant left septal deviation. No tonsillar inflammation was appreciated. Cervical lymphadenopathy was present bilaterally. CT neck revealed extensive neck adenopathy and right sided sinus disease. Biopsy of lymph node identified metastatic high-grade neuroendocrine carcinoma positive for CD56 and granzyme B. PET CT revealed extensive involvement of nasopharynx and right tonsil as well as multiple bone metastases. Despite chemotherapy and radiation, our patient died 5 months after diagnosis.

## 72) RENAL FAILURE & RASH WITH EOSINOPHILIA: A CASE OF ACUTE INTERSTITIAL NEPHRITIS SECONDARY TO CEPHALEXIN

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**Introduction:** Acute Interstitial Nephritis (AIN) is characterized by reduced creatinine clearance and inflammatory changes of renal interstitium. Most often, the condition is precipitated by drug therapy but may also be associated with systemic autoimmune disease or infections. The vast majority of AIN results from treatment with beta-lactam antibiotics but other medications including non-steroidal anti-inflammatories, antimicrobial sulfonamides, diuretics, H-2 blockers, proton-pump inhibitors, xanthine oxidase inhibitors, 5-aminosalicylates, quinolones, and protease inhibitors have been implicated. Drug-induced AIN typically presents with a clinical triad of symptoms of fever, rash, and eosinophilia.

**Case:** A 68-year-old female presented with fevers, diffuse macular rash, oliguria, and generalized weakness following multiple antibiotic courses for treatment of combined lung and brain infections with microaerophilic streptococci species. Notably, she had initially been treated with penicillin and had developed marked eosinophilia and generalized rash. This was transitioned to vancomycin, which was discontinued after precipitating acute tubular necrosis (ATN). After this renal injury subsided, she was briefly treated with cephalexin for refractory lung abscess. Once again, she developed worsening renal failure and oliguria, now accompanied by signs of systemic allergy (fever, diffuse maculopapular desquamating rash, peripheral eosinophilia, and eosinophiluria). AIN was strongly suspected based on the clinical picture, so no renal biopsy was attained. Once the offending agent was removed and she completed a course of intravenous steroids followed by an oral steroid taper, renal function normalized. Dialysis was not required.

**Discussion:** AIN should be suspected in patients with elevated serum creatinine and urinalysis revealing pyuria, white cell casts, hematuria, and eosinophiluria; particularly in the setting of exposure to drugs known to cause AIN. Proteinuria is common but typically <1gm/day. Definitive diagnosis of AIN is made by renal biopsy. Our patient's diagnosis was made by clinical history, marked serum/urine eosinophilia, and microscopic urine evaluation. If improved renal function is not observed within 5 to 7 days of treatment, a biopsy should be attained to exclude other diagnosis or the presence of severe interstitial fibrosis. The mainstay of therapy is discontinuation of offending agent if drug-induced AIN is suspected. There are no RCT guidelines for therapy, however one option for treatment is immunosuppressive therapy with glucocorticoids is typically employed if renal function does not improve within 3 to 7 days. In our patient, after initiating steroids, her eosinophilia went from 31% to 0% in 1 day. Additionally, it took 5 days for her urine output to normalize (>1L UOP / 24hrs). Other case reports suggest by utilizing steroids for AIN, one can prevent the need for hemodialysis, which was the case for our patient.

### 73) HETEROPHILE NEGATIVE EBV-ASSOCIATED MONONUCLEOSIS

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**Introduction:** Epstein Barr virus (EBV) is the primary etiologic agent responsible for infectious mononucleosis. Approximately 90-95% of adults are EBV-seropositive, with the majority of primary infections being subclinical. EBV is associated with the development of B cell lymphomas, T cell lymphomas, Hodgkin lymphoma, and nasopharyngeal carcinomas and reactivation can precipitate lymphoproliferative disorders in patients on immunosuppressive therapy. Infectious mononucleosis initially begins with malaise, headache, and low-grade fever followed by pharyngitis and cervical lymph node enlargement. Peripheral blood demonstrates lymphocytosis, with significant portion of atypical lymphocytes.

**Case:** A 19-year-old male presented with a 1-month history of intermittent fevers and sore throat. He was initially seen by his PCP, who noted exudative pharyngitis. He was prescribed a course of amoxicillin-clavulanate with marginal response. Rapid strep test was negative. His symptoms persisted and investigatory labs were notable for reactive lymphocytosis on peripheral blood smear and a negative heterophile antibody test. Due to refractory symptoms and unclear etiology of complaints, he was evaluated by infectious disease consult. His fevers worsened and were accompanied by drenching night sweats. He was admitted for in-patient evaluation after being found to have progressive thrombocytopenia, transaminitis, elevated LDH, and inflammatory markers including ferritin, and neutropenia with monocytosis and lymphocytosis. Manual blood smear was notable for reactive lymphocytes and spherocytes but no schistocytes to suggest hemolysis. Direct antiglobulin testing showed complement. Presenting chest x-ray did not reveal any mediastinal mass and CT chest/abdomen/pelvis with contrast noted mild splenomegaly. CT neck with contrast demonstrated non-specific cervical lymphadenopathy. Flow immunophenotype panel showed no evidence of lymphoproliferative. Infectious labs were negative for infectious mononucleosis screen, HIV screen, tuberculosis quantiferon, acute hepatitis panel, EBV panel IgM/IgG, CMV NAAT, gonorrhea/chlamydia NAAT, group A strep NAAT, malaria smear, cat scratch antibody, babesia serology, toxoplasmosis serology, and extended viral respiratory NAAT. Prior to attaining lymph node biopsy of enlarged cervical nodes, EBV NAAT tallied >17,000 copies so procedure was aborted. Patient was discharged with monitoring of liver function tests and EBV titer.

**Discussion:** Primary EBV infections are frequently asymptomatic but may manifest with fever, painful pharyngitis, and cervical lymphadenopathy. Laboratory studies typically show reactive lymphocytosis, commonly being atypical, and abnormal liver function tests. In most cases, serology is positive. Our case represented a serology negative infection with positive EBV DNA. In cases where clinical suspicion is high and serology is negative, one may consider EBV DNA versus serial EBV serology.

### 74) TENDER SKIN MACULES: CASE OF EARLY CUTANEOUS POLYARTERITIS NODOSA

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**Introduction:** Polyarteritis nodosa (PAN) is a systemic necrotizing vasculitis that typically affects medium-sized muscular arteries with occasional involvement of small muscular arteries. Patients typically present with systemic complaints (fatigue, weight loss, weakness, fevers, myalgias) as well as signs of systemic involvement (skin lesions, hypertension, renal insufficiency, neurologic dysfunction, and abdominal pain). Cutaneous symptoms (subcutaneous nodules, livedo reticularis, ulcers, and gangrene) are observed in 25-60% of polyarteritis nodosa patient. Most cases are idiopathic, although some cases have been associated with hepatitis B, hepatitis C, and hairy cell leukoplakia.

**Case:** A 25-year-old African American female presented with a 1-month history of recurrent fevers, sweats, myalgias, arthralgias, and fatigue accompanied by the development of small, tender, indurated plaques occurring on both arms and legs (more marked in lower extremities). She had been treated for multiple episodes of mycoplasma pneumonia and had recently completed a course of doxycycline at the time of rash development. Skin biopsy of the aforementioned rash revealed evidence of medium vessel vasculitis consistent with PAN. Laboratory studies were notable only for mild leukocytosis, marginally elevated acute phase reactants and rheumatoid factor, and mild microcytic anemia but no evidence of renal or hepatic dysfunction. Hepatitis B & C panel, HIV, RPR, antinuclear antibodies, serum complement components C3 & C4, cryoglobulins, antineutrophil cytoplasmic antibodies, aldolase, and serum & urine immunofixation electrophoresis for monoclonal gammopathy were not detected. Iron panel was suggestive both of iron deficient anemia and anemia of chronic disease. Hemoglobinopathy panel was unremarkable. She also reported having abdominal discomfort so CT angiography of the abdomen and pelvis was attained, without evidence of microaneurysmal changes of renal, hepatic, or mesenteric circulations. She was treated with glucocorticoids for mild disease severity with isolated cutaneous involvement.

**Discussion:** The diagnosis of PAN is typically a clinical picture based on the presence of characteristic symptoms, physical findings, and compatible laboratory studies. Our patient lacked many typical diagnostic markers but her tender, purpuric, palpable skin lesions prompted the pursuit of a tissue biopsy, which ultimately clinched the diagnosis. Treatment is contingent on the level of disease of severity, degree of organ dysfunction, and/or the presence of concomitant viral hepatitis infection. Treatment regimens consist of steroid, immunomodulatory, or antiviral therapy when indicated. Patients with cutaneous involvement or mild PAN (constitutional symptoms, arthritis, anemia, but normal renal function and no gastrointestinal, cardiac, or neurologic manifestations) should be treated initially with oral glucocorticoid monotherapy. Despite recurrence over time, the prognosis of cutaneous PAN is favorable with low risk of progression to systemic illness.

## 75) A RELATIVELY RARE CAUSE OF BRADYCARDIA

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**Introduction:** Fever is linked with an increase in heart rate. Physiologically, the temperature-heart relationship is linear with an increase in heart rate of 10 beats per minute (bpm) for each one degree Fahrenheit (°F) rise in core temperature. As a corollary, a temperature of 102°F is expected to increase the heart rate to approximately 120 bpm. Relative bradycardia (RB) is a phenomenon in which fever does not lead to the aforementioned predictable increase in heart rate. In addition, RB may be an important diagnostic clue associated with intracellular gram-negative pathogens including *Salmonella*, *Legionella*, and *Chlamydia*. To our knowledge, we report the first case of RB associated with *Pasteurella multocida* bacteremia.

