Abstracts from the 2016 Annual Louisiana American College of Physicians (ACP) Associates Meeting

Each year medical students in Louisiana and residents from the eight Internal Medicine training programs in Louisiana are invited to submit abstracts for the Annual Louisiana American College of Physicians (ACP) Associates Meeting. The content of these abstracts includes clinical case vignettes or research activities. The abstracts have all identifying features removed (i.e., names, institutional affiliations, etc.) before being sent to physician judges. Each judge scores each abstract independently and then the scores from all judges are averaged and ranked. This year we are excited to be able to publish most highly ranked abstracts presented at this year's competition. These abstracts (17 oral; 14 poster) were presented at the Associates Meeting held at LSU Health Sciences Center in Baton Rouge on January 19, 2016. We would like to thank the *Journal* of the Louisiana State Medical Society and appreciate its efforts to publicize the hard work of these trainees.

Lee S. Engel, MD, PhD, FACP
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A BONE TO PICK WITH DYSPHAGIA

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Case: A 55-year-old woman with a history of seizures and submandibular stones, presented with dysphagia. Five days prior to presentation, she woke up with urine in her bed, headache, and confusion consistent with prior nocturnal seizures; however that morning she had difficulty swallowing solids, accompanied by nausea and vomiting. Over the next few days, her symptoms worsened to include mild dysphagia to liquids, shortness of breath, and voice changes. On physical exam, she was hypertensive at 164/97 with oxygen saturation at 97% on room air. She had submandibular fullness bilaterally with a clear oropharynx. No focal neurologic deficits were appreciated. Flexible fiber optic laryngoscopy found the posterior pharyngeal wall significantly pushed anteriorly. Modified barium swallow demonstrated a narrow pharynx due to mass versus swelling of tissue on posterior pharyngeal wall at C3-C4, leading to decreased laryngeal elevation and poor epiglottic inversion. CT neck with IV contrast demonstrated exuberant osteophytes with preserved intervertebral disk height located at C3, C4, C5, C6, and C7 with C3-C4 osteophytes displacing the prevertebral soft tissues anteriorly at the level of the epiglottis, consistent with symptomatic Forestier's syndrome. A PEG tube was placed for nutrition requirements and neurosurgery recommended outpatient surgical decompression.

Discussion: Forestier's disease, also known as DISH (diffuse idiopathic skeletal hyperostiosis), was first described in 1950 by Forestier and Routes-Querol. While it is not rare, it is often under-diagnosed. In the United States the prevalence of DISH is approximately 25% of men and 15% of women older than 50 years. DISH is usually latent, and thus asymptomatic, though it can lead to compression of local tissues, manifesting symptomatically as dysphagia, dysphonia, stridor, and dyspnea. The treatment of DISH depends on disease severity with a conservative approach in mild to moderate cases, surgical treatment in severe or refractory cases. Muscle relaxants and anti-inflammatory drugs are recommended for initial treatment.

CAN SJŐGREN'S AFFECT YOUR CNS? – IT SJO' CAN!

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Introduction: Sjőgren's Syndrome is a common disease process with poorly understood variant features. One variant involving the central nervous system is known as CNS Sjőgren's and can be classified as: Transverse Myelitis, Neuromyelitis Optica (Devic's Disease), and Multiple Sclerosis like syndrome. Patients with central nervous system involvement generally tend to have more profound disability.

Case: Our patient first presented with right arm and leg pain for approximately 10 months. Abruptly, she developed contractures of her right 4th/5th digits, inward rotation of her right ankle, and increased spasticity of her right side. She was found to have a positive ANA and SSA but failed a course of prednisone/plaquenil. She was given the diagnosis of Sjőgren's while workup continued for her neurologic symptoms. These progressed leading to hospitalization for right sided neuropathic pain, dysphagia, dysarthria, profound ophthalmoplegia, and immobility from contractures. MRI of her brain demonstrated several T2 subcortical and periventricular hyperintensities localized primarily in the frontal cortex and pons. Serology was negative for NMO/

Aquaporin 4, Neuronal Nuclear Ab, Proteinase 3 Ab, and oligoclonal bands were absent. The patient was then diagnosed with CNS Sjőgren's and started on pulse dose Solu-medrol with mild improvement. A subsequent trial of intravenous Cytoxan was given prior to discharge and prednisone 50mg was to be taken daily. At follow-up she was able to ambulate, had clear pronunciation, less diplopia, and decreased pain. Unfortunately, remission was brief requiring a second course of steroids and Cytoxan without the previous improvements noted. Shortly thereafter, she re-presented with generalized seizures and respiratory failure and was ultimately transitioned to hospice care.

Discussion: Our case highlights the presentation of a poorly understood variant of a fairly common disease process. Although this patient did not demonstrate the typical features associated with exocrine glandular Sjőgren's, she was found to have CNS sequelae that was directly linked to her autoimmune disorder. This underscores the importance of maintaining a broad differential when patient's symptoms and physical/laboratory findings do not fit a particular disease process.

CHASING THE DRAGON: A CASE OF HEROIN INHALATION LEUKOENCEPHALOPATHY

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Introduction: "Chasing the Dragon" is a slang term which refers to the inhalation of the heated vapors of heroin. First described in 1982, heroin inhalation leukoencephalopathy (HIL) is a rare but serious complication of heroin use by inhalation.

Case Description: A 35 year old incarcerated woman with a history of intravenous heroin use was brought in by the sheriff's office for three days of altered mental status. The jail physician noted "unsteady gait, weakness, delayed responses, and lethargy". She complained of headache and generalized weakness, which she attributed to "withdrawing." Her last heroin use was four days prior to presentation. On exam, she was noted to be apathetic with generalized psychomotor slowing. Her neurologic exam was significant for right lower extremity weakness and diminished sensation, brisk patellar reflexes, and bilateral ankle clonus. She was uncooperative with cerebellar testing and was unable to stand due to right extremity weakness. Initial labs revealed a slight leukocytosis (13.6) without left-shift, hypokalemia (2.9), transaminitis (AST 292 U/L, ALT 147U/L) and elevated CK (1864). Urine drug screen was positive for opiates and benzodiazepines. Brain MRI demonstrated symmetric cortical/subcortical T2 hyperintensities in the watershed territories of the bilateral parietal and occipital lobes consistent with leukoencephalopathy. Spine MRI, EEG, CSF studies, rheumatologic panel, MS panel, and infectious work-up were unremarkable. On further questioning, she admitted to heroin use by inhalation which led us to the diagnosis of HIL. She was started on a trial of coenzyme Q and received intensive physical therapy resulting in improved mentation and physical function.

Discussion: Symptoms of HIL include cerebellar dysfunction, psychomotor retardation, soft speech, and apathy. Symmetric T2 hyperintensities in the posterior cerebral hemispheres and cerebellum are seen on MRI. Heroin vapors may be directly toxic to mitochondria, and result in a vacuolar degeneration of myelin sheaths. Treatment includes supportive care; anti-oxidants such as coenzyme Q have been used anecdotally. HIL should be considered in patient's presenting with a history of heroin inhalation and new onset neurologic or behavioral changes.

SYPHILIS MANIFESTING AS SYMPTOMATIC HEPATITIS

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Introduction: Abnormal liver enzymes are frequently encountered in the clinical medicine setting. With a myriad of potential causes, the etiology of liver damage may at times be difficult to determine. Although an uncommon precipitant of clinical hepatitis, syphilis should be considered in the differential diagnosis of these patients.