**Case:** A 56 year old man with a history of end-stage renal disease presented to the emergency department with a one day history of generalized weakness, fever of 102°F, and hypotension during routine outpatient dialysis. There were no sick contacts or recent travel. Family and social histories were remarkable for a pet cat at home. Review of systems was unremarkable. Admission blood pressure was 78/43 millimeters of mercury and heart rate was 70 bpm. Physical examination demonstrated an intact upper extremity arteriovenous fistula. Laboratory data was significant for a white blood cell count (WBC) of 25,100/ $\mu$ L and a serum lactic acid of 2.8 mmol/L. Chest radiograph was unremarkable. Blood cultures were obtained. He was admitted to the intensive care unit (ICU) where intravenous fluid resuscitation and empiric intravenous vancomycin and piperacillin-tazobactam were continued. Despite the hypotension and fever, the patient's heart rate remained below 80 bpm. On hospital day two, blood cultures were positive for *Pasteurella multocida*. Antibiotic regimen was tailored to intravenous penicillin G. The patient remained asymptomatic, hypotension resolved, and WBC decreased to 18,300/ $\mu$ L. The patient was transferred out of the ICU on hospital day two.

**Discussion:** We report a case of *Pasteurella multocida* associated RB. *Pasteurella multocida* can lead to respiratory tract, skin, or bloodstream infections. As a zoonotic organism the nidus of infection in humans is an animal bite or scratch, specifically domesticated cats in this case. In addition, non-infectious causes of RB need to be excluded. There was no evidence of beta-blocker or calcium channel blocker use. There were no signs or symptoms of CNS disorders, lymphoma, or new medications to suggest drug fever. The positive blood cultures for *Pasteurella multocida* coupled with RB suggest *Pasteurella multocida* induced RB. After excluding non-infectious and drug-related etiologies, RB can serve as a diagnostic tool for infectious pathogens.

## 76) OVARIAN VEIN THROMBOSIS, A RARE CAUSE OF ABDOMINAL PAIN IN A NON PREGNANT WOMAN

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**Learning Objectives:** Ovarian vein thrombosis is a rare condition, however associated with serious complications such as pulmonary embolism, sepsis and death.

Although it is commonly seen in postpartum period, in any female with lower quadrant abdominal pain, this diagnosis should be considered in the differentials.

**Case Report:** 29 year old previously healthy female, G4P4L4 presents to local emergency department with bilateral flank pain radiating to right groin of 1 week duration. She also had dysuria, urinary urgency and frequency. No fever or chills. She denied vaginal discharge, and uses depo provera for birth control. Her last pregnancy was over a year ago. She was otherwise well, with vitals of temp:98.2F, HR 71, BP 131/67, RR 17/min SpO2.

Physical exam revealed moderate suprapubic tenderness; bimanual exam revealed adnexal tenderness. Urinalysis, pregnancy test, CBC, BMP was within normal limits. CT abdomen/pelvis revealed thrombosis in the right gonadal vein.

Work up for possible malignancy such as CA 125 was negative. She was positive for heterozygous prothrombin gene mutation.

She had no signs and symptoms of infection and hence antibiotics were not started. Discussions with Hematologist and OB/Gynecologist were made. Patient was placed on oral anticoagulation for 3 months and improved symptomatically. Follow up imaging was considered not needed.

**Discussion:** Ovarian vein thrombosis in a non-pregnant woman is such a rare entity that there are only very few reported cases. There is no clear consensus about the duration of anticoagulation therapy or need for further imaging. Use of antibiotics has been reserved for patients with fever or endometritis. Surgical intervention is a preferred modality of those with free floating thrombus and recurrent pulmonary embolism or when anticoagulation is contraindicated.

## 77) AUTOIMMUNE THYROID DISEASE: A RARE PRESENTATION OF A COMMON CONDITION

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**Introduction:** Hashimoto encephalopathy (HE) is a rare, possibly underreported, autoimmune condition associated with Hashimoto thyroiditis (HT). The association is not well known, but HT is the most common cause of hypothyroidism making this case an imperative demonstration of recognizing a life-threatening and sometimes irreversible condition that can easily be mistaken for other common disorders.

**Case:** A 67 year old female was admitted for worsening altered mental status. She had a 3 week history of mild cognitive decline and complained of headaches. CT and MRI demonstrated no acute ischemia but cerebral atrophy and extensive hyperintense white matter changes were evident. Infectious etiology was ruled out. TSH was severely elevated at 258. Anti-thyroid peroxidase antibody (TPOAb) and antithyroglobulin antibody (TgAb) were both elevated at 3548 and 1858. Patient was started on levothyroxine but her mental status continued to worsen. EEG showed generalized slowing consistent with moderate diffuse encephalopathy. She developed myoclonus and somnolence. Hyperreflexia was present. Myxedema coma was considered, but she did not demonstrate signs of hypothermia, hypotension, bradycardia, or hypoglycemia. HE was considered and the patient was given IV methylprednisolone; within 24 hours her myoclonus and somnolence resolved, her mentation improved and she was able to converse again.

**Discussion:** This case emphasizes the challenge of diagnosing a rare condition in a patient presenting with altered mental status, a common diagnosis in emergency departments nationwide. This was particularly a difficult case because HE does not always present as hypothyroidism; thyroid status varies tremendously. This misled suspicion for other differentials such as severe HT and myxedema coma. Although HE is rare, thyroid dysfunction along with a similar clinical presentation to this patient should cause high suspicion for HE. Mild cognitive impairment persisted after treatment, but it is well documented that HE may take up to 1 year to resolve. In some cases, if left untreated, encephalopathy will not improve making recognition paramount for timely and effective treatment.

## 78) “EXPERIMENTAL” INGESTION OF CERBERIN

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**Introduction:** Cerberin is an active ingredient found in the seeds of the *Cerbera odollam* tree, also known as the “Suicide tree”. Cerberin mimics Digoxin and is commonly used as a suicide and homicide drug in South Asia. However, its use is relatively uncommon in the western world.

**Case:** A 25 year-old-male with a past medical history of depression and prior suicide attempts presented to the ED with confusion, lip numbness, nausea, vomiting, palpitations, and diarrhea. He admitted to ingesting a seed containing Cerberin which he purchased online. He reported experimenting with the seed in the event he was sentenced to a prison sentence. In the ED, he was found to have an irregular pulse with heart rate of 106 and blood pressure of 94/55. EKG revealed sinus tachycardia, Mobitz type I 2nd degree AV block, and T wave inversions in inferolateral leads. His labs were significant for a potassium of 5. His urine drug screen was positive for Cannabinoids. Toxicologist at Poison Control recommended Digibind, a Digoxin specific antibody. The patient’s overall condition remained stable. A repeat EKG 24 hours later was normal sinus rhythm.

**Discussion:** Cerberin ingestion is responsible for 50% of plant poisonings in South Asia. Since Cerberin poisoning is uncommon in the western world, its diagnosis presents a challenge to physicians. Cerberin and Digoxin are cardiac glycosides which inhibit the Na-K-ATPase pump in the myocardium. Overdose may present with a variety of systemic symptoms including nausea, vomiting, diarrhea and any dysrhythmias of which AV block is more commonly seen. Physicians must suspect Cerberin poisoning in patients with unexplained digoxin-overdose like presentation. Management includes appropriate administration of Digibind and monitoring on telemetry for any further arrhythmias.

## 79) A RARE PRESENTATION OF HIDRADENITIS SUPPURATIVA WITH SIMULTANEOUS MANIFESTATION OF MULTIPLE COMPLICATIONS

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**Introduction:** Hidradenitis Suppurativa (HS) is a well described inflammatory dermatological disease associated with a variety of systemic complications. We present a rare case of a patient with simultaneous presence of Hidradenitis Suppurativa, acne conglobata, rapid degenerative and erosive polyarthritis, synovitis, pyoderma gangreosum, and early spondyloarthropathy.

**Case:** A 35 year old male with HS presented with four weeks of left hand immobility and an ulcerating lesion on the left leg. On physical examination, the patient was found to have disfiguring scars in the R axilla, facial acne, severe deformities of the left hand including synovitis of the DIP, PIP, and MCP joints with mild ulnar deviation. There was also hyperpigmentation of hand joints and a large ulcerating lesion on the left leg. An initial workup was negative for ANA, HLA B27, RPR, and viral hepatitis. RF was negative but ESR and CRP were elevated. Imaging of the hand revealed synovitis, peri-articular erosions, myositis, and tenosynovitis. Spinal x-ray was consistent with degenerative changes at C5 & C6. A biopsy of the ulcerating lesion revealed histological findings consistent with pyoderma gangreosum. The patient was started on NSAIDs and low dose steroids before transfer to a different hospital with inpatient rheumatology and dermatology services. He was started on IV Solumedrol. It was anticipated that he would eventually be started on TNF alpha inhibitors.

**Discussion:** HS is a chronic inflammatory disorder. Its pathogenesis is unclear; however, the current understanding suggests follicular occlusion of the apocrine and sebaceous glands. Secondary complications include scarring, infection, reactive arthritis, pyoderma gangreosum, and squamous cell carcinoma. Diagnosis is mainly clinical. In recent years, TNF alpha inhibitors have been the mainstay of treatment. Given the severity of the disease, it is important for clinicians to recognize and treat the disease appropriately.

## 80) EPINEPHRINE INDUCED STEMI IN A YOUNG HEALTHY MALE

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**Introduction:** STEMI following administration of intramuscular epinephrine for the treatment of Anaphylaxis is a rare phenomenon. Although the true incidence of STEMI post epinephrine administration is unknown, this presentation is relatively uncommon.

**Case:** A 20-year-old healthy male patient with no past medical history presented to the Emergency Department with an allergic reaction following ingestion of seafood. He reported throat “itchiness and tightness.” At home, he took Benadryl without improvement in his symptoms. Soon after arriving to the ED, he was treated with intramuscular epinephrine 0.3mg which was given in the right anteromedial thigh. Shortly after, he developed flushing, diaphoresis and crushing sub-sternal chest pain. His vitals were significant for a blood pressure of 215/95, pulse of 92/minute, and respiratory rate of 33/minute. EKG demonstrated anterolateral ST segment elevations and reciprocal ST segment depressions in inferior leads. Troponin I was initially elevated at 0.06 and peaked at 1.06. He received aspirin, 1mg/kg enoxaparin and nitroglycerin paste. His chest pain resolved after these medications. An echocardiogram showed a normal ejection fraction without any wall motion abnormalities.

**Discussion:** STEMI following an anaphylactic reaction is uncommon but has been reported and is known as Kounis Syndrome. However, STEMI caused by epinephrine is a very rare phenomenon, and only a few case reports have been reported sporadically in the literature. Epinephrine is the mainstay of treatment for an anaphylactic reaction and should be administered intramuscularly into the lateral thigh to decrease the risk of accidental intravenous administration. We hypothesize that in our case, since the epinephrine was given in the anteromedial thigh instead of the lateral thigh, the medication likely entered a vein inadvertently causing coronary artery spasm mimicking STEMI.

## 81) RIGHT SIDED HEMIPARESIS IN A 26 YEAR OLD WOMAN

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**Introduction:** Behcet's syndrome is a unique immune mediated systemic vasculitis that can affect variably sized vessels in both venous and arterial circulation. Though Behcet's classically presents with genital ulcers, oral ulcers, and uveitis, it may also present with less common symptoms due to infiltration of a wide variety of organs. Given increased morbidity associated with involvement of the GI tract, pulmonary vasculature, and central nervous system, diagnosis and treatment of the less common sequelae of Behcet's deserves attention in the differential diagnosis.

**Case:** A 26 year old woman with past medical history of Behcet's syndrome presented with symptoms of headache and progressive right sided weakness. 4 days prior to presentation symptoms began with throbbing headache, nausea and vomiting with progression to upper and lower extremity weakness. She was unable to ambulate on 4th day after onset of symptoms, leading her to seek medical care. Physical exam revealed dense right sided hemiparesis with intact sensation. Non contrast head CT revealed focal area of low attenuation within left anterior pons. Given concern for infarct versus Behcet's, follow up CT head venogram with IV contrast, MR brain with and without contrast, and MRA neck with and without contrast were obtained. Imaging revealed enhancement and edema in the left paramedian brainstem, cerebral peduncle and thalamus. Venogram did not reveal dural venous sinus thrombosis, or thrombosis of internal cerebral veins or superior sagittal sinuses. Given history, physical exam, and imaging findings, our patient was diagnosed with parenchymal neurologic Behcet's disease. She was treated with three days of high dose methylprednisolone. Her hospital course was complicated by anxiety and tremulousness beginning shortly after admission. By hospital day 3, she began to experience improvement of motor deficits and mood disturbance. She was discharged on hospital day 4 in stable condition on an oral prednisone taper. Ultimately her symptoms recurred, and were treated successfully with another course of IV steroids followed by an oral taper.

**Discussion:** Though Behcet's is a relatively rare syndrome, complications of untreated disease can be serious. Neurologic Behcet's is classified as parenchymal and non parenchymal, both are seen in less than 10 percent of individuals with Behcet's syndrome. Untreated parenchymal Behcet's can have a waxing and waning course, or progressive decline leading to brainstem atrophy and increased debility or death. Given potential for severe neurologic compromise, early, aggressive treatment is warranted in those with neurologic complications of Behcet's. Treatment modalities for neurologic Behcet's may include glucocorticoids, azathioprine, cyclophosphamide, TNF alpha inhibitor therapy, or interferon alpha.

## 82) THE GREAT PRETENDER: MORE THAN MEETS THE EYE

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**Introduction:** Syphilis has emerged in the last decade as a formidable infectious disease in the United States, with the rates of primary and secondary syphilis doubling since the turn of the century. Ocular manifestations of syphilis present less commonly, though the true epidemiology is unclear. We present a rare case of ocular syphilis presenting as unilateral blurriness.

**Case:** A 29-year old previously healthy bisexual African-American male presented with a three-week history of right eye blurriness. Ophthalmology was consulted and performed a dilated fundus exam that revealed a right eye with severe vitritis, panuveitis and retinal vasculitis and a left eye with mild vitritis. Snellen vision of R 20/300 and L 20/30 was noted. Extensive workup to evaluate for Lyme disease, sarcoidosis, tuberculosis, syphilis, toxoplasmosis, and rheumatic disease was performed. Workup revealed a positive serum treponemal antibody testing (FTA-Abs) and RPR (1:256). Patient was admitted for treatment with aqueous crystalline penicillin-G 3 million units every 4 hours for a 14-day duration. A lumbar puncture was performed and cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis (97%), leukocytosis (22), and elevated protein (54), all highly characteristic findings of neurosyphilis. CSF VDRL studies were negative, however. Upon examination, patient had multiple genital chancres along the base of his glans penis. Skin examination revealed tan-brown macules and patches along the right palm and the plantar surfaces of his feet bilaterally. Neurological exam was unremarkable, other than the noted visual deficits. Subsequent workup for HIV was positive with a CD4 count of 255. With treatment, patient reported gradual improvement in right eye vision and improvement in vitreous haze on ophthalmic exam. He was referred to a local HIV/AIDS clinic for continued therapy.

**Discussion:** Ocular syphilis, an insidious but potentially sight-threatening illness, has been of special concern for the CDC which has seen an alarming rise in the number of cases over the past two years. The rising rates of primary and secondary syphilis may be largely attributed to unprotected sex in a time period of major advances in HIV treatment. The CDC has issued a clinical advisory regarding the increasing incidence of ocular syphilis, especially in the men who have sex with men population. The condition most commonly presents as posterior uveitis or panuveitis, two conditions with many infectious, autoimmune, and rheumatic mimickers. Clinicians should have a high index of suspicion for a syphilitic etiology in patients presenting with the characteristic ocular complaints and a suggestive social history.

### 83) AN UNCOMMON CULPRIT OF RHABDOMYOLYSIS

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**Introduction:** Rhabdomyolysis manifests as myalgia and weakness with severely elevated serum levels of muscle enzymes, and can become a life-threatening disorder when complicated by acute renal failure. While myopathy is not unusual in hypothyroidism, this case demonstrates how hypothyroidism, although rare, can also lead to rhabdomyolysis.

**Case:** A 32-year-old male with a past medical history of Graves' disease status-post radioablative iodine therapy presented with a 1 month history of fatigue and worsening generalized myalgia. Of note, the patient had undergone radioablative iodine therapy 6 months prior to presentation, but had failed to follow-up with endocrinology for thyroid function testing and further management. Review of systems revealed additional complaints of a 7 lb. weight gain, cold intolerance, tea-colored urine, and constipation. The patient denied any recent history of trauma, prolonged immobilization, or marked exertion. The patient reported use of acetaminophen/oxycodone and smoking marijuana without any pain relief. No other medication use was reported. The patient was hemodynamically stable on presentation with exam significant for dry mucus membranes, thyromegaly, hypoactive bowel sounds, and generalized muscle pain. Initial labs revealed mild normocytic anemia, acute kidney injury (Cr = 1.34), elevated creatine kinase (11821), elevated thyroid stimulating hormone (88.59), low thyroxine (0.1), and normal random cortisol. Urine studies were notable for the presence of blood without RBCs, and a drug screen positive for marijuana, but negative for cocaine. EKG revealed normal sinus rhythm. The patient was diagnosed with rhabdomyolysis secondary to iatrogenic hypothyroidism. Rhabdomyolysis was treated with aggressive IV fluids and acetaminophen. Endocrinology was consulted and the patient was started on levothyroxine as well. The patient's symptoms and labs improved over several days and he was discharged home on levothyroxine with endocrinology follow-up.

**Discussion:** Common causes of rhabdomyolysis include trauma, prolonged immobilization, marked exertion, and exogenous toxins or medications. However, in evaluating a patient with rhabdomyolysis, it is important to conduct a thorough history and physical so that less common etiologies are considered as well. The exact mechanism behind hypothyroid-induced rhabdomyolysis remains unclear, however, it likely involves a combination of hypoperfusion and impaired mitochondrial activity resulting in muscle necrosis and the accumulation of nephrotoxic muscle enzymes. Thyroid hormone replacement therapy improves thyroid function and, in addition to aggressive IV fluid replacement, reverses rhabdomyolysis.

### 84) VASCULITIS- NOT ALWAYS WHAT IT SEEMS

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**Introduction:** The most common causes of vasculitis are the primary vasculitides. However, there are many secondary causes of vasculitis, including drug reactions, autoimmune, neoplastic, and infectious processes. Despite the best efforts of clinicians, these secondary causes of vasculitis may not become apparent until well into the course of illness.

**Case Report:** A 70 year-old male was transferred with a 3 week history of progressive purpuric rash, acute renal failure, anemia, and thrombocytopenia. He had recently begun venlafaxine for four months of depressed mood, fatigue, anhedonia, and a 20-pound weight loss. Diagnostic testing prior to transfer was notable for monoclonal gammopathy of undetermined significance, low-level cryoglobulinemia, and skin biopsies consistent with a leukocytoclastic vasculitis. In the three days immediately following transfer, the patient's rash worsened, spreading from his legs across his entire body. He also had persistent tachycardia, a 2/6 systolic murmur at the left sternal border, and diffuse arthralgias. Diagnostic considerations included cryoglobulinemic vasculitis versus a primary vasculitis; the patient was deemed unstable for renal biopsy, so after subspecialty consultation empiric corticosteroid therapy was initiated. Thirty-six hours later, the patient developed severe metabolic acidosis, acute delirium, and finally respiratory failure requiring ICU transfer and endotracheal intubation. Blood cultures drawn shortly after ICU transfer grew viridans Streptococci, and trans-esophageal echocardiography demonstrated valvular vegetations. Infectious endocarditis was diagnosed, and in retrospect, subacute bacterial endocarditis was believed to be the driver of his entire course.