Case: A 34-year-old African American man presented to clinic with a chief complaint of right upper quadrant abdominal pain for one month. He had a history of HIV diagnosed in 2012 with most recent CD4 count of 597 cells/mm3 and HIV viral load of 4,349 copies/mL. He was on antiretroviral therapy with darunavir, tenofovir, emtricitabine and ritonavir. His labs showed elevated liver chemistries with alanine aminotransferase (ALT) of 169, aspartate aminotransferase (AST) of 109, and alkaline phosphatase (ALP) of 250. Total bilirubin was within normal limits. HIV medications were held at that time. One week later, repeat labs showed worsening liver chemistries with ALT of 472, AST of 326, ALP of 559 and total bilirubin of 1.5. His acute hepatitis panel was negative and he denied the use of any alcohol or hepatotoxic medications. Abdominal ultrasound showed mild hepatomegaly without obstruction or stones. MRCP was negative for biliary or pancreatic ductal dilation. Physical exam revealed a diffuse hyperpigmented maculopapular rash on abdomen, extremities, soles of feet and palms of hands concerning for secondary syphilis. RPR was reactive with a reflex titer of 1:64. Treponemal pallidum IgG which was previously negative returned reactive. Patient was given penicillin G 2.4 million units IM once for treatment. Two weeks later, liver chemistries had normalized, RPR titer had decreased to 1:4 and his abdominal pain had resolved.

Discussion: Syphilis is typically known to present with mucocutaneous disease and lymphadenopathy but less commonly with liver involvement. Due to the prompt and effective response to penicillin G, it is important to consider syphilis in the differential diagnosis of hepatitis, particularly in HIV infected patients. In addition, early recognition could prevent unnecessary medical

testing and cost.

GIANT CORONARY ARTERY ANEURYSM WITH 3 VESSEL INVOLVEMENT IN A PATIENT WITH ACROMEGALY

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Introduction: Coronary artery aneurysm (CAA; defined as the dilation of coronary artery to greater than 1.5 times the normal diameter) is a rare anomaly with an incidence of 0.15% to 5.0%. Even rarer is a giant coronary artery aneurysm (G-CAA; defined as >20mm in size of the artery) with an incidence of 0.02% to 0.2%.

Case: A 47-year-old man with acromegaly presented with a ST-elevation myocardial infarction and on coronary angiogram was noted to have one G-CAA involving the left anterior descending (LAD) and two CAA involving the left circumflex (LCX) and right coronary artery (RCA). A very large LAD artery was noted with mid and distal thrombotic occlusion. The LCX and RCA were also aneurysmal and did not fill distally. There was no coronary atherosclerosis noted. Due to inability to perform any percutaneous coronary interventions in the large aneurysmal LAD artery, the patient was treated medically with aspirin, clopidogrel, and low molecular weight heparin. Chest CT angiography confirmed the aneurysms: the LAD artery had a 33 mm in diameter x 70 mm in length proximal aneurysm, proximal RCA with 13 mm aneurysm, and proximal and distal LCX artery with 8mm and 10mm aneurysm respectively. Transthoracic echocardiography showed severe systolic dysfunction with approximate LVEF of 30%. A life vest was placed on the patient with a plan to place an implantable cardioverter- defibrillator or plan for cardiac transplantation and the patient was discharged on Aspirin, Clopidogrel, Metoprolol and Rosuvastatin.

Discussion: This is the only reported case of G-CAA in an acromegalic patient in the literature where there is no evidence of vasculitis, no previous left heart catheterization, or any genetic connective tissue disorder; traditional risk factors predisposing the development of the coronary artery aneurysms. Complications of CAA include rupture, distal embolization, thrombosis and vasospasm. We emphasize the fact that all patients with CAA need to be on antiplatelet therapy and anticoagulation. Treatment options vary depending on the presence of atherosclerosis: Surgical treatment is recommended in the presence of atherosclerosis and severity of stenosis distal to the CAA while medical therapy is limited to the patients with absence of atherosclerosis and minimal stenosis distal to CAA.

THE POSSIBILITY OF PRE-EXPOSURE PROPHYLAXIS IN AN UNIDENTIFIED SUB-GROUP IN LOUISIANA

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Introduction: The CDC estimates that our preventative efforts thus far have averted more than 350,000 HIV infections as well as more than $125 billion in costs of medications. Further, the CDC estimates that an average of $355,000 is saved for every HIV infection prevented. Thus far, very few completed Pre-exposure prophylaxis (PrEP) trials have included persons under the age of 18.

Methods: This study looked at the population of Louisiana and data obtained by the Louisiana Department of Health and Hospitals Office of Public Health STD/HIV Surveillance program, specifically the age at detection of HIV, number of cases and percent of cases within age groups in both 2014 and 2013 data. A comparison between two populations between January to December 2013 and January to December 2014 comparing the number of cases detected in age groups from 0-12, 13-24, 25-34, 35-44, 45 + was made. A Student 2 Sample T-test comparing these two groups was used to examine for statistical difference between the mean numbers of cases between the two groups ranging from 2013 to 2014. A comparison of the total percent of cases of newly diagnosed HIV in each group was made between the ages of 13-24.

Results: The number of cases of newly diagnosed HIV has higher in the 2014 data group than the 2013 group [344 cases compared to 321]. Comparing the two groups the T-value is 0.056102 with a P-Value of 0.956637. This shows no statistical difference between the groups in the number of cases in the respective years 2013 and 2014.

Conclusion: Comparing the number of cases of HIV diagnosed in Louisiana from 2013 to 2014, there is no significant difference in the total number of cases. The question proposed by this study is whether our efforts at minimizing the incidence of HIV through preventative measures has therefore been sub-optimal as there is no significant difference between total incidence in the span of 1 year. However, it should be noted when comparing data the percentage of cases diagnosed from ages 13-24 had increased from 24% to 26% in the span of 1 year. Current guidelines do not advocate for PrEP in persons under the age of 18. We propose that further studies are required to establish whether an under-represented age group in HIV incidence exists in the age ranges of 13-24.

WHERE SUPPLEMENTS CAN GO WRONG: HYPERVITAMINOSIS DUE TO OTC ERGOCALCIFEROL

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Introduction: There are multiple causes of hypercalcemia, the most common being primary hyperparathyroidism and malignancy. With the increasing incidence of Vitamin D insufficiency, Vitamin D supplements have been marketed as an over-the-counter medication. However, the numerous available dosages and formulations can lead to confusion amongst consumers.

Case: A 71-year-old woman with a history of COPD, CAD, CHF, and CKD3 was admitted with 4 days of generalized weakness and shortness of breath after stopping furosemide 4 days prior. Her heart rate was 67 bpm, blood pressure was 159/79 mm Hg, respiratory rate was 20 bpm, and her oxygen saturations were 94% on 3L nasal cannula with no prior oxygen requirement. She had bibasilar rales, mild lower extremity edema, and a CXR consistent with fluid overload. The patient had a corrected calcium of 11mg/dL, magnesium of 1.5 mg/dL, a phosphorus of 2.8 mg/dL and a creatinine of 1.1mg/dl on admission. She was initially treated for both a CHF exacerbation and COPD exacerbation. During this admission a daily trend of increasing serum calcium levels was noted up to a peak of 14.7mg/dL. She had a 25-hydroxycholecalciferol of >96 ng/mL, 1,25-dihydroxycholecalciferol of >156pg/mL and a parathyroid hormone level of <5.0 pg/mL. Upon further questioning, her vitamin D had been prepackage to provide vitamin D2 50,000U daily instead of once weekly. Her hospitalization was complicated by the development of acute pancreatitis (lipase of 753 U/L) secondary to her hypercalcemia. She was started on calcitonin (4U/kg), IV fluids, IV furosemide and denosumab 60mg subcutaneously and her calcium slowly decreased to 10.7 mg/dL corrected on discharge.

Discussion: Vitamin D toxicity is a rare condition usually caused by ingestion of excessive amounts of Vitamin D. Treatment includes corticosteroids, bisphosphonates, low calcium diet, IV fluids and furosemide. In this case, bisphosphonates were contraindicated due to renal insufficiency, and thus denosumab was used. This case highlights the importance for greater monitoring of supplements marketed to the public as misuse can cause significant harm to the public.