**Discussion:** Multiple conclusions may be drawn from this case. In retrospect, some degree of anchoring bias at multiple levels may have led to focus on either a primary vasculitis or one of the already-identified issues as a driver of the patient's presentation rather than all potential causes. Next, infectious endocarditis can be a difficult-to-recognize condition with a diverse array of clinical presentations. Finally, blood cultures should be considered as part of the initial evaluation for a new-onset vasculitis, possibly even in the absence of specific infectious symptoms.

## 85) TO TEST OR NOT TO TEST (URINARY ANTIGEN): LEGIONELLA PNEUMONIA AND MIMICKING ACUTE CORONARY SYNDROME

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**Introduction:** In multiple epidemiologic studies, *Legionella pneumophila* has been shown to be a common causative organism of community acquired pneumonia (CAP) but may be under diagnosed due to non-specific symptoms and lack of sufficiently sensitive testing. At the same time, routine testing for *Legionella* in every patient with suspected pneumonia is expensive and may not change treatment in patients who are already receiving guideline-based antibiotic coverage for CAP.

**Case Report:** Our patient was a 61 year old woman with an extensive cardiovascular disease history, including coronary artery disease with multiple percutaneous interventions, peripheral artery disease, carotid stenosis, and ischemic stroke. She presented in transfer from a referring hospital with chest pain. Given her history, the initial concern was for acute coronary syndrome, so she was admitted to the cardiology service for further evaluation. On admission, her troponin was elevated to 0.09, seemingly confirming a diagnosis of primary non-ST elevation myocardial infarction in the absence of other symptoms. However, prior to a planned cardiac catheterization, she became febrile to a temperature of 103.1F. A broad infectious work-up, including legionella urinary antigen, was ordered. The urinary antigen was positive. In retrospect, she had multiple non-specific features consistent with *Legionella* pneumonia including general malaise, gastrointestinal symptoms, and mild hyponatremia. Her chest pain was attributed to pneumonia, and myocardial ischemia was deemed due to demand. She was treated with levofloxacin and made a full recovery, leaving the hospital four days later.

**Discussion:** *Legionella* pneumonia is an important cause of community-acquired pneumonia. It is important to balance the overall cost of unnecessary testing with maintaining a high clinical suspicion in patients for whom a positive legionella urinary antigen would change management.

## 86) BACTRIM-INDUCED ASEPTIC MENINGITIS

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**Case:** A 48-year-old male with HIV-1 presented with a headache and neck stiffness for 6 days. He reported photophobia and diffuse myalgia and arthralgia. Temperature was 100.1 F. On exam, he had nuchal rigidity. Labs showed WBC of 6000 and lumbar puncture revealed WBC of 11 with predominantly lymphocytes. MRI of the head showed non-specific inflammatory changes. Patient was started on IV acyclovir, ceftriaxone, and vancomycin.

His most recent HIV-1 viral load was 80,000 and CD4 count was 58. He had recently started his HAART regimen, along with initiation of Bactrim and Azithromycin for prophylaxis.

Infectious studies for meningitis were negative and by day 3 and the diagnosis of Bactrim-induced aseptic meningitis (BIAM) was made. His Bactrim was discontinued and he report resolution of symptoms 3 days later.

**Discussion:** The etiology of aseptic meningitis is viral, fungal, mycobacterial, or drug-induced, although enterovirus is the most common cause. Unlike most cases of aseptic meningitis which resolve spontaneously, BIAM resolves after withdrawal of Bactrim.

Other cases of BIAM, that include patients with HIV, have followed similar courses with similar CSF composition. Our patient's symptoms began to improve 24 hours after Bactrim discontinuation, which is consistent with other reports of BIAM, and also reflects the pharmacokinetics of Bactrim.

The best way to confirm this diagnosis is to perform a drug challenge with Bactrim. This diagnosis poses a dilemma for physicians treating persons with HIV/AIDS prophylactically for pneumocystis pneumonia and *Toxoplasma gondii*, as Bactrim is the first-line agent that offers coverage for both organisms.

The purpose of this report is to alert physicians to the possibility of BIAM. Furthermore, it is our hope to reduce harm to patients by assisting physicians in identifying BIAM upon initial presentation without the patient having repeated exposure to Bactrim before arriving at the proper diagnosis. Here we reported a case of clinically diagnosed BIAM in a 48-year-old male with HIV/AIDS.

## 87) VANCOMYCIN INDUCED LINEAR IGA BULLOUS DERMATOSIS (VILAD)

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**Patient:** 86 year-old male with 4 day history of rash

**Learning Objectives:** 1) recognize linear IgA bullous dermatosis (LAD) as an eruption associated with vancomycin i.e. VILAD 2) learn about diagnosis/management of LAD.

**Case Description:** Patient presented after recent knee revision for prosthetic joint infection with vancomycin/tobramycin antibiotic spacer placement + IV vancomycin. Vesicular peri-incisional rash began 9 days later progressing to diffuse urticaria 2 days later with continued progression despite stopping IV vancomycin. Knee pain and rash only complaints. On exam, vitals stable, + yellow peri-incisional crusting, tense serous/hemorrhagic bullae, annular erythematous urticarial lesions with perilesional vesicles, palatal ulcers, periorbital erythema and conjunctivitis. Labs showed leukocytosis without bandemia/eosinophilia and normal transaminases. Dermatology consultation led to tissue biopsy + direct immunofluorescence (DIF) confirming the diagnosis of LAD. Vancomycin/tobramycin spacer left in as risk of losing ability to ambulate outweighed benefit of removal. LAD treated with colchicine due to patient's comorbidities. He made a full recovery after 2 weeks of therapy.

**Discussion:** LAD is a rare immune-mediated mucocutaneous blistering disorder. Causes are idiopathic, drug induced or infectious. Incidence globally ~ 0.5 – 2.3 cases/million/year. Mean onset between 3-4 and 52 years old. Clinical findings heterogenous but often see tense bullae, mucocutaneous eruptions, extensor involvement and perilesional vesicles. DIF is critical for diagnosis. Vancomycin is the commonest drug cause. While drug induced cases typically resolve in weeks with drug cessation, treatments in severe / non-drug cases include dapsons, sulfonamides, topical steroids, colchicine, or systemic steroids or IVIG. His case adds to the literature, because 1) the literature on VILAD is small 2) further support of the role of renal insufficiency in precipitating the condition 3) resolution despite leaving the vancomycin-containing spacer and using colchicine, a fourth line agent.

**Conclusion:** LAD is a rare, but important disease to recognize as a mucocutaneous bullous eruption associated with common medications requiring unique diagnostic and treatment modalities.

## 88) DYING OF A BROKEN HEART: CARIOGENIC SHOCK IN TAKOTSUBO CARDIOMYOPATHY

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**Introduction:** Takotsubo Cardiomyopathy, or stress induced cardiomyopathy, is a syndrome that usually resolves with supportive therapy, but recognizing and understanding the pathophysiology of this disorder can help prevent cardiogenic shock.

**Case Presentation:** A 43 year old female with a PMH of HTN presents with dyspnea and chest pain, after her father's death earlier that day. She was at her parent's home and found her father collapsed dead on the floor. An hour later, she began to develop symptoms. On presentation, she endorsed 6/10 non-radiating substernal chest pressure. VS were notable for a HR of 110 and a BP of 199/145. Troponin was 1.23, CXR revealed pulmonary edema. EKG showed small amount of ST depression. A rapid bedside echocardiogram was performed which showed global hypokinesis with preserved apical regional function consistent with reverse takotsubo cardiomyopathy. Patient improved with gentle diuresis. Cardiac cath the following day showed no evidence of CAD and the patient was discharged on a B-blocker and ACE-I.

**Discussion:** The pathophysiology of stress induced cardiomyopathy is not completely understood, but one theory is that adrenergic surge from a stressful event can cause over stimulation of the beta receptors of the heart leading to myocardial stunning. In younger individuals like the patient above, these receptors tend to be more highly concentrated in the base of the heart, leading to intact apical function with akinesis of the base. This is known as the reverse takotsubo pattern. Stress induced cardiomyopathy primarily occurs in older women however, where beta receptors are primarily concentrated in the apex of the heart. As a result, the apex of the heart becomes akinetic with a hyperkinetic base. This more frequent presentation is the typical pattern of takotsubo. Unfortunately, the hyperkinetic base can act as a left ventricular outflow tract obstruction in patients who don't have a high enough pre-load. This is likely why up to 10% of patients who develop takotsubo progress into cardiogenic shock even in the absence of a severely reduced EF. Therefore, it is imperative to suspect takotsubo cardiomyopathy early and obtain proper diagnostic imaging to prevent over diuresis which could lead to cardiogenic shock.

## 89) CRYPTOCOCCUS MENINGITIS IN AN IMMUNOSUPPRESSED PATIENT

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**Introduction:** *Cryptococcus neoformans* is a known cause of fungal meningitis. Non HIV infected patients on immunosuppressive drugs make up approximately 20% of cases in medically developed countries. Onset can be subtle and treatment complicated by the need to reduce immunosuppressive drugs for treatment of active infections.

**Case:** A 27 year old female with history of Systemic Lupus Erythematosus (SLE) on immunosuppressant therapy (prednisone, cellcept and plaquenil) presented with a 5-day history of unrelenting headache. Associated symptoms included phonophobia, photophobia, and 1 day of nausea and vomiting. Additional past medical history includes migraines, lupus anticoagulant, and remote history of cavernous sinus thrombosis. On exam, she had no focal neurological findings however Kernig sign was positive. CT, MRI, and MRV head were unremarkable. Lumbar puncture and CSF studies revealed an elevated WBC, normal glucose and protein, and a positive cryptococcal antigen of 16. Serum cryptococcal antigen was positive at 16. CSF culture isolated *C. neoformans* at day six. Chest x-ray and CT scan also demonstrated new ill-defined lower lobe opacities consistent with cryptococcal pneumonia. Initial treatment consisted of liposomal amphotericin B and flucytosine. The patient's cellcept was held to minimize immunosuppression. Following discharge and completion of four weeks of treatment, she was transitioned to oral fluconazole. Her treatment course was suddenly complicated by respiratory distress and she was admitted to the ICU. Repeat CSF cryptococcal antigen was negative, but serum cryptococcal antigen remained at 16. The mild CSF pleocytosis had resolved and both glucose and protein remained within normal limits. Cultures were negative for *Cryptococcus*. She rapidly developed worsening pulmonary infiltrates and thrombotic thrombocytopenic purpura complicated by multiple right MCA strokes which ultimately led to her death.