AN UNUSUAL CASE OF ACUTE RESPIRATORY PARALYSIS

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Introduction: West Nile Virus (WNV) should be included in the differential in patients who present with focal neurological deficits and weakness even if the patient does not report a mosquito bite. Although the incidence peaks in late summer or early fall, sporadic cases can occur throughout the year in southern states

Case: A 56 years old Caucasian man with no past medical history was transferred from an outside facility for further management of community-acquired pneumonia diagnosed by CT Chest. He initially presented with fever, vomiting and non-bloody foul smelling loose stools. Upon arrival at our facility, he was found to have severe bilateral proximal upper extremity weakness. He was alert and oriented. There was no sensory deficit. The lower extremity strength and bilateral handgrip were preserved, but motor strength was 1/5 on adduction and abduction of his bilateral arms. Bilateral triceps and biceps deep tendon reflex were absent. His condition rapidly deteriorated by day 2 of hospital stay with worsening inspiratory effort and change in his voice that necessitated emergent intubation due to neuromuscular paralysis. Pharyngeal-cervico-brachial type Guillain-Barré syndrome (GBS) was suspected. Lumbar puncture was done. Initial cerebrospinal fluid (CSF) analysis showed 86mg/dl of protein and WBC count of 53. Weight based intravenous immunoglobulin was initiated. Electro-diagnostic study was deferred until the patient's condition stabilized. He also developed dysautonomia manifested by tachy and brady arrhythmias and labile blood pressure. However, on day 7 of hospital stay, CSF WNV IgM Antibody came back positive. He eventually required a tracheostomy. He remained ventilator-dependent, awaiting transfer to a long-term acute care facility.

Discussion: Although involvement of anterior horn cells is the major site of spinal cord pathology in WNV, involvement of spinal sympathetic neurons can explain the dysautonomia seen in these patients. As in GBS, early recognition of possible respiratory failure is important when patients present with an unknown reason for acute paralysis.

ENDOCRINE AND CHEMOTHERAPEUTIC RESISTANCE IN BREAST CANCER IS DRIVEN BY LIGHT AT NIGHT-INDUCED DISRUPTION OF THE CIRCADIAN MELATONIN SIGNAL

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Introduction: Endocrine (tamoxifen, Tam) and chemotherapeutic (doxorubicin, Dox; Paclitaxel, Pax) resistance represent major impediments to the successful treatment of breast cancer and are coupled to increased tumor metabolism and tumor overexpression and activation of several families of receptor- and

non-receptor-associated kinases. Dim light at night (dLAN) exposure, as occurs in shift work and/or perturbed sleep-wake cycles, disrupts circadian time structure and nocturnal melatonin production, which is associated with a significantly increased risk of an array of diseases, including breast cancer. Melatonin inhibits human breast cancer growth via mechanisms that include suppression of tumor linoleic acid (LA) metabolism, aerobic glycolysis (Warburg Effect), and expression and/or phospho-activation of AKT and ERK1/2 receptor kinases along with several other kinases and transcription factors, including STAT3.

Methods and Results: Female nude rats bearing tissue-isolated estrogen receptor positive (ERα+) MCF-7 human breast cancer xenografts were maintained on a light/dark cycle of LD 12:12 in which dLAN is present during dark phase (suppressed endogenous nocturnal melatonin), significant ($p < 0.001$) decreases in tumor latency-to-onset, increased tumor metabolism and growth, and total intrinsic resistance to either Tam, Dox, or Pax therapy. Conversely, a LD 12:12 dLAN environment incorporating nocturnal melatonin replacement resulted in markedly increased latency-to-onset, tumor regression, suppression of tumor LA metabolism and Warburg Effect, and inhibition of kinase and transcription factor phosphorylation, while Tam, Dox, and Pax sensitivity was completely restored.

Conclusion: Melatonin behaves as both a tumor metabolic inhibitor and circadian-regulated kinase inhibitor to reestablish human breast tumor to Tam, Dox or Pax and drive tumor regression further demonstrating that dLAN-induced circadian disruption of nocturnal melatonin production contributes to a complete loss of tumor sensitivity to endocrine or chemotherapeutic interventions.

A RARE COMPLICATION OF CHRONIC MYELOID LEUKEMIA

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Introduction: Hematologic malignancies are often staged by blast cells in the blood or bone marrow. Metastasis to solid organs or tumor transformation can also affect prognosis. The solid phase of chronic myelogenous leukemia (CML), myeloid sarcoma, is described in less than 5% of cases. Myeloid sarcomas are poor prognostic factors and often occur as result of medication non-compliance.

Case: A 40-year-old woman was diagnosed with CML. She was initially compliant with imatinib, however self-discontinued the medication three years later after feeling well. She then presented to the emergency room with right-sided facial droop and extremity weakness. Subsequent imaging showed multiple non-enhancing lesions in the posterior fossa associated with hemorrhage, edema, and effacement of the 4th ventricle. On presentation her white blood cell count (WBC) was 571,000 (cell/ul). She was immediately started on hydroxyurea, nilotinib, and underwent 2 cycles of leukapheresis. Her WBC responded and within ten days was within normal limits. Twelve days after admission repeat imaging showed resolution of the hemorrhage, a decreased number of brain lesions, and hydrocephalus. She continued to do well and before discharge had only minor deficits. One month after discharge repeat brain imaging showed resolution of masses, hydrocephalus, and no new hemorrhage. She was diagnosed with CML myeloid sarcoma presenting as a new brain mass.

Discussion: The solid phase of CML was first described in 1811. Myeloid sarcomas consist of immature cells of granulocytic lineage that are rarely associated with chronic phase CML. Chemotherapy is recommended for myeloid sarcomas as it improves progression to AML and survival. Radiotherapy and surgery decreases symptoms related to mass effect of tumor, but do not improve survival. In the case described, progression to myeloid sarcoma was due to medicine non-compliance. She was transitioned to nilotinib due to the decreased chance of intracranial hemorrhage compared to imatinib. Diagnosis of Myeloid sarcoma should be considered with a tumor in the setting of CML. If in the CNS, strong consideration should be given to nilotinib over imatinib or dasatanib.

ADRENAL INSUFFICIENCY PRESENTING AS END STAGE CARDIOMYOPATHY

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Introduction: Cardiomyopathy is a disease of weakened or defective heart muscle that can cause heart failure. There are many causes of cardiomyopathy including genetic, toxic, infectious and inflammatory. Rarely, cardiomyopathies can be of endocrine etiology. This clinical vignette describes a case of severe dilated cardiomyopathy caused as a result of Sheehan Syndrome.

Case: A 40-year-old woman presented with chief complaint of dyspnea upon walking, paroxysmal nocturnal dyspnea and lightheadedness. She had a past medical history significant only for Sheehan Syndrome after a post-partum hemorrhage complicated her last pregnancy. Upon admission, echocardiography was performed which demonstrated a normal sized left ventricle and an ejection fraction of 10%. Her laboratory studies were significant for a TSH of 6.8 μU/mL and a BNP of 1400 pg/ml. The patient was evaluated by the heart failure team for a possible heart transplant. Further laboratory testing demonstrated a decreased cortisol level. LH, FSH, Prolactin, estradiol and IGF-1 were also decreased. A cortisol stimulation test was found to be positive and the patient was diagnosed with secondary adrenal insufficiency. An MRI of the pituitary was performed which demonstrated an empty sella. She was subsequently started on stress dosing of Hydrocortisone. The patient was started on Hydrocortisone and her lab abnormities normalized. She saw an

increase in her energy levels and improvement in her shortness of breath. In subsequent months, her cardiac function improved dramatically. More recent echocardiography demonstrated ejection fractions of 55-65% and the patient no longer has any symptoms of heart failure.

Discussion: Genetic, infectious, toxic and ischemic causes of cardiomyopathy are well documented and seen frequently on both cardiology and general medicine services. However, endocrine abnormalities causing severe cardiomyopathy are far less common. What makes this case even more unusual is the dramatic improvement the patient experienced over a very short period of treatment. This case demonstrates the importance of keeping a broad differential, and considering endocrine etiologies, when evaluating cardiomyopathies

AN UNUSUAL PRESENTATION OF DRESS SYNDROME

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Introduction: Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome is a potentially fatal condition that typically presents as fever and a cutaneous morbilliform exanthem preceding internal organ involvement 2-6 weeks following ingestion of the offending agent.