**Discussion:** *C. neoformans* is a major opportunistic pathogen worldwide that is found in soil, decaying wood, and bird droppings. Susceptible hosts are infected after inhalation. Clinical presentation is variable and patients may present acutely or after several months of symptoms such as headache, fever, and lethargy. Immunosuppressants, such as this patient's SLE regimen, increase the risk for cryptococcal infection. Treatment consists of antifungal induction, consolidation, and maintenance therapy with amphotericin B, flucytosine, and fluconazole along with careful reduction in immunosuppression. Treatment response can be monitored with antigen assay, repeat lumbar puncture, and culture. Clinicians must always consider fungal causes of CNS disease in immunosuppressed persons even when the corresponding CSF abnormalities are mild.

## 90) NOT YOUR EVERYDAY ASTHMA: DIFFERENTIATING PRIMARY PULMONARY HYPERTENSION FROM EISENMENGER SYNDROME

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**Introduction:** Pulmonary hypertension (PH) is a serious, progressive disease with a high mortality rate if untreated. It typically presents with non-specific symptoms and can often be missed without a low threshold to screen for it. This delay in diagnosis can lead to devastating outcomes for patients.

**Case:** This is the case of a 23 year old female with a history of asthma, hypertension, and obesity, who presented to the ED with a 3 day history of worsening cough, dyspnea, and chest tightness. On arrival, she was hypoxic with oxygen saturations in the low 80's. She was admitted to the medicine service for what was thought to be her fourth asthma exacerbation in the past year and started on treatment with steroids, antibiotics, and duonebs. However, her symptoms failed to improve. A chest CT was done given her persistent hypoxia and recent right leg swelling, and showed no pulmonary embolism, however did identify significant enlargement of her main pulmonary artery and right ventricle. A transthoracic echocardiogram then showed a moderately dilated RA, markedly dilated RV with severely reduced function, pulmonary artery peak pressure of 74 mmHg, and a large right to left shunt across the atrial septum. Given this, the main concern was for either primary PH vs Eisenmenger Syndrome. She underwent a transesophageal echocardiogram that showed a small PFO with bidirectional shunting. Right heart catheterization then confirmed the PFO, however did not identify any significant shunt. Given this, it was felt that her elevated PA pressures and right heart failure were secondary to primary PH as opposed Eisenmenger syndrome, and that her large right to left shunt was secondary to an incidental PFO that opened due to her high right heart pressures. She was started on treatment with sildenafil and discharged home with close follow-up with pulmonology.

**Discussion:** This case illustrates the importance of recognizing when a patient's clinical picture does not fit with their current diagnosis, and taking the next step to further investigate it. This ultimately lead to the diagnosis of PH in this patient, which had been missed during her previous admissions. This case also shows that it can be difficult to differentiate primary PH from Eisenmenger syndrome, especially in the setting of an ASD or PFO. However, deciphering between these two diagnoses is critical and can have a significant impact on patient management.

## 91) AN UNUSUAL CASE OF SQUAMOUS CELL CARCINOMA OF THE SKIN

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**Introduction:** Squamous cell carcinoma (SCC) of the skin is one of the most common skin cancers. Early detection is common, and 5 year survival with therapy is over 90%. We present an unusual case of SCC of the skin presenting as large back mass with local and distant metastases.

**Case:** 60-year-old African American male presented following a fall. Interestingly, he was noted to have a mass in the mid back and the right axilla. He endorsed weight loss, fever and fatigue. He was diagnosed with SCC at an outside facility 4 months earlier, and underwent staging and evaluation but was lost to follow up. Physical examination revealed a large fungating mass over the T11 vertebrae with surrounding hyperpigmentation. Prominent axillary, and to a lesser degree supraclavicular and cervical lymphadenopathy was present. Laboratory analysis showed severe microcytic anemia and mildly elevated liver enzymes. Imaging revealed bilateral pleural effusions, numerous non-cystic focal hepatic lesions, intra-abdominal and pelvic lymphadenopathy and a lytic lesion of T11 vertebra. Biopsy of pelvic lymph node showed poorly differentiated metastatic SCC. Oncology consultation recommended chemotherapy despite expected poor prognosis. Unfortunately, the patient left against medical advice.

**Discussion:** Cutaneous SCC is the second most common type of skin cancer in the United States. The primary risk factor is ultraviolet light exposure. SCC on non-sun-exposed skin is less common, but represents the most common distribution in individuals with dark skin. SCC on shoulder and back account for less than 4% of the cases. Only 2-5% of SCC has regional lymphadenopathy or distant metastases. This case illustrates the challenges of managing patients who present with late stage SCC of the skin, typically an otherwise treatable form of cancer.

## 92) CASE OF 'NON-RESPONDING' DVT

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**Introduction:** Deep venous thrombosis (DVT) is a common condition encountered in medical practice. We present a case of DVT that seemed to progress despite appropriate pharmacologic therapy.

**Case:** A 70 year-old female presented to an outside hospital with a chief complaint of left leg swelling of 3 days duration. She has history of bilateral leg swelling due to chronic venous insufficiency. Lower limb venous ultrasound (US) revealed a left lower extremity (LLE) DVT. She was otherwise stable and was discharged the next day with therapeutic Lovenox for 5 days in addition to warfarin. Despite continuation of therapy she noted an increase in the size of her swelling and presented to our hospital a week after her initial diagnosis. Exam showed a grossly swollen LLE. Repeat US confirmed DVT with proximal extension and a high clot burden. She was immediately started on a heparin infusion and vascular surgery consult was obtained. The decision was made to proceed with catheter directed thrombolysis (CDT) with tPA. After therapy was complete, an angiogram was obtained showing complete lysis of the clot. May-Thurner syndrome was diagnosed, and she underwent stenting of her left iliac vein and was discharged home on warfarin at therapeutic INR. Several months later a follow up US showed patent veins.

**Discussion:** May-Thurner abnormality is a hemodynamically significant compression of the left common iliac vein between the overlying right common iliac artery and the underlying vertebral body. It is a common anatomic pattern in normal subjects, and is a cause of DVT in a small fraction of patients. It should be suspected in patient presenting with LLE DVT, especially with high clot burden. Therapy involves anticoagulation and may require additional therapy such as CDT and surgical or percutaneous interventions.

### 93) METHOTREXATE INDUCED PANCYTOPENIA IN RHEUMATOID ARTHRITIS TREATMENT COMPLICATED BY IATROGENIC FEVER

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### 94) THROMBUS IN TRANSIT THROUGH PFO: SUCCESSFUL NON-OPERATIVE TREATMENT IN THE SETTING OF ACUTE ISCHEMIC STROKE

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**Introduction:** Methotrexate (MTX) is a commonly used medication for a multitude of uses from rheumatoid arthritis and psoriasis to various solid tumors and leukemia. MTX has been being used safely for decades with well characterized side effects, which commonly cause isolated elevated liver enzymes, nausea/vomiting/diarrhea, anemia, leucopenia, and very rarely serious complications of pancytopenia, hepatotoxicity, and interstitial pneumonitis.

**Case:** A 71 year old AA female with past medical history of rheumatoid arthritis treated with 20mg MTX weekly for the past 32 years, chronic seizure disorder treated with lamotrigine, and hypertension presented with 2-3 days of painful mouth ulcers, fever, chills, and spontaneous epistaxis for 3-4 days. Her exam showed fever 101.2F, bilateral buccal mucosal ulcers, no petechiae, and no bruising. Lab studies were significant for pancytopenia with platelets at 8,000, WBC of 1800, ANC 420, hemoglobin at 10.3g/dL and methotrexate level was undetectable. For treatment, her methotrexate and lamotrigine were discontinued. Despite IV Cefepime and Vancomycin she continued to have fevers above 100.4F by day 2 and her WBC further dropped to 1200 with ANC of 200. An infectious and malignant work-up was conducted with normal viral titers and without any abnormalities in flow cytometry concerning malignancy. Filgrastim (G-CSF) was started and the neutropenia resolved by day 6 and was discontinued on day 7, however the patient became febrile measuring between 100.6-103.0F without any clear source. Day 8 the patient became afebrile, antibiotics were discontinued, and discharged on day 9 after being asymptomatic and afebrile for over 36 hours with outpatient follow up.

**Discussion:** The diagnosis of MTX induced pancytopenia is made on a clinical basis after other organic causes have been evaluated without definitive findings. The key pieces of evidence were the negative infectious work-up and negative flow cytometry study. MTX has a long history of causing mouth ulcers and pancytopenia, with cessation of MTX and supportive therapy with filgrastim all cell lines should improve back to baseline within 1-2 weeks. The patient's course was complicated by fever at admission and after resolution of neutropenia that was most likely iatrogenic in origin, between drug side effect and drug fever. Overall the treatment of pancytopenia from MTX toxicity is primarily ruling out more troublesome etiologies, providing a supportive role treating symptoms, preventing infection when susceptible, and awaiting the recovery of cell counts while averting further problems caused by the hospital or treatment itself.

A 59 year old female with hypertension and obesity presented with a one month history of worsening dyspnea on exertion and the acute onset of vertigo. She presented with normal vital signs and horizontal nystagmus. Her medications were limited to Metoprolol and Lisinopril. A non-contrast head CT demonstrated an acute ischemic infarction of the medial aspect of the left cerebellum. The patient was admitted for further evaluation to the inpatient stroke unit and promptly started on aspirin and high intensity statin.