Case: A 64-year-old woman with past medical history of psoriatic arthritis and pustular psoriasis on chronic anti-TNF inhibitors presented with bilateral cervical and submandibular lymphadenopathy following an URI treated with amoxicillin. She subsequently underwent multiple dental extractions for caries. Her post-op course was complicated by persistent lymphadenopathy and stomatitis treated with amoxicillin, clindamycin, and trimethoprim-sulfamethoxazole. She re-presented with progressive lymphadenopathy, fever, and a productive cough. She was diagnosed with community-acquired pneumonia and was discharged on amoxicillin and azithromycin following resolution of pyrexia. Three days later, she presented with a diffuse morbilliform rash and fever of two days duration. After improvement with a dose of methylprednisolone, the rash was attributed to an underlying vasculitis. A month later she was admitted to our care with extensive mucositis and painless, firm lymphadenopathy. She underwent further evaluation for atypical viral, mycobacterial, and fungal organisms, autoimmune, and neoplastic etiologies including excisional lymph node and cutaneous biopsies. Histopathology was consistent with a drug reaction, and the patient was diagnosed with DRESS likely secondary to amoxicillin. She exhibited remarkable improvement with prednisone and was discharged on a steroid taper with dermatology follow-up.

Discussion: The initial manifestation of DRESS in this patient was lymphadenopathy. This exceptionally uncommon presentation obscured the underlying disease process for several weeks. Further complicating the presentation was the patient's intermittent use of low dose steroid therapy for her comorbid psoriasis. The use of chronic immunosuppressive therapy led clinicians to investigate infectious and neoplastic etiologies. A notable feature is the rapid acceleration of DRESS symptoms following re-challenge with amoxicillin. This case illustrates the importance of considering delayed onset drug reactions in patients with persistent lymphadenopathy.

DON'T WAKE UP THE FOLLICULARLYMPHASAURUS REX

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Case: A 58-year-old man with history of coexistent stage IV follicular lymphoma and multiple myeloma presented with a one month history of a burning pruritic rash. His rash started as a single blister on his right hand and gradually progressed to the arms, trunk, back, abdomen, legs, periorbital area, and oral cavity. He had not been able to eat due to the pain and blisters in his mouth. His cancers had been treated with rituximab, bendamustine, and velcade about one year prior and were considered in remission. On physical exam, he had annular erythematous erosions and desquamating plaques of the bilateral upper and lower extremities, abdomen, trunk and upper back. He had mucositis, but no ocular involvement. A skin biopsy showed suprabasal acantholytic dermatitis suspicious for paraneoplastic pemphigus. Direct immunofluorescence test showed deposition of IgG, C3 along the basement membrane correlating with high titers of desmoglein antibodies (243u\ml). Repeat myeloma panel was unremarkable, however abdominal imaging showed multiple newly enlarged lymph nodes concerning for recurrence of malignancy. Bone marrow and lymph node core biopsies revealed recurrence of grade IIIa follicular lymphoma. Immunohistochemical stains were positive for CD20, PAX5, CD10, and BCL-2 with a high proliferative index. He was started on high dose steroids and broad spectrum antibiotics along with aggressive daily dressing, but had no response to treatment. He expired three weeks later following progression of mucocutaneous, periorbital and facial disease and sepsis.

Discussion: Follicular lymphoma is the most common of the indolent Non-Hodgkin's lymphomas with a median survival of ten years. Treatment is often not required for asymptomatic patients. Although incurable, serious life threatening complications of Follicular lymphoma can occur and require immediate therapy. Paraneoplastic pemphigus (PNP) is a rare fatal skin blistering disorder occurring in association with variety of neoplastic diseases most commonly Non-Hodgkin lymphomas. The latter induces an immune response with antibodies formation directed against epithelial antigens playing integral role in cell adhesion. The detection of antibodies against two desmoplak-

ins (periplakin and envoplakin) are the most specific laboratory findings in PNP. Although the disease is initiated by the tumor, simply debulking the tumor or reducing tumor burden through chemotherapy will not halt disease progression. There is limited data on treatments involving steroids, Rituximab and immunosuppressive medications.

ONE OF THE BEST MIMICKERS OF METASTATIC DISEASE

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Introduction: Histoplasmosis is a common endemic mycosis and its clinical presentation varies greatly posing a diagnostic challenge.

Case: A 48-year-old, retired military officer presented to Emergency Department with complaints of generalized abdominal pain, intermittent headaches, weakness, vomiting and diarrhea along with 60 pounds weight loss in the last 3 months. His past medical history was significant for hypertension and an 80 pack year smoking history. His physical exam revealed hypotension, hepatosplenomegly and generalized abdominal tenderness. His labs showed hyperkalemia, anion gap metabolic acidosis, acute kidney injury with creatinine of 4.5 and adrenal insufficiency. CT Thorax and Abdomen without contrast revealed pulmonary nodules, diffuse abdominal lymphadenopathy, massive splenomegaly and 7cm bilateral adrenal masses. MRI of the brain showed 3 ring enhancing lesions. Our workup for HIV came back positive, however, lumbar puncture, flow cytometry and all other infectious disease workup were unremarkable. His renal function improved with hydration. After ruling out pheochromocytoma, core biopsy of Left Adrenal gland was performed which revealed Histoplasmosis. We initiated treatment with Fludrocortisone, IV Amphotericin, combined anti-retroviral therapy (CART) and empirically covered him for Toxoplasmosis. The patient's symptoms, laboratory findings and repeat imaging studies showed marked improvement.

Discussion: Histoplasmosis can present as an asymptomatic, pulmonary, progressive disseminated and primary cutaneous disease. The progressive disseminated form of the disease is rare and usually occurs in the immunocompromised patients. In Louisiana, it should always be considered in a patient with mediastinitis, pulmonary nodules, lymphadenopathy, hepatosplenomegaly, chorioretinits, adrenal enlargement and calcifications.

WHAT'S SO FUNNY?

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Introduction: Herpes simplex virus (HSV-1) encephalitis is the most common cause of sporadic fatal encephalitis in the US and worldwide. With an incidence of 2.2 per million, early recognition is crucial for prompt therapy. Despite those on therapy with IV acyclovir, the mortality can be as high as 30% with many survivors having significant long-term morbidity. We present a patient who appeared to have disseminated Herpes Zoster, but actually had disseminated HSV with suspected encephalitis.

Case: An 86-year-old African-American woman with a history of rheumatoid arthritis (on methotrexate) presented with delirium and fever of 103°F. Previously, the patient was fully independent. She was tachycardic, noted to have inappropriate laughter during the exam and appeared apathetic to her condition. She was found to have a disseminated non-painful, non-pruritic vesicular rash across the left labia majora, the posterior aspect of her thigh and face that appeared consistent with disseminated zoster. She was oriented to person, place, but not time. As she met SIRs, she was started on empiric antibiotics and IV acyclovir, and blood cultures were negative. The daughter refused lumbar-puncture for diagnosis and the patient was not cooperative for MRI. However, given her inappropriate laughter, we were not convinced this was disseminated zoster. Punch-biopsies of the rash confirmed our suspicion and revealed HSV-1 with no evidence of zoster. The patient remained afebrile on IV acyclovir, which was continued for 21-days. Mentation improved and she no longer laughed inappropriately.

Discussion: Patients with HSV encephalitis may present with severe behavioral abnormalities (as it did in our patient with her inappropriate laughter), anterograde amnesia, dysnomia, impaired learning for new verbal and visual material, and cognitive impairment. When suspected, HSV encephalitis requires early aggressive treatment with IV acyclovir to prevent mortality and decrease the risk of long-term morbidity. Empiric therapy with IV acyclovir at 10mg/kg q8H with treatment course of 14 to 21 days is recommended. Patients who survive HSV encephalitis can have significant long-term morbidity from post-encephalitic syndrome.

A VERY RARE ETIOLOGY OF RESPIRATORY FAILURE

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Introduction: The association between polymyositis/dermatomyositis and interstitial lung disease is well established. Dermatomyositis/dermatomyositis-induced respiratory muscle weakness is a very rare but reported complication of the disease.

Case: A 67-year-old African American woman with recently diagnosed dermatomyositis /polymyositis confirmed by muscle biopsy and MRI presented to our hospital with shortness of breath and acute hypoxemic respiratory failure. Initial workup revealed diffuse muscular weakness and very poor inspiratory effort. Chest X Ray was consistent with low lung volumes. Initial ABG revealed pH 7.247, pCO2 80, pO2 73, HCO3 34 on 2 L of oxygen per nasal cannula. Given her muscular weakness, recent diagnosis of polymyositis/dermatomyositis and ABG consistent with ventilatory failure, polymyositis/dermatomyositis induced respiratory muscle weakness was very likely. BiPAP was not successful and she was intubated shortly thereafter. Post-intubation ABG showed pH 7.47, pCO2 40.2, pO2 107.8, HCO3 28.7 on 100% FiO2 on the ventilator, consistent with intact lung parenchyma. A CT of the chest was done to rule out interstitial lung disease, a much more common pulmonary complication of polymyositis/dermatomyositis. She was noted to have a critically low NIF (negative inspiratory force) and unmeasurable vital capacity. She was initially started on pulse dose steroids, and later IVIG with no improvement. Finally, she was given two doses of rituximab and continued on PO steroids. Her measured vital capacity slowly improved, however in anticipation of continuous severe respiratory muscle weakness a tracheotomy tube was placed. The patient was transferred to an LTAC and was weaned from mechanical ventilation.

Discussion: Here a very rare, but still important complication of polymyositis/dermatomyositis is reported. Considering the critical nature of polymyositis/dermatomyositis-induced respiratory muscle weakness prompt diagnosis and management is crucial. Although interstitial lung disease is a more common pulmonary complication, internal medicine physicians should keep in mind the potential for respiratory muscle involvement and diaphragmatic dysfunction and respiratory distress in patients with polymyositis/dermatomyositis.

TTP, TERRIBLE THORACIC PAIN, AND A TOUCH OF TROUBLING PYLORI

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Introduction: Thrombotic thrombocytopenic purpura (TTP) is an occlusive thrombotic microangiopathy characterized by a systemic platelet aggregation, organ ischemia, and significant thrombocytopenia. TTP is frequently caused by either a congenital or acquired deficiency of ADAMTS13, a von Willebrand factor (VWF)-cleaving protease. The incidence of acquired TTP is 3 cases per 1 million adults per year with the median age at diagnosis of 41.

Case: A 41-year-old African American woman with a history of type 2 diabetes, HTN, HLD, and hypothyroidism presented with substernal chest pain with associated nausea, diaphoresis, and shortness of breath for 4 days. The patient was admitted for chest pain work-up but was later found to have symptomatic anemia and thrombocytopenia. Labs also revealed schistocytes on peripheral blood smear, elevated indirect bilirubin, elevated reticulocytes, and a markedly elevated LDH of 1700. Although she did not present with fever, neurologic defects, or renal defects, the patient was urgently treated for presumed TTP with plasma exchange (PEX) and prednisone 1mg/kg daily. TTP was confirmed by reduced ADAMTS13 activity. She initially responded to PEX, however due to recurrence, she was treated with Rituximab for 4 weeks. Due to gastrointestinal complaints, the patient tested and found to have H pylori infection. The patient improved after completion of rituximab and H pylori treatment, without any recurrence of TTP.

Discussion: Although TTP can present as chest pain (either as a form of acute coronary syndrome or from symptomatic anemia), this case is unique because of the patient's H pylori infection. Observational studies have suggested treating for H pylori infection in patients who present with gastrointestinal symptoms can improve platelet counts in idiopathic thrombocytopenic purpura (ITP). Just as in this case, there have been other documented cases of patients with TTP also presenting with H pylori infection. Some studies have implied H pylori infection could function as a triggering factor in TTP by inducing platelet aggregation through an interaction with VWF, however the exact pathophysiology remains unknown. Currently, testing for H pylori is not indicated for patients with TTP patients but might be a consideration for refractory cases.

RARE PHENOMENON OF ISOLATED AORTIC VALVE PROLAPSE

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Introduction: Aortic regurgitation is third most common valvular heart disease after Aortic stenosis and Mitral regurgitation. Prolapse of an aortic cusp is uncommon, present in approximately 15% of patients with ventricular septal defect but may also occur as an isolated phenomenon or as a consequence of myxomatous degeneration associated with mitral and/or tricuspid valve involvement.

Case: A49-year-old man with a past medical history of hypertension presented with isolated speech difficulties. He was a professional athlete and never smoked in his life. On physical examination his vitals were unremarkable except for an elevated blood pressure of 180/110 mm Hg. Central nervous system examination showed 'expressive aphasia' without any other neurological deficits. Cardiovascular examination and other systems examination was unremarkable. Electrocardiogram showed normal sinus rhythm with mild left ventricular hypertrophy. Noncontrast CT of the head showed a large left parietal lobe infarct and

a later MRI revealed a large middle cerebral artery distribution ischemic infarct. The patient was started on antiplatelet therapy. Transesophageal Echocardiography (TEE) showed isolated aortic valve prolapse with clear and significant prolapse of the noncoronary aortic cusp into the left ventricular outflow tract (LVOT). The prolapsed cusp almost impinged on the anterior mitral leaflets, occupying up to 68% of the LVOT width and with a pressure half-time (PHT) of 142 milliseconds. The diameter of the aortic root, sinotubular junction, and tubular ascending aorta were all within normal limits with no evidence of aneurysm, dissection, or other pathology. The valve itself was trileaflet with otherwise structurally normal leaflets without any vegetations or masses attached. Chamber sizes were also within normal limits. Due to lack of cardiorespiratory symptoms specifically chest discomfort or shortness of breath, and relatively normal measurements of chamber sizes on the echocardiograms, conservative management was initiated.

Discussion: Isolated aortic valve prolapse is a rare phenomenon and is commonly associated with a bicuspid aortic valve. TEE is diagnostic in almost every case and severity is easily estimated based on existing criteria. Definitive management depends on the stage of aortic regurgitation and these stages are well described in the 2014 ACC/AHA valvular disease guidelines. To date we have not come across a case of isolated aortic valve prolapse presenting with an ischemic cerebrovascular accident.

STRESS INDUCED SHORTNESS OF BREATH

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Case: A 60-year- old woman with a history of hypertension, tobacco abuse, and recent family stress presented to an urgent care clinic for chest pain and dyspnea and was transferred to the Emergency Department intubated secondary to respiratory failure. The patient was afebrile on admission but had a leukocytosis. Broad spectrum antibiotics were started for presumed lung infection based on radiographic evidence. CTA Chest showed pneumonitis, cardiomegaly and minimal dependent pleural effusions but was negative for pulmonary embolism. Initial troponin was 2.3 ng/ml and BNP 1270 pg/ml. The first EKG showed sinus tachycardia and diffuse T wave inversions were present in all leads except I and a VL. Wall motion abnormalities were observed on echocardiographic analysis. The mid septum, inferoseptal, posteroseptal, and apex of the heart were akinetic with minimal ballooning of the apex. The repeat EKG demonstrated worsening deep symmetric T wave inversions in the lateral leads and QT interval prolongation to 606 msec. Ciprofloxacin was discontinued. The troponin transiently increased hospital day 3 then trended back down within 12 hours. The patient went for a coronary angiogram hospital day 5 secondary to hemodynamic instability. The angiogram showed LAD free of atherosclerosis. The left ventriculogram demonstrated that the inferior wall was akinetic but with adequate basal contraction with anterior and apical hypokinesis, diagnostic of Stress induced (Takotsubo) cardiomyopathy.