Soon after admission, the patient was noted to be hypoxic with an oxygen saturation of 85% breathing room air. The remainder of her vital signs remained within normal limits. Her chest x-ray was free of any identifiable pathology. A d-dimer was notably elevated at 2950 ng/mL. A CT pulmonary angiogram was performed and demonstrated multiple pulmonary emboli within the subsegmental branches of the left upper and lower lobes as well as the bifurcation of the right main pulmonary artery. In addition to the presence of bilateral pulmonary emboli, a large left atrial thrombus approximately 4.5 cm in length was found to be extending from the intra-atrial septum towards the area of the mitral valve. Transthoracic echocardiography (TTE) was then performed which again demonstrated the left atrial thrombus but also revealed an aneurysmal appearing intraatrial septum with extension of the left atrial thrombus into the right atrium through a patent foramen ovale. The left atrial portion appeared to prolapse into the left ventricle during diastole.

It was determined, in the setting of acute ischemic stroke of this location, the patient was at too high of a risk for hemorrhagic conversion to allow for urgent cardiac surgery. Unfractionated heparin was continued and remarkably, a repeat TTE 2 days later showed both right and left atria clear of any identifiable thrombus. The patient had no evidence of symptomatic embolization identifiable on repeat physical examination.

Paradoxical embolism through an intracardiac shunt is a well described cause of acute ischemic stroke. However, the demonstration of thrombus traversing an intracardiac shunt is very rare and only described in a handful of case reports over the last 20 years. Management often includes operative removal of the thrombus. However, in the setting of acute ischemic stroke, the risk of hemorrhagic conversion with the high doses of UFH that are involved is excessive. This case report describes successful treatment with UFH in a high risk patient presenting with a large intracardiac thrombus.

## 95) CRYSTALLINE INDUCED CHRONIC KIDNEY DISEASE

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**Introduction:** Hyperoxaluria, a risk factor for crystal induced kidney disease, can be primarily due to genetic disorders or secondary, often due to malabsorption.

**Case:** This is the case of a 64 year old man with a history of cholangiocarcinoma status post Roux-en-Y hepaticojejunostomy surgery performed in 2003, type 2 diabetes mellitus, essential hypertension and chronic pancreatitis who presented for evaluation to the general medicine inpatient service after incidental discovery of a serum creatinine value of 4.1 mg/dL, compared to a normal value obtained 5 months ago. Hypertension and diabetes mellitus were well controlled. There was no evidence of oliguria, hematuria, edema, or uremia. Renal biopsy demonstrated severe chronic active interstitial nephritis and oxalate crystals consistent with oxalate nephropathy. A 24-hour urine collection showed elevated oxalate excretion with low calcium and citrate excretion. Serum oxalate levels were elevated (11 umol/L). He was started on a low oxalate and low fat diet, pancreatic enzymes, and potassium citrate. He was given intravenous fluids and underwent 4 sessions of hemodialysis.

**Discussion:** Enteric hyperoxaluria occurs in about 5-24% in patients with malabsorption. The prevalence of oxalate nephropathy, however, is not well defined. In enteric hyperoxaluria, calcium binds non-absorbed fatty acids instead of oxalate, which allows the colon to absorb oxalate in higher concentrations. The kidneys excrete oxalate and intratubular precipitation of oxalate crystals can cause obstruction and inflammation. While this patient had other risk factors for chronic kidney disease (DM2, HTN), the diagnosis was made by pathology. The goals of treatment are to decrease the amount of dietary oxalate, increase oxalate precipitation in the GI tract by increasing calcium available to bind oxalate, and prevent oxalate precipitation in the kidney. Hemodialysis, although not an approved treatment, can help to lower serum oxalate levels.

## 96) A HEART POUNDING CASE OF HEART FAILURE

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**Introduction:** Heart failure is a common symptom in the United States, affecting 5.1 million adults in 2013. A known complication of pheochromocytomas is cardiovascular pathology, and includes ischemic heart disease, arrhythmias, and myocardial infarctions. In a small sub-segment of patients, heart failure is an observed sequela, and is present in only 2 - 8 per 1 million adults of the general population in the United States.

**Case Presentation:** A 57 year old male presented with recent onset of progressive chest tightness, shortness of breath, lower leg swelling, and 3 pillow orthopnea. Additional history revealed that he had experienced at least 2 years of paroxysmal rage, mood swings, and palpitations. Vitals on presentation were notable for mild tachycardia with an irregularly irregular rhythm, hypertension, and 12 pound weight gain. Physical exam demonstrated 4cm of jugular venous distention at 30 degrees, bilateral crackles on lung auscultation, and 2+ pitting edema bilaterally to the knees. His urine normetanephrine was 6818 pg/mL; N-Terminal Pro BNP was elevated to 3019 pg/mL. His thoracic echocardiogram noted concentric left ventricular hypertrophy with preserved left ventricular ejection fraction. CT abdomen showed a 5 cm right adrenal mass. He required surgical mass resection, and pathology confirmed a pheochromocytoma.

**Discussion:** Heart failure is a known complication of pheochromocytoma, and is found in 3.4% of these patients. Typically, patients present with reduced ejection fraction secondary to a dilated cardiomyopathy, but some patients like ours can present with preserved ejection fraction, which is more unique. Additionally, it is known that patients can present with heart failure secondary to a Takotsubo-like cardiomyopathy. Generally, heart failure secondary to a pheochromocytoma is reversible after removal of the tumor. Our patient's function has not yet recovered, but he is less than 6 months post-operative. The reversible nature is dependent on the process being identified early to allow adequate treatment before irreversible changes occur. This patient likely had a prolonged duration of disease, making it less likely that his heart failure will be reversed. While pheochromocytomas are a rare cause of heart failure or cardiovascular disease, it is highly likely this condition is underdiagnosed as a recent study noted these tumors were identified in up to 0.05% of all autopsies. Also, a post-mortem study performed in Australia found a similar incidence of pheochromocytomas, and the study estimated that the tumor may have contributed to death in half of these cases. Therefore, the clinician must have an appropriate index of suspicion in new heart failure patients.

## 97) NOT CHILD'S PLAY: ADULT VARICELLA MENINGITIS

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**Introduction:** Acute aseptic meningitis is a rare complication that can occur in both primary and reactivation of Varicella viral infections. It is also not uncommon for patients to have no corresponding zoster lesions present at the time the patient develops aseptic meningitis.

**Case:** A 57-year-old woman with ESRD on dialysis, cirrhosis secondary to hepatitis C and alcohol abuse who initially presented following episodes of melena at home and was noted to have significant hyperkalemia. Patient was admitted to the medical intensive care unit for urgent dialysis and over the course of the next several hours proceeded to have acutely worsening mental status. Hemodialysis was completed without improvement in altered mental status. The next day patient had an acute febrile episode along with altered mentation raised concern for infectious meningitis. He was started on vancomycin, ceftriaxone and ampicillin. Patient continued to clinically worsen to severe sepsis with increased respiratory distress and tachycardia with heart rates as high as the 150s. Ceftriaxone was broadened to cefepime and patient was intubated. Lumbar puncture was negative for bacterial meningitis. CSF testing was negative for HSV but positive for Varicella Zoster Virus. Therapy was narrowed to acyclovir 10 mg/kg/dose and other antibiotics were discontinued. Over the next several days, patient improved clinically with resolution of altered mental status. Patient was then able to report that about one to two months prior to admission she had experienced a diffuse rash which she thought was chicken pox. On history, the patient had no prior history of varicella nor prior vaccination. Close examination of skin revealed minor scarring, but no active varicella lesions.

**Discussion:** When evaluating patient with suspected meningitis, VZV should be considered as a possible etiology. Fortunately PCR analysis for VZV remains an effective diagnostic measure. With appropriate initiation of acyclovir, patients with VZV meningitis have been shown to have improved mortality and morbidity. As such, it is recommended that empiric acyclovir be initiated any time HSV or VZV meningitis is suspected. Once confirmed, patients should complete 10 days of acyclovir 10 mg/kg/dose every 8 hours. Adults without prior VZV immunity should also receive two doses of the vaccination 4-8 weeks apart.

## 98) CUTANEOUS CLUES TO CAUSE OF COUGH

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**Introduction:** Lupus pernio is particularly difficult to treat lesion that frequently associated with and pathognomonic for pulmonary sarcoidosis.

**Case:** Patient is a 52 year old African American male who initially presented to the outpatient internal medicine clinic with complaints of “asthma”, and a nasal tip rash. The rash on his nasal tip had been present for at least 4 years, slow growing, nontender. The rash was “apple jelly”-like, somewhat vesicular on appearance, though the patient noted that when he had tried to express fluid from the lesions, blood was all that came out. He had tried multiple treatments ranging from petroleum jelly, topical steroids and various courses of antibiotics, with no effect. Seen previously by pulmonology, he had known pulmonary fibrosis and hilar lymphadenopathy seen on CT chest, in addition to obstructive lung disease proven by pulmonary function testing. He had poor control of his shortness of breath and a chronic dry cough, poor response to bronchodilators.

At a follow up appointment, patient was found to be hypoxic on room air, and was admitted to the hospital for further workup. Pulmonary consultation was obtained, and confirmed what the medicine team had suspected, that the rash was lupus pernio, which if biopsy proven, would be confirmatory of a sarcoidosis diagnosis. Biopsy done by dermatology revealed granulomatous inflammation, confirming the diagnosis of sarcoidosis. At the time of biopsy results, patient was readmitted for spontaneous pneumothorax, requiring video-assisted thoracic surgery with closure of an upper lobe air leak and talc pleurodesis. Patient has been doing well since pleurodesis with good activity tolerance.