Discussion: The most common cause of global deep T wave inversion is myocardial ischemia. However, multiple other etiologies including stress induced (Takotsubo) cardiomyopathy exist. Our patient met all four criteria for diagnosis of Stress induced Takotsubo cardiomyopathy: 1) transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement. Regional wall motion abnormalities typically extend beyond a single epicardial coronary distribution, and a stress inciting event may or may not be present. 2) Absence of obstructive coronary disease or angiographic evidence of plaque rupture. 3) New ECG abnormalities (ST segment changes, T wave inversions) or modest elevation in troponin. 4) Absence of pheochromocytoma or myocarditis.

DRUG-INDUCED CATATONIA, A CASE FOR EARLY DETECTION

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Case: A 70-year-old man with history of advanced microvascular dementia, hypertension, and alcohol abuse in remission presented with a four-day history of decreased verbal responsiveness, urinary incontinence, and inability to ambulate. He previously took quetiapine, haloperidol, and sertraline for treatment of aggressive features. Outpatient records revealed that haloperidol had been exchanged for risperidone with rapid increase in risperidone dose over the prior week, however he continued to receive high doses of both antipsychotics due to misunderstanding of physician instructions. At presentation, vital signs were within normal limits. Physical exam was significant for decreased attention and tracking, increased rigidity, hypertonia, hyporeflexia, cogwheel rigidity in right arm and resting tremor in both hands. He was reactive to verbal and painful stimuli. Laboratory results including TSH and basic chemistry were within normal limits. Creatinine Kinase was 1903. CT brain showed only chronic microvascular changes consistent with previous imaging. EEG indicated generalized slowing without seizure activity. MRI brain was deferred as the patient was unable to cooperate without sedation. The patient slowly improved over the next week with supportive treatment and withholding of all home psychotropic medications. He remained afebrile with down trending of CK without indication for treatment with benzodiazepines or dantrolene. At discharge, he continued to have some mild cogwheel rigidity but was verbally responsive and improving in strength with physical therapy.

Discussion: The existence of catatonia in patients with dementia has been rarely reported and can be confused with symptoms of the underlying disease, causing delay in diagnosis and adverse outcomes. The mechanism of drug-induced catatonia is unclear, but likely involves dopamine, gamma-aminonutyric acid, and glutamate neurotransmitters. Unlike NMS and serotonin syn-

drome, drug-induced catatonia has variable presentation, including dysautonomia and hyper or hyporeactivity. Early detection and initiation of supportive treatment such as intravenous fluids and withholding of the offending medication can result in complete resolution and prevention of worsening to NMS, suggested to be on the same continuum as drug-induced catatonia.

AN UNUSUAL CASE OF SPHINGOBACTERIUM CELLULITIS

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Introduction: Sphingobacterium species are non-fermentative, gram-negative rods. This bacterium was first described as a Flavobacterium species but due to the large amounts of sphingophospholipid compounds in their cell membranes distinguish them the Flavobacterium species. Shingobacterium species have been isolated from soil, plants, foodstuffs, and water sources, but the isolation of the species from human clinical specimens has been rarely reported worldwide.

Case: An 81-year-old man presented with 3 day history of right arm swelling with pain and erythema extending into his forearm. The patient was started on Clindamycin therapy for cellulitis after blood cultures were obtained. A Gram-negative rod was isolated in both anaerobic bottles and one aerobic bottles. Speciation and susceptibilities returned with Sphingobacterium spiritivorum and antibiotics were changed to ciprofloxacin. The patient was discharged in stable condition and completed 7 days of antibiotics.

Discussion: In the present case, this immunocompetent patient presented with cellulitis and bacteremia that was identified as S. spiritivorum. Only 4 cases of S. spiritivorum infection have been reported worldwide. The isolate was susceptible to ciprofloxacin, piperacillin-tazobactam, and trimethoprim-sulfamethoxazole. The source of infection was probably environmental and this is another case of cellulitis due to this organism that might have been missed if blood and tissue or wound cultures were not collected, emphasizing the usefulness of these cultures in patients with cellulitis.

EPSTEIN BARR VIRUS ASSOCIATED DIFFUSE LARGE B CELL LYMPHOMA

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Introduction: Epstein Barr virus positive diffuse large B cell lymphoma of the elderly is a provisional entity in the 2008 WHO classification of lymphoid neoplasms. Few studies have reported EBV positive large B cell lymphomas in young patients without immunodeficiency.

Case: A 20-year-old Hispanic man presented to the ED after noticing swelling to bilateral sides of his neck for the past 2 months. He denied any fevers, chills, night sweats, weight loss or weakness. On physical exam, he was noted to have bilateral preauricular, postauricular, anterior cervical, posterior cervical, supraclavicular and axillary lymphadenopathy. Initial showed total protein of 9.6, globulin of 6.0, normal renal function and liver function tests, mild leukocytosis with WBC=11.3, hypochromic microcytic anemia, normal platelets and normal calcium. CRP and ESR were increased. HIV, RPR, acute hepatitis panel and Monospot were all non-reactive. CT-neck/thorax/abdomen/pelvis showed extensive lymphadenopathy above and below diaphragm. FNA biopsy of left post auricular lymph node showed cytomorphology of atypical lymphoid proliferation. Excisional biopsy was done of left neck with pathology report revealing EBV positive diffuse large B cell lymphoma. EBV in situ hybridization was positive in many cells. Serology showed positive IgG EBV antibody. Bone marrow biopsy of left posterior iliac crest showed no evidence of lymphoma. Patient was diagnosed with Stage III diffuse large B cell lymphoma. He completed 6/6 cycles of R-CHOP therapy to date with complete response by Recist criteria on imaging.

Discussion: EBV positive diffuse large B cell lymphoma (DLBCL) of the elderly is defined as patients older than 50 years alone. Recent studies have shown that patients with sound immune status can also be affected. EBV positive patients, both in the elderly and young groups, showed significantly worse overall survival than negative cases. In one study, no significant differences of outcomes were identified between different age groups with EBV positive DLBCL. This case illustrates that EBV positive DLBCL can occur in young patients without immunodeficiency.

PARAESOPHAGEAL HERNIA MASQUERADING AS RIGHT HEART FAILURE

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Introduction: A paraesophageal hernia is an uncommon type of hiatal hernia accounting for approximately 5 percent of all cases. They are most often asymptomatic or associated with vague and intermittent symptoms of gastroesophageal reflux.

Case: A 59-year-old woman with a past medical history of anemia and obesity presented to the hospital on five separate occasions within one year for recurrent symptoms of substernal chest pain, shortness of breath and bilateral lower extremity edema. During these hospitalizations, she underwent repetitive workups to rule-out cardiopulmonary etiologies. All work-up including chest x-rays, electrocardiograms, echocardiograms, and

labs continued to be negative with the exception of a mild anemia and a CTA of the chest which was repeatedly negative for a pulmonary embolus but revealed a large paraesophageal hernia. Because of the well-known illness script of right heart failure being shortness of breath with a clear chest x-ray and systemic edema, she was labeled and treated as such on previous admissions. Upon thorough review of diagnostics performed, she did not have evidence of left heart failure, high pulmonary artery pressures, or right heart dilation or dysfunction. However, upon review of the CTA chest images, the hernia sac was large and appeared to be compressing the inferior vena cava. This was believed to be a possible etiology for dyspnea and lower extremity edema. The gastroenterology team was consulted, an esophagogastroduodenoscopy was performed which confirmed the large paraesophageal hernia and was diagnostic for multiple associated Cameron erosions which likely explained the etiology of her chest pain and anemia. General surgery was consulted and the patient underwent a robotic-assisted paraesophageal hernia repair with Nissen fundoplication. One month after the surgery, the patient was doing well with less shortness of breath and resolution of chest pain and peripheral edema.
Discussion: This case is a representation of a rare cause of a very common presentation. It should also raise awareness about the possible mechanical effects of a large hiatal hernia on the cardiopulmonary system.