**Discussion:** Lupus pernio is the most common cutaneous manifestation of sarcoidosis, and when seen, is nearly always associate with pulmonary involvement.

## 99) SPONTANEOUS SPLENIC RUPTURE

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**Introduction:** Splenic rupture can occur in the absence of trauma, often as the presenting symptom of an underlying disorder.

**Case Description:** Patient was a 46 year old male who presented to the ED after a sudden onset of left lower quadrant abdominal pain, diaphoresis and lightheadedness, which started while he was sitting. He described pain as severe, constant, radiating all over his abdomen without aggravating or relieving factors. In the ER, patient was hypotensive with a blood pressure of 80/50. He was stabilized with 3 L of normal saline, and a CT scan of the abdomen revealed a ruptured spleen with blood around the spleen, liver and a small moderate free fluid in the pelvis as well.

Surgery was consulted initially. Bleeding was found to be coming from a 3.7 cm splenic hemangioma which was subsequently addressed by interventional radiology. Patient's superior pole of the spleen was embolized using Gelfoam, and then the main splenic artery was embolized using 5 coils. He was monitored for symptoms of postembolization syndrome, which included nausea, vomiting, fever, headache, abdominal pain. Consideration for antibiotic prophylaxis was given, and per infectious diseases, patient was started on clindamycin. As patient was about to be functionally asplenic, vaccines were administered per infectious diseases input, and recommendations by Advisory Committee on Immunization Practices. His hemoglobin remained stable following the procedure, trending up by discharge. Patient was discharged home in stable condition with prescription for pain medications in anticipation of ongoing abdominal pain due to splenic infarct and irritation from hemoperitoneum. Patient did well on follow-up, having completed vaccination series.

**Discussion:** Splenic rupture without trauma or previously diagnosed disease is not an uncommon occurrence; common predisposing factors include infection, hematologic and nonhematologic tumors.

## 100) A CASE OF LYMPHANGIOLEIOMYOMATOSIS PRESENTING AS RECURRENT PNEUMOTHORAX

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**Introduction:** Lymphangioliomyomatosis (LAM) is a rare disease characterized by smooth muscle proliferation in the lung, causing cystic destruction leading to dyspnea and recurrent pneumothorax. The objective is to present a case of recurrent pneumothorax in sporadic LAM and review its diagnosis, management, and prognosis.

**Case:** Our patient is a 50-year-old female with chronic respiratory failure secondary to sporadic LAM diagnosed in 2003, requiring baseline 4 liters of nasal cannula oxygen. She presented in the ED with a 2-day history of anxiety, labile blood pressures, and dyspnea after hospitalization 1 week before for a pneumothorax. Exam showed tachycardia, tachypnea, and bibasilar crackles. Workup revealed a right apical pneumothorax and respiratory acidosis. She received a chest tube and was admitted to the MICU with BiPAP. After 2 days in stable condition, she was transferred to the floor when she suffered a repeat pneumothorax, resolved again with chest tube. She had an uneventful rest of her hospital course and declined pleurodesis and lung transplant, preferring palliative care. Of note, until her 30s, she maintained a physically active lifestyle, running 6 miles/day without symptoms. She has no family history of LAM. In 1998, she was misdiagnosed with exercise-induced asthma, but in 2003 during an "asthma attack," CT scan revealed LAM. She was told she had 5 years to live and declined lung transplant due to possible complications. Since then, she has suffered progressive dyspnea, requiring continuous nasal cannula oxygen. She credits her body's resilience to spirituality and alternative therapies.

**Discussion:** 2 types of LAM exist—tuberous sclerosis-associated and sporadic, which is not heritable. LAM is rare, with 1300 patients on the North American registry. Only females are affected, with average symptom onset at age 39--always after menarche but rarely after menopause. Clinical course is characterized by progressive dyspnea, with 60% experiencing spontaneous pneumothorax. Other complications include abdominal/thoracic lymphadenopathy, angiomyolipomas, and lymphangiomyomas. It is often misdiagnosed as asthma or COPD, as diagnosis requires CT or lung biopsy. Management is based on supportive care, avoidance of estrogen, and cautious use of sirolimus, which can slow its progression but is not curative. Pleurodesis is recommended at the first pneumothorax given its high rate of recurrence. Eventually, patients with respiratory failure require lung transplantation. Prognosis is much better than reported in the 1990s--median transplant-free survival time is 29 years from symptom onset and 23 years from diagnosis.

## 101) AN UNUSUAL CASE OF ACUTE HEART FAILURE IN A YOUNG WOMAN: ATYPICAL POSTPARTUM CARDIOMYOPATHY

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**Introduction:** Postpartum Cardiomyopathy (PPCM) is a rare cause of heart failure affecting women late in pregnancy and early in the postpartum period. It remains rare that this occurs early in pregnancy. We present a case of possible PPCM after first trimester abortion.

**Case:** Our patient is a 29-year-old female G3T1P1A1L1 with history of end-stage renal disease secondary to focal segmental glomerulosclerosis, abortion by dilation and curettage 4 weeks prior, moderate pulmonic regurgitation, and unspecified cardiomegaly on CT scan 5 months prior. She presented with a 1-month history of dyspnea on exertion and cough starting 1 week after D+C. On presentation, she was tachycardic, tachypneic, and hypertensive to 152/107. Her elevated B-HCG of 43.4 was reviewed with OBGYN and found to be appropriate, which downtrended during her course. CXR showed marked cardiomegaly, along with elevated troponin and new T wave inversions on EKG. TTE revealed new dilated cardiomyopathy with severe combined systolic and diastolic HF, with LVEF 20-25%. Left heart catheterization showed no obstructive coronary artery disease. The patient was discharged in stable condition with medical management, with future cardiac MRI to evaluate for infiltrative cardiomyopathy, as possible HF etiologies included PPCM, amyloidosis, myocarditis, and severe HTN.

**Discussion:** PPCM is a rare cause of heart failure usually presenting in the first month postpartum and rarely before 36 weeks of gestation, with typical heart failure symptoms. The incidence varies from 1:2289-4000 live births in the USA to 1:100 in Zaria, Nigeria. The etiology remains unknown, with research suggesting impaired angiogenesis from placenta-secreted VEGF inhibitors. Risk factors include age >30, African descent, pregnancy with multiple fetuses, history of preeclampsia, maternal cocaine abuse, and long-term oral tocolytic therapy with beta agonists. Diagnosis is based on 3 criteria: 1) development of heart failure at the end of pregnancy or the months following delivery, 2) absence of another identifiable cause, and 3) LV systolic dysfunction with LVEF <45%. For women who have delivered and are not breastfeeding, treatment mirrors standard heart failure therapy. One study suggests a mortality reduction with bromocriptine, but evidence is insufficient to establish safety and efficacy. While the mortality rate is 10% in 2 years, complete LV function recovery is reported in 20-60% of patients.

## 102) CONGENITAL LYMPHANGIOMA: AN UNUSUAL CAUSE OF RECURRENT TONGUE AND FACIAL EDEMA

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**Introduction:** Congenital lymphangioma is a rare malformation of the lymphatic system involving the skin and subcutaneous tissues that can present in the tongue. Here we present a case of lymphangioma causing recurrent tongue and facial edema.

**Case:** Our patient is a 34-year-old female with a history of congenital lymphangioma of the tongue and neck who presented with several days' history of tongue and facial edema, with 10/10 burning pain but no dyspnea or dysphagia. Since birth, she has undergone at least 6 operations to excise tongue cysts, always with recurrence. Over the past year, she has experienced painful episodes of tongue edema every 2 months, each requiring hospitalization with steroids for 4-5 days. On exam, tender angiomatous lesions were present on the anterior and mid-portion of the tongue, along with tongue and left facial edema, and significant bilateral tender cervical lymphadenopathy. Vital signs and labs were unremarkable. She was treated with dexamethasone, and IV morphine and was discharged 6 days later with resolution of pain and edema.

**Discussion:** Lymphangiomas are classified into Macrocystic (cystic hygroma) and microcystic malformations (lymphangioma circumscriptum). These can occur together and are likely two groups along a pathologic continuum. They account for 4% of all vascular tumors and are associated with some congenital disorders, including Turner, Down, and Noonan syndromes. 50% present at birth, with most by age 5. Cystic hygromas are composed of interconnected lymphatic cysts lined by endothelium, commonly in the neck, face, axilla, and chest wall. Complications include dysphagia and dyspnea from neck cysts. Lymphangioma circumscriptum is characterized by verrucous-appearing clusters of vesicles that contain clear lymph fluid, representing lymphatic vessel dilations. They appear pink to dark red secondary to serosanguinous fluid and hemorrhage, and may intermittently leak, increasing risk for cellulitis. The tongue is commonly affected, as are the proximal extremities, trunk, axilla, and oral cavity. Diagnosis is made with histologic and immunohistochemical studies, and MRI can be used to determine the extent of lesions. Treatment is surgical excision, but local recurrence is common because of inability to completely remove subcutaneous tissue. While lymphangiomas normally do not progress to malignancy, two cases of lymphangiosarcoma arising from lymphangioma circumscriptum have been reported after attempted radiation therapy.

### 103) DULAGLUDITE RISK FOR ACUTE KIDNEY INJURY

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**Introduction:** Dulaglutide is a glucagon-like peptide-1 receptor agonist (GLP-1) approved for the treatment of type 2 diabetes mellitus (T2DM). GLP-1 agonists work by slowing gastric emptying while both increasing insulin and decreasing glucagon secretion. Because of their low hypoglycemia risk and benefit of clinically relevant weight loss, dulaglutide and other GLP-1 agonists are becoming popular drugs for T2DM management. Despite its efficacious treatment and reported safety profile, there are 78 reported cases of acute renal failure or renal insufficiency in patients using exenatide, another GLP-1 agonist.