KIDNEY STONES…NO! IT'S A SARCOIDOSIS

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Introduction: Sarcoidosis is a multisystem disorder produced by non caseating granulomas that infiltrate various organs. The cause is unknown and it occurs in 1 per 20,000 in the US. It is more prevalent in the African American population compared to Caucasian and typically affects both men and women with the peak onset age 20-40yrs. It typically presents with bilateral hilar adenopathy, pulmonary reticular opacities, skin, joint and/or eye lesions. Diagnosis is usually made by evidence of non-caseating granulomas on biopsy. Treatment includes steroids and immunosuppressives such as methotrexate.

Case: A 35-year-old man with PMH hypertension, BPH, type 2 diabetes, and recurrent nephrolithiasis presented to the Emergency Department for worsening bilateral flank pain of 1 week duration. The patient had multiple visits to the ED and clinic during the prior month for hypercalcemia with nephrolithiasis/hydronephrosis. During this time, he had worsening fatigue but denied fever, chills, nausea, vomiting. At presentation, vital signs were stable. Physical exam was remarkable for CVA tenderness. Labs was notable for anemia, hypercalcemia, elevated LFTs and renal failure. Further workup for the hypercalcemia revealed an elevated ACE level, low PTH, elevated 24hr urinary Ca, elevated calcitriol, low Vit D. Imaging showed granulomatous disease with non-calcified mediastinal/hilar lymph nodes on CT chest and enlarged pericardiophrenic lymph nodes, and multiple non obstructing renal calculi. The hypercalcemia and renal failure resolved with fluids. A CT guided biopsy of the liver revealed non caseating granulomas consistent with sarcoidosis. The patient was started on low dose prednisone which improved his symptoms as well as his hypercalcemia.

Discussion: This case illustrates the importance of appropriate recognition of affected organ systems with clinical and radiologic findings plus support with histologic evidence for the confirmation and diagnosis of disease. Although ACE level (Angiotensin Converting Enzyme is an insensitive and nonspecific diagnostic test, the elevated level provided a higher index of suspicion for sarcoidosis since ACE levels are elevated in 60% of patients with sarcoidosis.

UNCOMMON CAUSE OF A COMMON CONDITION

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Introduction: Acute recurrent pancreatitis (ARP) is a clinical condition characterized by repeated episodes of acute pancreatitis; ARP is therefore diagnosed retrospectively by clinical definition after at least the second episode of acute pancreatitis. Etiology of ARP can be identified in 70% of the cases with most common causes being common bile duct stones or sludge and bile crystals; sphincter of oddi dysfunction; anatomical ductal variants interfering with pancreatic juice outflow; obstruction of the main pancreatic duct or pancreatico-biliary junction; alcohol consumption. Drugs as a cause for acute pancreatitis have been described in 5% of the cases2, with only 8 previously described cases of metronidazole induced acute recurrent Pancreatitis. Here we present the 9th reported case of ARP due to Metronidazole.

Case: A 26-year-old woman with one previous episode of Acute pancreatitis presumed to be due to metronidazole came to the hospital with epigastric abdominal pain radiating to the back, associated with nausea and vomiting for the past 24hrs. The patient was prescribed metronidazole 2 days ago for bacterial vaginosis. She was found to have a Lipase level of 650U/l and the common causes of pancreatitis such as CBD stones, gallbladder sludge, Hypertriglyceridemia, Alcohol, sphincter of oddi dysfunction, anatomic ductal variants, pancreatic divisum and autoimmune pancreatitis were ruled out. The patient was not on any medications that are known to cause Pancreatitis.

Discussion: Metronidazole is commonly used in the hospital and community setting as it is a potent and effective treatment for many anaerobic infections. The mechanism of Metronidazole induced is unknown but one possible mechanism of action could be that under aerobic conditions Metronidazole may undergo redox cycling and yield hydrogen peroxide, superoxide, and oth-

er free radicals which are toxic to the pancreatic beta cells and induce pancreatitis. The incidence of severe Metronidazole-induced pancreatitis is relatively rare and the causative association was confirmed in the Midwest multicenter Pancreatic Study; however, it can cause serious morbidity in patients after minimal exposure, and identification of Metronidazole as the causative agent is the key to recovery. Early diagnosis, discontinuation of the drug, and supportive care will lead to a successful recovery in the majority of cases. Re-challenge should always be avoided.

DIAGNOSIS "PROBABLE?" DIAGNOSTIC DIFFICULTY IN CARDIAC SARCOIDOSIS

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Case: A 47-year-old woman with hypertension, dyslipidemia, and diabetes presented to her PCP with two weeks of dyspnea. An ECG a year prior showed normal sinus rhythm. The rhythm strip from a transthoracic echocardiogram incidentally showed complete heart block. An ECG confirmed new persistent third degree AV block. A left heart catheterization ruled out coronary artery disease and workup for other reversible causes such as Lyme disease, syphilis, autoimmune (ANA, ANCA, and CCP), thyroid disease was negative. A transesophageal echocardiogram was negative for valvular abnormalities. A cardiac MR was nondiagnostic for an infiltrative process, but showed late gadolinium enhancement in the inferolateral septum, indicative of nonspecific scarring. Nuclear myocardial perfusion study then showed abnormal perfusion along the anterior wall and areas of late enhancement, again nonspecific findings for cardiac sarcoid. A cardiac positron emission tomography scan showed multiple hypermetabolic regions within the heart consistent with a sarcoid pattern as well as mediastinal lymphadenopathy. In collaboration with Rheumatology, a diagnosis of "highly probable" primary cardiac sarcoidosis was made without histology and she was started on methotrexate. Electrophysiology implanted a dual chamber pacemaker with moderate symptomatic improvement with ICD placement subsequently planned.

Discussion: Cardiac sarcoidosis (CS) may be present in 5 to 25 percent of patients with sarcoidosis and causes significant morbidity and mortality. Its effects are often clinically silent and may precede other organ involvement. Further, CS manifests as diverse pathologies such as arrhythmias, heart failure, sudden cardiac death, valvular dysfunction, and pericardial disease. While sarcoid is frequently on the differential as a great imitator, definitive diagnosis remains problematic despite advanced cardiac imaging modalities like magnetic resonance and dedicated cardiac PET. Internists should consider cardiac sarcoidosis in the differential for any new unexplained cardiac abnormalities, particularly ECG findings in younger adults.

WEST NILE VIRUS WITH WEAKNESS OF UNILATERAL EXTREMITIES AND HYPONATREMIA

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Introduction: Here we present a case of WNV encephalitis which presented with meningitis as well as SIADH and later developed acute onset of left sided weakness.

Case Description: A 58-year-old woman with no known medical history presented with complaint of five days of fever, headache, malaise, nausea, and vomiting. On examination, she was hemodynamically stable and febrile (38.6°C) with epigastric tenderness. Labs revealed leukocytosis (12.9x109/L) and hyponatremia (130 mmol/L). The morning after admission she complained of worsening headache. She experienced altered confusion, neck stiffness, and a diffuse erythematous rash. CSF studies demonstrated 256 WBCs/µL, total protein of 168 mg/dL and glucose of 73 mg/dL, with negative gram stain. Empiric treatment was initiated for bacterial and HSV meningitis. Serum sodium was 134 mmol/L with urine osmolality of 784 mosm/kg and urine sodium of 65 mmol/L. The fourth day of hospitalization she had acute onset of left facial droop and weakness of her left upper and lower extremities (1/5 strength) and a stroke activation was called. CT was negative for intracranial hemorrhage. tPA was administered, with no improvement of her weakness. MRI of the brain and spinal cord were unremarkable. She became increasingly somnolent and was intubated. Her hyponatremia worsened to the 120s mmol/L, which was managed with hypertonic saline after transfer to the ICU. CSF IgM returned positive for WNV. CSF HSV PCR and VDRL were negative and bacterial cultures were no growth. She was extubated after 4 days and was discharged to a nursing facility with persistent weakness in her left arm (3/5) and leg (1/5).