**Case:** A 60-year-old female presented with full body tremors that made it difficult to walk for two days prior to admission. Additionally, she reported blurry vision, weakness, feeling cold, dysuria, increased urinary frequency, and urge incontinence. Her PCP had switched her from exenatide to dulaglutide one day before the onset of symptoms. She was also taking Humulin, canagliflozin, valsartan, furosemide, and metolazone. Her past medical history was significant for diabetes, renal dysfunction, heart failure, hypertension, and vascular disease. Physical exam was significant for intention tremor in all four extremities and asterixis superimposed on upper extremity intention tremors. In the emergency department, she was found to have a creatinine of 4.84 mg/dl and a BUN of 40 mg/dl. Her baseline creatinine was 1.11 mg/dl from 1/26/14 but had climbed steadily to 1.75 mg/dl on 7/31/2015. Her presenting FEUrea was <35%, which was consistent with prerenal etiology. Her dulaglutide, canagliflozin, valsartan, and diuretics were discontinued at the time of admission. Over two days, her creatine returned to baseline of 1.14 mg/dl and BUN trended down to 20 mg/dl with aggressive fluid management.

**Discussion:** In the United States, polypharmacy leading to acute renal dysfunction is an all too common problem, as witnessed in our patient. In combination, canagliflozin, valsartan and furosemide are known to cause renal insufficiency. Yet, our patient had been on these medications long before her onset of uremic tremors. It seems more than coincidence that her symptoms and acute rise in creatinine began one day after starting dulaglutide, and that her kidney function significantly improved immediately following discontinuation of all offending agents. Accordingly, physicians must be cognizant of reported side effects when starting new medications; in particular, the potentially deleterious effects of dulaglutide on kidney function especially in combination with other potentially nephrotoxic agents. When patients present in the setting of worsening organ function, a comprehensive reassessment of the medication list needs to be done as these clearly can be the pathological sources. In this case, the patient's kidney damage does seem to be related to the addition of dulaglutide.

### 104) NOT YOUR AVERAGE SORE THROAT: ACQUIRED HEMOPHILIA A PRESENTING WITH SPONTANEOUS NECK HEMATOMA

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**Introduction:** Acquired hemophilia A is an exceedingly rare bleeding disorder. We present a case of acquired hemophilia A with that presented with a spontaneous neck hematoma causingodynophagia and dyspnea.

**Case:** A 73-year-old male with a history of diabetes mellitus and atrial fibrillation on warfarin presented with a sore throat for the past several days and two weeks of easy bruising. Physical exam was notable for anterior neck ecchymosis with palpable hematoma, extensive ecchymosis of his extremities, and a hoarse voice. Laboratory evaluation was significant for a hemoglobin of 8.9 g/dL, white blood cell count of 15,600/uL, platelet count of 359,000/uL, PTT >100 seconds, and an INR of 1.0. Mixing studies did not correct on 1:1 mix. A factor VIII activity level was undetectable with a factor VIII inhibitor present at 29 Bethesda units. Computerized tomography (CT) scan demonstrated a large hematoma in his submandibular gland and pharyngeal space with narrowing of the airway. He was initially treated with recombinant factor VIII and corticosteroids. Later he was treated with recombinant porcine factor VIII given it provided the ability to monitor factor VIII activity levels. Additionally, he was treated with three cycles of rituximab 375 mg/m<sup>2</sup>. He had no further bleeding events and his anterior neck hematoma improved significantly.

**Discussion:** Acquired factor VIII inhibitors are exceedingly rare in the non-hemophiliac with an estimated incidence of 1-4 per million/year. In contrast with congenital hemophilia A, where patients suffer from chronic hemarthroses, those with the acquired disorder suffer instead from skin, muscle and soft tissue, gastrointestinal, genitourinary, and retroperitoneal bleeds. Diagnosis is usually made when a patient presents with excessive bleeding and no prior history of coagulopathies with a prolonged PTT that does not correct upon mixing with normal plasma. Treating the disorder focuses on two things: addressing any associated pathologies, and the bleeding diathesis, including the permanent elimination of the inhibitor and management of acute bleeds. Common strategies to treat or prevent acute bleeds are to use agents that either bypass the inhibitor, such as activated factor VIII, or to directly raise factor VIII concentrations. When inhibitor titers are prohibitively high for use of human factor VIII, recombinant porcine factor VIII is useful. For elimination of the inhibitor, convincing evidence for the superiority of one immunosuppressive agent compared to the others is not currently available. Commonly used agents include cyclophosphamide, prednisone, and rituximab.

## 105) THE AFFORDABLE CARE ACT AND PREVENTIVE SERVICES: THE ARGUMENT FOR FOCUS

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The Affordable Care Act of 2010 significantly changed how healthcare is delivered in the U.S. Section 2713 of the ACA specifically created incentives to lessen the cost burden on consumers by requiring zero cost sharing from consumers for clinically proven primary preventative services. Secondary and tertiary preventions were, however, left out of the ACA despite opportunities to offer better cost-effectiveness compared to primary prevention interventions. This study looked at differences in cost effectiveness of breast and colon cancer screening for primary versus secondary screening based on number needed to screen analysis (NNS). We also studied the cost effectiveness of medications for primary prevention versus prevention of complications in population with high risk diseases. For cancer screening, values were generated from general population studies and epidemiologic data. We found that the NNS for populations at high risk for breast cancer, specifically BRCA1 and BRCA2 mutations, are much lower than the NNS for the general population. Similarly, the NNS for populations at high risk for colon cancer, specifically FAP, Lynch syndrome, Peutz-Jeghers, and Ulcerative Colitis are much lower compared to the NNS for the general population. For medications, ICER values were obtained from primary literature searches and adjusted for inflation. The results looking at Aspirin, Statins, Beta blockers, ACEi, or bisphosphonates, the cost-effectiveness of these medications increases drastically in secondary or tertiary prevention measures compared to primary or secondary prevention counterparts. Given the current disease burden within the U.S., this study would indicate the benefits of reducing cost sharing for consumers for secondary or tertiary prevention as well as primary prevention as a means to reduce overall healthcare costs and improve overall health outcomes.

## 106) TO FLY OR NOT? AIR TRAVEL IN PATIENTS AT RISK FOR PNEUMOTHORAX

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A 64 year old female with history of stage IIIC clear cell ovarian carcinoma treated with total abdominal hysterectomy and bilateral salpingo-oophorectomy and chemotherapy and also stage I clear cell renal cell carcinoma status post left nephrectomy was found to have multiple small perivascular and subpleural thin walled air cysts found incidentally on surveillance CT scan. Her oncologic history and now pulmonary findings was suggestive of Birth Hogg Dube syndrome, a rare autosomal dominant inherited genetic syndrome characterized by pulmonary cysts, fibrofolliculomas, as well as predisposition to renal and other cancers. She was brought into clinic for further counseling, where on physical exam she was noted to have characteristic flesh colored facial papules. She was subsequently referred to genetic clinic, where testing confirmed her diagnosis – a missense mutation to the FLCN gene called c. 1433-2 A>G. Given risks of cancer recurrence and also spontaneous pneumothorax, there were many questions that arose regarding surveillance and flying.

Birt Hogg Dube was first described in 1977, when 6 siblings were identified to have thyroid neoplasms and fibrofolliculomas. 80% of patients are known to have pulmonary cysts and while it is unusual to have progressive pulmonary disease, risk of pneumothorax is 50 times higher than the general population. Risk of renal cancer is approximately 15% by 70 and there is no consensus on screening.

Rare genetic conditions for which there exists little available information often pose a challenge to the primary care provider, as questions arise in both management and prognosis. In this case, it is helpful to extrapolate information from other similar conditions in order to best guide clinical decision making. Lymphangiomyomatosis, another rare pulmonary disease characterized by numerous cysts and progressive lung function decline where patients are more at risk for complications, offers some insight. In analysis of patients flying and transporting by ground to NIH to participate in research, spontaneous pneumothorax was more common in those with a prior history, as well as those with more severe disease. Still, frequency was overall low and there has been no incidents requiring hospitalization in over 500 patients for over 10 years.

## 107) ANOMALOUS CONNECTIONS OF THE HEART: A CASE OF CORONARY ARTERY FISTULA

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**Introduction:** Coronary artery fistulas are rare connections that directly link a coronary artery to a major thoracic vessel or a cardiac chamber without an interposed capillary bed. Many of these fistulas are incidentally found in asymptomatic children, but if found later in life can be a rare cause of severe heart failure.

**Case Presentation:** An 82-year-old male with a history of heart failure with preserved ejection fraction and tricuspid regurgitation presented with worsening shortness of breath. On exam he had 3 distinct murmurs; a 2/6 systolic murmur at the base, a 3/6 systolic murmur at the left lower sternal border (LLSB) that increased with inspiration, and a 3/6 high pitched diastolic murmur heard best at the LLSB. Canon V waves and a pulsatile liver were also present. Transthoracic echocardiogram showed a strange honey-comb appearing image in the right atrial free wall with an abnormal diastolic jet. Bubble study was grossly positive but with the bubbles appearing from the sites of the pulmonary veins. Transesophageal echocardiogram showed a fistula to the RA and RV. Coronary angiography revealed a grossly ectatic RCA and cardiac MRI showed a fistula to the RA. Coiling of the aneurysm was considered but thought to be of extremely high risk of causing thrombosis and subsequent infarction. The patient decided to go hospice and died shortly after.

**Discussion:** Coronary artery fistulas only represent 0.1% of cardiac anomalies. They can be acquired from instrumentation or trauma but are most commonly congenital in origin. The RCA is the involved vessel in 50-60% of cases with drainage in the right chambers 90% of the time - usually to the RV. This patient only had confirmed drainage into the RA but his echocardiographic findings were suspicious for additional connections. It is likely the increased flow from his left to right shunt was the cause of his dilated tricuspid annulus and severe right heart failure. Treatment options include surgical ligation or catheter device closure but intervention is controversial due to the largely unknown natural history of the disease. The effect of intervention on mortality is also unclear.

## NOTES



**ACP**<sup>SM</sup>

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