Discussion: Neuroinvasive WNV is associated with a highly variable clinical course and a fatality rate of approximately 10%. The most important risk factor is older age. Symptoms typical of meningitis/encephalitis as well as acute onset of poliomyelitis-like flaccid paralysis of the extremities may occur. Probable diagnosis requires IgM antibody to WNV in serum or CSF. Viral encephalitis may also present with hyponatremia secondary to SIADH. Treatment for WNV is supportive.

RECTAL CARCINOID: AN EVOLVING PHENOMENON

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Introduction: Adenocarcinoma is the most common malignan-

cy found in the rectum. However, the incidence of rectal carcinoid has been rising after implementation of screening colonoscopy. The rectum is the second most common anatomic site for carcinoid tumor. Embryologically, rectal carcinoids are hindgut tumors which are non-secretory. Hence, they rarely present with carcinoid syndrome even in patients with metastatic disease. A recent systematic review of rectal carcinoid by McDermott et.al was unable to perform a meta-analysis on optimal treatment strategies for localized and widespread disease due to the wide variety of data recorded from various sources. Our aim is to make physicians aware that the rectal carcinoid is becoming a common diagnosis thus clinicians should have current knowledge of classification, management and treatment options available.
Case: A 62-year-old African American man with a significant past medical history of hypertension and tobacco abuse presented to the gastrointestinal (GI) lab for colorectal cancer screening colonoscopy. The patient had no specific GI complaints and a 10-point review of symptoms was unremarkable except for decreased exercise tolerance. Physical examination was unremarkable including digital rectal exam. During colonoscopy, a 7-10 mm firm polypoid submucosal lesion was noted in distal rectum and was resected using an electrocautery snare and ERBE endocut current. Histologic evaluation of the resected polyp demonstrated a well-differentiated neuroendocrine neoplasm that stained positive for chromogramin, synaptophysin, and Ki-67 proliferation marker. The patient underwent repeat colonoscopy about a month later. Biopsy obtained from previous polypectomy site was negative for carcinoid tumor cells.

Discussion: The rectal carcinoid classification, management and treatment options have been defined based on the size and aggressiveness of the lesion. As there is no defined TNM system classification, there is no homogenous data available yet on how to manage rectal carcinoid. Treatment strategy is based on tumor size, radiologic testing and surveillance examination including use of rectal EUS.

I GOT "C" AND I CANNOT SEE

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Introduction: Until 2010, 36 cases of Group C streptococcus meningitis had been identified. Group C streptococci (GCS) are common causes of veterinary diseases but they are not commonly recognized as causes of human disease.

Case: A 31-year-old Hispanic man who worked as an auto mechanic and had no known past medical history presented with a five day history of fevers, chills, lethargy, nausea with vomiting, non-bloody diarrhea, photophobia, severe headache and neck stiffness. He had been taking antiemetics and antidiarrheal along with acetaminophen and ibuprofen without relief. He also reported right shoulder pain with associated weakness, decreased mobility due to unsteadiness and diplopia. The patient was febrile on presentation and had physical exam findings suggestive of meningitis. Also noted was a painful erythematous nodule was noted on his bottom lip, Janeway lesions noted on his fingertips, unsteady gait with ambulation, as well as left sided hearing loss. Lumbar puncture revealed CSF consistent with bacterial meningitis: cloudy fluid, elevated WBC, predominantly neutrophils, elevated RBC, decreased glucose, elevated protein. Blood cultures and CSF cultures were positive for Group C Streptococcus equi. Transthoracic and transesophageal echo were unrevealing of vegetation. MRI of the brain revealed a pattern consistent with septic emboli with multiple infarcts in the brain including the cerebellum. Due to multiple infarcts, the patient was treated presumptively for endocarditis despite negative studies. The patient was treated with intravenous penicillin G (for 4 weeks) and gentamycin (for 2 weeks) for Group C streptococci endocarditis and meningitis. At 4 week follow up, the patient had greatly improved.

Discussion: GCS can cause cellulitis, bacteremia, endocarditis, meningitis, septic arthritis, and upper respiratory infections. Patients with GCS infection may present with symptoms such as nausea, vomiting, fever, chills, and typical meningitis symptoms (headache, photophobia, neck stiffness). Even though some patients who had a GCS infection reported exposure to animals or contaminated/unpasteurized dairy products, more than 50% of patients do not have an identifiable etiology of their infection.

A NARROW ESCAPE FROM THE KISS OF DEATH

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Background: Despite the fact that snakebite is a significant source of morbidity and mortality, it often goes unreported.

Case: A 30-year-old man presented with a venomous water moccasin bite on his right hand that occurred while he was working in his yard an hour prior to his arrival to the ED. He felt nauseated and was in excruciating pain. Initial physical exam revealed erythematous and tender bite marks on the dorsum of his hand, in the web between his thumb and index finger. There was swelling that extended from the fingers to the mid of his forearm along with numbness and parasthesias. There was no discoloration and pulses in the right upper extremity were palpable. Lab finding revealed Rhabdomyolysis and coagulation studies were unremarkable. He was immediately reported to poison control and given 6 vials of Cro-fab and a tetanus shot. Despite the initial treatment the swelling worsened and progressed up to his right axilla within 12 hours during which he continued to receive 6 vials of Cro-fab repeatedly. Physical exam at this time revealed pale hand, loss of flexion and extension of the fingers due to tense edema, tenderness, and absent pulses at the wrist. Orthopedic surgery was consulted for Compartment syndrome and

he underwent immediate decompressive fasciotomy. On his follow up visit after 1 week, his pain had resolved. Physical exam revealed normal hand movements and pulsation with minimal edema.

Discussion: Annually about 45000 snake bites occur in the US of which approximately 8000 are from venomous snakes. Snake bites, though not uncommon can lead to a rare complication of Compartment syndrome, very few cases of which have been reported throughout the globe. In addition to sound clinical judgment, continuous close monitoring is required for prompt diagnosis and treatment to avoid severe necrosis requiring amputation.

TREATMENT OF T2DM WITH SODIUM-GLUCOSE COTRANSPORTER LEADING TO EUGLYCEMIC DIABETIC KETOACIDOSIS

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Introduction: Sodium–glucose cotransporter 2 (SGLT-2) inhibitors are the most recently approved antihyperglycemic medications used as an adjunctive treatment in managing type 2 diabetes. This new class of drugs have been associated with diabetic ketoacidosis (DKA) with uncharacteristically mild to moderate glucose elevations (euglycemic DKA [euDKA]). The atypical clinical presentation makes euDKA difficult to recognize.

Case: A 40 year- old woman with past medical history significant for T2DM controlled with metformin 500 mg BID and canagliflozin 300 mg qday, presented to the emergency department with a 1-week history of increasing fatigue and generalized weakness. The patient had also been complaining of diffuse abdominal pain, associated with nausea, vomiting and decreased oral intake. The symptoms persisted despite using antiemetic medication at home. The patient noted blood glucose at home in the 150s with adherence to medication regimen. Physical exam was unremarkable except for, dry mucous membranes, tachycardia and mild epigastric tenderness. Laboratory data yielded a blood glucose of 125, bicarbonate of 10, venous ph of 6.630 and calculated Anion Gap of 26. Ketonemia was noted, Beta hydrohybutyrate was 39. C- peptide was WNL. Ethanol, methanol and ethylene glycol was negative. Latic acid was 1.4. No source of infection was detected. The patient was started on DKA protocol with IV fluids and insulin and canagliflozin was discontinued. After all interventions above ketoacidosis eventually resolved. Patient's symptoms resolved as well completely.

Discussion: This case illustrates the potential complications seen with the use of (SGLT2) inhibitors in diabetic patients. Increased renal clearance of glucose mediated by the SGLT-2 inhibitor led to deceivingly low blood glucose levels in the setting of acute illness. Due to the odd presentation of this DKA case with the abnormally low to normal serum glucose level, timely identification of ketoacidosis with increased anion gap is critical to institution of appropriate medical therapy to avoid further metabolic derangements.